Volume 95 No. 5 May 2012

Medicine & Health
RHODE ISLAND

# Medical Imaging
# Part II

# We're not LIKE A Good Neighbor,

# WE ARE

# The Good Neighbor Alliance


*Specializing in Employee Benefits since 1982*

Health Dental Life Disability Long Term Care
Pension Plans Workers' Compensation Section 125 Plans

**The Good Neighbor Alliance Corporation**

*The Benefits Specialist*

*Affiliated with*

RHODE ISLAND MEDICAL SOCIETY

RIMS-INSURANCE BROKERAGE CORPORATION

401-828-7800 or 1-800-462-1910

P.O. Box 1421 Coventry, RI 02816

www.goodneighborall.com

*Cover*: "Ripened Vine," watercolor, by Sally Ann Martone. Sally Ann is an alumnus of RISD and Bryant College, and has been painting for over 20 years. She specializes in portraits, watercolors, and medical and scientific illustrations. She is a past president of the Rhode Island Watercolor Society and a signature member of the New England Watercolor Society. Inspiration for Martone's work comes from a "Une joie de vivre." Her website can be found at www.sallyannmartone.com.

**Medicine & Health**
# RHODE ISLAND

**VOLUME 95 NO. 5 May 2012**

**PUBLICATION OF THE RHODE ISLAND MEDICAL SOCIETY**

**COMMENTARIES**



**CONTRIBUTIONS**
**SPECIAL ISSUE: IMAGING ISSUE, PART II**
**Guest Editor: *Kevin J. Chang, MD***

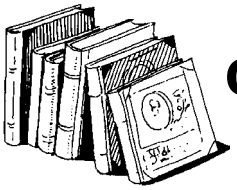


**COLUMNS**



***Medicine and Health/Rhode Island*** *(USPS 464-820), a monthly publication, is owned and published by the Rhode Island Medical Society, 235 Promenade St., Suite 500, Providence, RI 02908, Phone: (401) 331-3207. Single copies $5.00, individual subscriptions $50.00 per year, and $100 per year for institutional subscriptions. Published articles represent opinions of the authors and do not necessarily reflect the official policy of the Rhode Island Medical Society, unless clearly specified. Advertisements do not imply sponsorship or endorsement by the Rhode Island Medical Society. Periodicals postage paid at Providence, Rhode Island. ISSN 1086-5462. POSTMASTER: Send address changes to Medicine and Health/Rhode Island, 235 Promenade St., Suite 500, Providence, RI 02908. Classified Information: Cheryl Turcotte/Rhode Island Medical Society, phone: (401) 331-3207, fax: (401) 751-8050, e-mail: cturcotte@rimed.org. Information on permissions and reprints available from jdteehan@rimed.org.*

**Note:** *Medicine & Health/Rhode Island* appears on www.rimed.org, under Publications.



# Commentaries

# – Rejections –

**There is a wonderful YouTube speech** given by Jon Nakamatsu called, "I'm a Loser." He was the winner of the Van Cliburn piano competition in 1993 and was serving on the jury for the same competition several years later. He is one of the great classical musicians of our time, and, if you listen to his talk, you will agree that he's a most sensitive and thoughtful man. He discusses his many "failures" in competition over many years until he won the Van Cliburn, one of the most competitive piano competitions in the world. He describes not making the first cut, as well as scoring higher but not winning, hence "losing," many events, all of which were of lesser significance. He even describes a judge, trying to be supportive, suggesting that he refrain from playing Chopin, since Japanese are unable to interpret his music properly, that is, with the same sensitivity as Europeans. Nakamatsu's point was, of course, that no one wins all the time, and that there should be no stigma in losing.

As in the world of music, literary submissions are routinely savaged and few authors are "winners." Revealing the all-too-human failures of the rejecters, the people who judge submissions, are published collections of rejection letters of works that subsequently became famous. You can imagine the gnashing of teeth in the publishing houses that rejected the first Harry Potter book. Tony Hillerman's books were thought to be well written but an editor suggested *that the "Navajo bit"* should be deleted. Jack London wrote about his many early failures in his novel, Martin Eden, and made a point in his later, famous years, both as the character, Martin Eden, and in real life, of forcing editors who wanted to publish his new work to publish his old rejected work. Rubbing their noses in it was part of his game plan. "*Gone with the Wind* is going to be the biggest flop in Hollywood history." Anne Frank "doesn't, it seems to me, have a special perception or feeling which would lift that book above the 'curiousity' level." And even in the "objective" sciences, discoveries, especially if they broke new ground, have often been rejected. When Rosalyn Yalow gave her Nobel Prize address in 1977, she showed slides of the nasty rejections she had received when her newly-developed techniques demonstrated that insulin and glucose control did not behave the way they were thought to. The theory that peptic ulcers were caused by bacterial infection was denigrated for decades until these very persistent researchers also won the Nobel Prize. In my own field, Stanley Prusiner, who won a Nobel Prize for helping to define and explain prion diseases, was not above denouncing the people who had rejected his theories and his papers.

And here I am, peer reviewing for a number of journals, rejecting articles, even as my own submissions to the same journals are also rejected. I sometimes reflect on an early rejection for my first "major" paper. I had observed a neuroleptic malignant syndrome-like reaction in two **Parkinson's Disease (PD)** patients when they were taken off their PD medications for a so-called "drug holiday." I submitted my report to the main neurology journal, from which I got a review stating that not only was this problem already reported, but, paradoxically, should not be further reported because it might make doctors fearful of using L-Dopa to treat PD. The *New England Journal* liked it but not enough to print it. *JAMA* published it, both in English and Japanese. I learned the lesson of being persistent if I believed the material was deserving and I've lived through this business of being rejected one place but accepted somewhere else to have become somewhat inured to the rejections.

I was recently entertained by a rejection. My column in last month's MHRI was a satirical piece in the form of what a modern review of James Parkinson's famous monograph, *The Shaking Palsy*, might look like. I chastised the author for excessive wordiness, ornate phrasing, lack of IRB approval and for reporting on patients he had only seen from a distance and had never actually examined. My satire was rejected by a prominent neurology journal It had been reviewed by five peer reviewers. Usually manuscripts are reviewed by either two or three reviewers. I am unsure why five were involved, whether it was due to the section of the journal it was intended for (humanities rather than clinical research) or whether the editors-in-chief didn't like it and the first three reviewers did, so they roped in two more reviewers to kill it. I'll never know the answer. But I found the reviews interesting. The first three reviewers found the article humorous, satirical, perhaps even caustic. Reviewer five provided the coup de grace, "This simply isn't funny," which is certainly not a statement one can argue with. But one reviewer was clearly incensed that I failed to understand the nature of 18th century neurology, citing a number of publications of the era and the stylistic differences between today's reports and those, referring me to other publications of the time that I must have been ignorant of, how medicine was practiced differently then, and even wondered whether a citation I made up (*The British Empire's Classification of Diseases*) was real or not. He/she noted that 18th century physicians wrote monographs so that my critique of the manuscript as if it was a journal submission was misplaced. And I apparently failed to understand that there were stylistic differences between writings then and now. I could not help wondering how one can parody oneself. That reviewer was able to do it. It appears that some people just can't take a joke.

**– Joseph H. Friedman, MD**

### Disclosure of Financial Interests

Lectures: Teva, Ingelheim Boehringer; General Electric

Consulting: United Biosource; Bubaloo, Halsted, Reitman LLC; EMD Serono; Genzyme; Teva; Acadia; Addex Pharm; Schwarz Pharma

Research: MJFox; NIH: Cephalon; EMD Serono; Teva; Acadia

Royalties: Demos Press


### Correspondence

e-mail: joseph_friedman@brown.edu

# When May Ignorance Become a Blessing?

**A wondrous event recently took place in a Providence** elementary school classroom. It happened quietly, with little fuss and with no newspaper reporters to record the precious moment. The class teacher was discussing events surrounding World War II and noted, in passing, that President Franklin D. Roosevelt had been a victim of polio. A young student raised her hand and earnestly inquired: "What's polio?" It is likely that most if not all of her fellow students were equally ignorant of the term; and if their teacher were younger than age 55—born in this nation—she too might have known the word only through the reading of history books. Were Jonas Salk and Albert Sabin still alive, they would have glanced at each other and grinned broadly.

Acute poliomyelitis is caused by a virus that principally attacks the brain cells that subserve and control the voluntary muscles of the body. The virus circulates via sewage-contaminated water and often attacks the healthiest, most physically vigorous youngsters in a community. The result is a flaccid (limp) paralysis of one or more limbs, and sometimes, in more severe cases, even the muscles that control breathing are compromised.

The Salk vaccine, rendered through injection, was first used in trials in 1956; and the Sabin oral vaccine, some years later. On the 20th anniversary of the first polio vaccine trials in Rhode Island, Jonas Salk, as guest of Brown University, joined the local physicians in celebration.

Although an occasional case of polio arises in the United States, often in a migrant child who had been infected overseas, this nation has been essentially polio-free for many decades, in contrast to the earlier decades of the 20th century when as many as 200,000 children were afflicted annually.

And the remainder of the world? Polio transmission, thanks to extensive use of the vaccines, has been interrupted in all nations except for four countries: Nigeria, Afghanistan, Pakistan and India, four nations burdened by extensive poverty, forced migrations, civil wars, a paucity of health education facilities and inadequate medical resources. In Nigeria, for example, rumors that the virus caused sexual sterility in children caused many ill-advised parents to shun the vaccine.

Until recent years, India was heavily plagued with yearly epidemics of polio, often exceeding 50,000 victims per year. A conscientious effort, beginning in 1988, concentrated on the children of the urban slums and particularly children of migrant workers. This strategy diminished the annual number of paralyzed child to about 800. By the year 2009, the Indian government estimated that the number of children not provided with the polio vaccine was down to 12.3%. A large-scale effort was then undertaken in 2010 and, since then, only two cases of verified polio hace been recorded: one in West Bengal (January, 2011) and one in Jharkhand (October, 2010). In a vast country of some one billion persons, 31% of which are below the age of 15, and in an area of about 1,269,000 square miles, this was an immense, an awesome accomplishment.

Beginning with Jenner's vaccine to prevent smallpox back in 1796 and with the subsequent vaccines to prevent childhood measles, rubella, diphtheria, tetanus, pertussis, pneumonia, hepatitis and a handful of other childhood communicable ailments, the world has witnessed a miracle: Through the 16th century, a newborn child, say in England, had about one chance in three of surviving until age 18. Today, in that same England, the chances of survival to adulthood hover about 98%. Historians will agree: The preventive vaccines against childhood diseases have saved more lives than any other single factor in the life-preserving resources of the medical profession. Mothers—whether they be English, Lithuanian or Cambodian—no longer must plan for at least three pregnancies to ensure that perhaps one child will survive to adulthood and thereby insure for their declining years.

Today, a class of school children blissfully unaware of the threats of diphtheria, measles, whooping cough, poliomyelitis and smallpox—or even the meaning of those names of the pestilences—is cause for all of us to smile. At this very moment there are scientists, here and elsewhere, working diligently so that school children, generations hence, may also raise their hands to inquire of their teacher; "What is cancer?"

**– Stanley M. Aronson, MD**

*Stanley M. Aronson, MD is dean of medicine emeritus, Brown University.*

**Disclosure of Financial Interests**

The author and his spouse/significant other have no financial interests to disclose.

**Correspondence**

e-mail: SMAMD@cox.net

# Imaging Issue: Part II

*Kevin J. Chang, MD*

**In this second part of our two-part Imaging Issue for** *Medicine & Health/Rhode Island*, we share another dozen of our most interesting imaging-related cases. A few topics addressed in this issue include endorectal MRI of the prostate, "4D CT" in the detection of parathyroid adenomas, advanced cardiac imaging including ECG-gated MRI and CT angiography, the use of MRI to evaluate tumor perfusion and gynecologic anomalies, specific oncologic applications for FDG PET-CT, as well as interesting cases from Interventional Radiology, chest imaging, and pediatric imaging. This issue serves to illustrate not only some of the current capabilities of diagnostic imaging but a few of the image-guided therapeutic options available for treatment as well. It is hoped that this two-part Imaging Issue will demonstrate the wide variety of imaging examinations and treatment options available to many subspecialties of medicine as well as illuminate future directions and possibilities in the ever-changing world of high-end sub-specialized imaging.

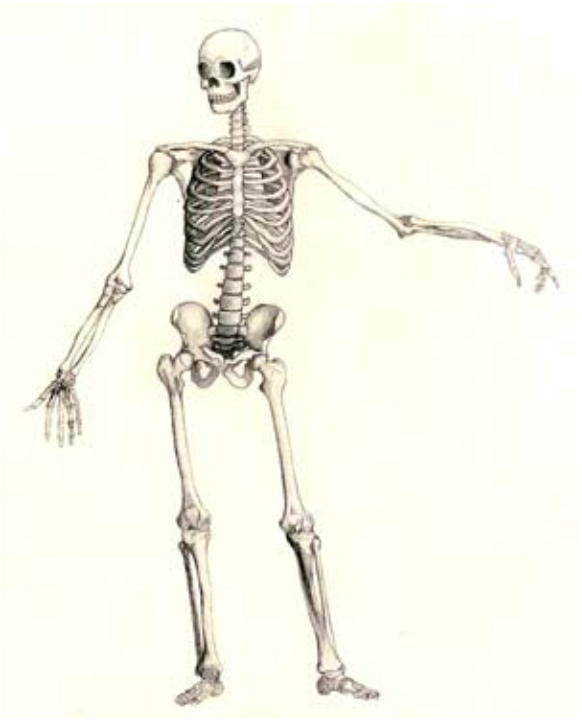
*Kevin J. Chang, MD is Director of CT Colonography at Rhode Island Hospital and The Miriam Hospital and Assistant Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University.*

**Disclosure of Financial Interests**

The author and/or his spouse/significant other has no financial interests to disclose.

**Correspondence**

Kevin J. Chang, MD
Dept. of Diagnostic Imaging
Rhode Island Hospital
593 Eddy St.
Providence, RI 02903
phone: (401) 444-5184
e-mail: kchang@lifespan.org



# Prostate MRI For the Detection of Extracapsular Extension

*Kevin J. Chang, MD, and Courtney A. Woodfield, MD*

**A SIXTY-YEAR OLD MALE WAS FOUND TO HAVE** a palpable prostatic nodule on screening digital rectal examination. A serum **prostatic specific antigen (PSA)** assay yielded a concentration of 25.8 ng/ml (normal range 0-4 ng/ml). A transrectal ultrasound guided 12-core biopsy was then performed and confirmed Gleason 8 (4+4) and 9 (4+5) disease involving both sides of the prostate. A staging CT of the chest, abdomen, and pelvis (not shown) revealed a borderline-enlarged left obturator lymph node without other evidence of distant metastatic disease. Given a clinically palpable tumor with a significantly elevated serum PSA and relatively high Gleason grade, the use of Partin tables predicted a high probability of extracapsular tumor extension. To help guide clinical management, an MRI of the prostate gland using an endorectal coil was ordered. Multiplanar T2 weighted images of the prostate confirmed a large bilateral tumor involving the peripheral zones of the bilateral base and mid-portions of the prostate with extension into the central gland on the left. (Figures 1 and 2b) This was confirmed on **diffusion weighted imaging (DWI)** with calculation of **apparent diffusion coefficient maps (ADC)**. (Figures 2a and 2b) In addition, direct invasion of the seminal vesicles bilaterally, left side greater than right, was also demonstrated with associated hematospermia on the left. (Figures 3a, 3b, and 3c) The previously identified left obturator lymph node was also re-identified and noted to be mildly enlarged with restricted diffusion, remaining suspicious for regional nodal metastasis. (Figure 4)

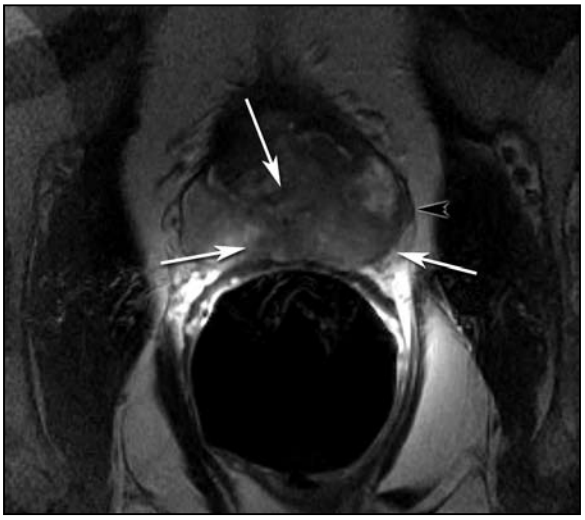


Figure 1. Axial T2 weighted image of the prostate using an endorectal coil at the level of the mid-gland showing hypointense tumor (white arrows) bilaterally, left side larger than right, extending to the prostatic capsule on the left with focal extracapsular extension laterally on the left (black arrowhead). There is also extension into the central gland on the left.

While surgical resection remains the most widely accepted curative treatment option for those with organ-confined disease, if there is evidence of extracapsular extension (T3 disease), surgery may result in an increased risk of incomplete resection, higher likelihood of micrometastatic disease elsewhere in the body, and higher morbidity. High-risk patients, therefore, especially those with evidence of seminal vesicle invasion or nodal metastases, are better served with alternative therapies such as external beam radiation and/or brachytherapy, hormonal therapy, and/or chemotherapy. For high-risk patients such as this, an MRI of the prostate gland using both a torso phased array coil and an endorectal coil remains the most sensitive imaging technique for local staging to guide Urologic management between primary surgical resection versus other less invasive treatment options. The use of an endorectal coil is essential to obtain adequate signal and spatial resolution to resolve and stage tumors of the prostate. Traditionally, this has involved the use of a small field of view with high resolution T2 weighted images obtained in the axial, coronal, and sagittal planes where tumors appear hypointense upon a background of the normally hyperintense peripheral zone. Extracapsular extension is evident when there is disruption of the prostatic capsule, asymmetry in the neurovascular bundle, seminal vesicle invasion, or invasion into adjacent organs such as the bladder or rectum. While T2 signal hypointensity can also be seen with nonmalignant findings such as hemorrhage, prostatitis, and

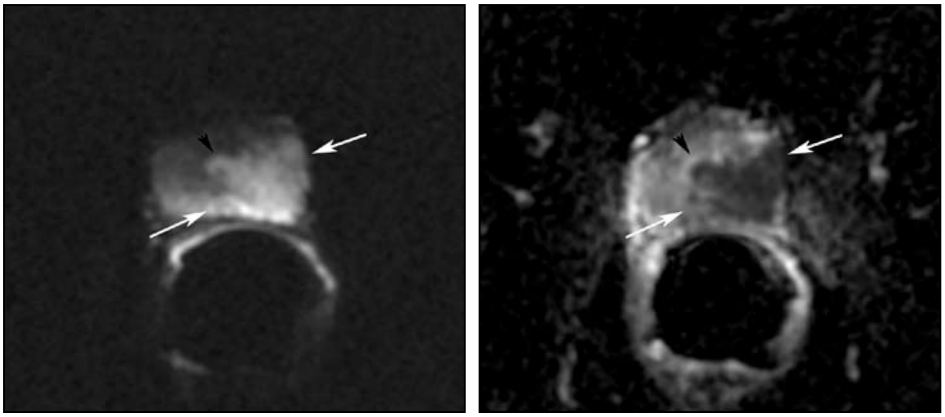


Figure 2. Diffusion weighted imaging of the prostate at the level of the mid-gland. a. (left) Diffusion weighted imaging with a b-value of 1000. b. (right) Apparent Diffusion Coefficient (ADC) map at the same level. Tumors, especially those of higher Gleason grade, show increased signal on DWI and correspondingly decreased values on ADC map (white arrows). Note the increased conspicuity of central gland involvement compared to T2 weighted images (black arrowhead).

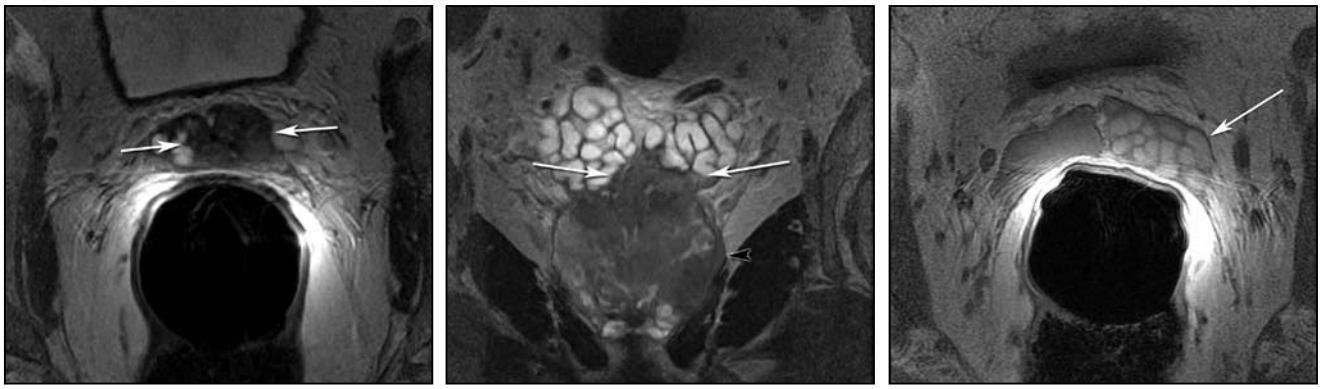


Figure 3. Seminal vesicle invasion on T2 weighted imaging. a. (left) Axial T2 weighted image at the level of the seminal vesicles shows bilateral hypointense tumor extension (white arrows). b. (center) Coronal T2 weighted image shows left sided seminal vesicle invasion is greater than on the right (white arrows). Also note the other focal area of extracapsular extension seen in Figure 1 (black arrowhead). c. (right) Axial T1 weighted image at the level of the seminal vesicles shows hyperintense signal within the seminal vesicles on the left (white arrow) consistent with hemorrhage and hematospermia which is not an uncommon finding following a prostate biopsy.

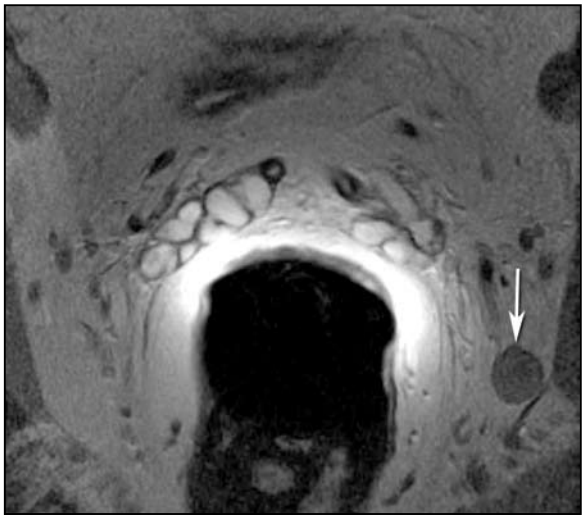


Figure 4. Axial T2 weighted image demonstrating an enlarged 12 mm left obturator lymph node. This showed restricted diffusion on DWI and ADC (not shown) raising suspicion for nodal metastasis.

changes following hormone or radiation therapy, correlation with T1 weighted images as well as the use of other functional techniques such as diffusion weighted imaging, MR spectroscopy, and dynamic contrast-enhanced MRI greatly aid in improving specificity.[1] As in this case, tumors that show higher signal on DWI and correspondingly lower ADC values also tend to correlate with a higher Gleason grade on histology.[2]

## References


1. Verma S, Rajesh A. A clinically relevant approach to imaging prostate cancer: review. *Am J Roentgenol*. 2011 Mar;196(3 Suppl):S1–10.
2. Woodfield CA, Tung GA, Grand DJ, Pezzullo JA, Machan JT, Renzulli JF 2nd. Diffusion-weighted MRI of peripheral zone prostate cancer: comparison of tumor apparent diffusion coefficient with Gleason score and percentage of tumor on core biopsy. *Am J Roentgenol*. 2010 Apr;194(4):W316–22.



*Kevin J. Chang, MD is Assistant Professor of Diagnostic Imaging, Warren Alpert Medical School of Brown University.*

*Courtney A. Woodfield, MD is an Attending Radiologist at Diagnostic Imaging, Inc. based in Philadelphia, PA.*


### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


Kevin J. Chang, MD
Deptarment of Diagnostic Imaging
Rhode Island Hospital
593 Eddy St.
Providence, RI 02903
phone: (401) 444-5184
e-mail: kchang@lifespan.org

# Nodular Pulmonary Sarcoidosis

*Eyad Kawar, MD, Sajid Y. Saraf, MD, Sidney S. Braman, MD, and Terrance T. Healey, MD*

**Sarcoidosis is a multiorgan disease that** most commonly affects adults in the twenty to forty year age group. Typically symptoms are referable to the lungs and a chest radiograph is obtained. The radiographic findings are often so typical that the diagnosis can almost be certain without further investigation. The radiographic findings characteristic of sarcoidosis are bilateral symmetrical hilar lymphadenopathy, often accompanied by right paratracheal nodes with or without parenchymal air space disease. High resolution CT scan typically shows micronodules with a perilymphatic distribution and in later stages of the disease pulmonary fibrosis.[1] The outcome of sarcoidosis in most patients is favorable except in those with extensive fibrosis. Occasionally, however, patients with sarcoidosis present with atypical findings on the chest imaging making the diagnosis more challenging. One pattern on the chest radiograph that is quite atypical for this disease has been called nodular sarcoidosis. Radiography and CT scanning show multiple nodules (less than 3 cm) or masses (more than 3 cm) that resemble metastatic cancer.

## Case report

A twenty four year old previously healthy male presented to the hospital emergency room with four day history of constant, moderate intensity, right lower chest pain that was aggravated by movement and deep breathing. He reported similar pain on his left side two days later that radiated to his left arm. He denied palpitations, dyspnea, cough, fever, diaphoresis or recent weight loss. He denied smoking cigarettes.

On examination he was found to be afebrile and hemodynamically stable. There were no palpable lymph nodes. His chest was clear to auscultation and abdominal examination was benign. External genitalia were well developed with no masses. His neurological examination was within normal limits.

He had normal blood cell counts and indices as well as serum chemistries. An electrocardiogram was within normal limits and there was no elevation of cardiac enzymes.

A plain chest x-ray revealed multiple pulmonary nodules including a 2 cm opacity in the right lower lobe, a 1.5 cm opacity in the right upper lobe and a 3.2 cm opacity in the left upper lobe. (Figure 1) Computerized tomography verified these to be parenchymal nodules of soft tissue density without calcification. There was no associated cavitation, bony erosions or pleural effusions. Multiple enlarged lymph nodes were present including a 1.3 cm anterior para-tracheal node, and a two cm subcarinal node.

Further laboratory testing revealed a sedimentation rate of 10 mm/hr and C-reactive protein concentration of 3 mg/L. A screen for HIV was negative. Anti-nuclear antibody was positive with a dilution of 1:640 with nucleolar appearance. He was negative for RA, dS DNA, SSA, SSB and Scl-70 antibodies. Anti-proteinase-3 and antimyeloperoxidase antibodies were also negative. Angiotensin converting enzyme level was slightly elevated at 70 U/L (reference range nine to 67) as were SGOT at 4I IU/L (reference range 15-35) and SGPT at 58 IU/L (reference range 13-45). Alkaline phosphatase, bilirubin and INR were within normal limits. Mycoplasma IGM antibody titer was negative as was serum cryptococcal antigen. He had a negative PPD test and the sputum did not grow **acid-fast bacilli (AFB)**, other bacteria or fungi.

A CT guided biopsy of the largest mass in the left upper lobe demonstrated multiple non-necrotizing granulomas with negative AFB, PAS and GMS stains. Based on the clinical, radiological and histological features and exclusion of other suspected conditions, a final diagnosis of Stage II Sarcoidosis was made. Since the patient did not have any significant symptoms he was prescribed analgesics, provided information about his new diagnosis and scheduled for a follow up as an outpatient.

## Discussion

The vast majority of patients with sarcoidosis present with typical radio-

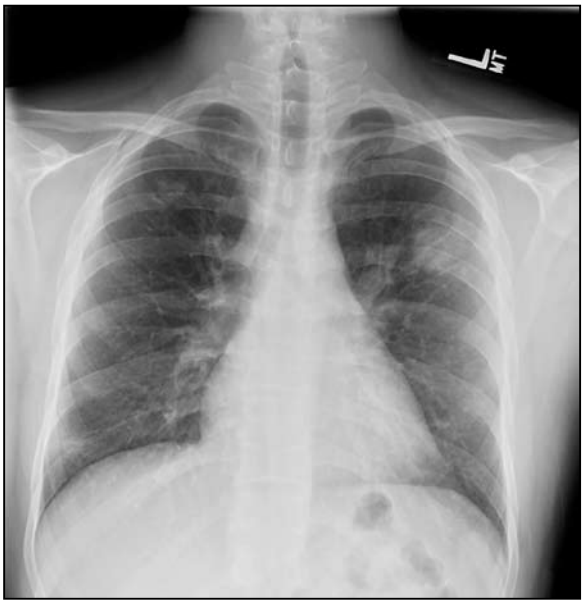


Figure 1. Chest X-ray revealed multiple opacities in the lungs including a 2 cm opacity in the right lower lobe abutting the pleura, a 1.5 cm focal density in the right upper lobe as well as a 3.2 cm rounded density in the left upper lobe.

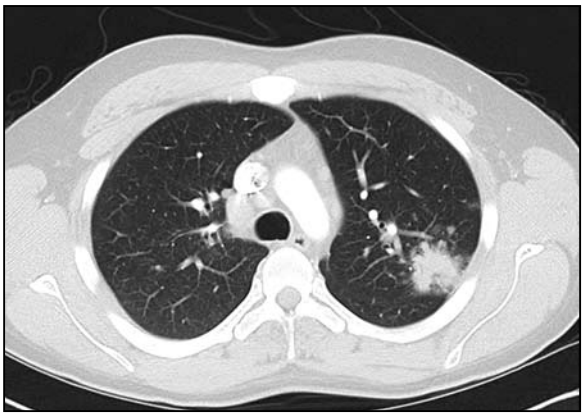


Figure 2. Computed tomography of the chest showing the largest mass surrounded with smaller nodules (sarcoid galaxy sign).

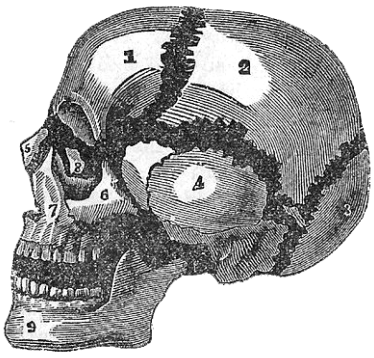
graphic findings of bilateral hilar adenopathy, right paratracheal adenopathy and interstitial lung disease. Occasionally, patients will present with an atypical findings such as seen in our case. The differential diagnosis of large nodules and masses in the lungs is extensive and includes metastatic disease to the lungs, Wegener's granulomatosis, amyloidosis, fungal infections, lymphoproliferative diseases, lymphoma, pneumoconiosis and diffuse bronchoalveolar carcinoma.[2]

Most of the available knowledge concerning nodular sarcoidosis has been gathered from case reports and small case series.[3-10] The incidence of this presentation is estimated to be approximately 1.5%. The majority of reported cases from the United States were in African-Americans, however, a review of 126 cases of pulmonary sarcoidosis in a Scandinavian cohort found a similar incidence.[11] In the largest series reported in the literature,[12] the majority of patients were females and smokers. Cough and shortness of breath were the most common presenting symptoms and chest pain was present in 51% of the patient's. Chest pain was the chief complaint and only complaint of our patient.

While nodular sarcoidosis is usually associated with multiple pulmonary nodules, occasionally a solitary mass may the only finding.[12] Since most nodules or masses are sub-pleural in location, pleural irritation results in chest pain as seen in our patient. The masses may demonstrate air bronchograms and have been reported to cavitate. These masses are likely caused by the coalescence of smaller nodules within the interstitium. Nodular sarcoidosis however is a different entity than the conglomerate masses that can be seen in the upper lobes of some patients with advanced sarcoidosis resulting from intense peribronchovascular fibrosis. The latter is complicated by traction bronchiectasis and upper lobe volume loss.

Our patient was a young male with no significant past medical history. Testicular carcinoma was considered and the negative physical exam and pelvic CT scan were reassuring. Other malignancies were also considered as a transthoracic needle biopsy of the lung was promptly performed. It showed non-caseating granulomas consistent with sarcoidosis. Patients with nodular sarcoidosis in general have a favorable prognosis. Complete resolution of the masses or nodules can be expected either spontaneously or with oral corticosteroid therapy.

In summary, the presence of multiple masses and large nodules on a chest roentgenogram is often reason for concern. The differential diagnosis includes metastatic or primary malignancy. One benign disease that must be considered is nodular sarcoidosis. This diagnosis can be confirmed bronchoscopically with a transbronchial biopsy or by a CT guided transthoracic needle biopsy of the pulmonary lesion.



## References


1. Kuhlman JE, Fishman EK, Hamper UM, et al. The computed tomographic spectrum of thoracic sarcoidosis. *RadioGraphics*. 1989;9:449–66.
2. Webb WR, Muller NL, Naidich DP eds. *High resolution computed tomography findings of lung disease* 3rd Ed. Philadelphia, Lippincott Williams and Wilkins, 2001;124–5.
3. Sharma OP, Hewlett R, Gordonson J. Nodular sarcoidosis: an unusual radiographic appearance. *CHEST.* 1973;64(2):189–92.
4. McCord MC, Hyman HL. Pulmonary sarcoidosis with the roentgenologic appearances of metastatic neoplasm; a report of two cases. *Am J Roentgenol Radium Ther Nucl Med*. 1952;67(2):259–62.
5. Nakamura H, Kashiwabara K, Watanabe T, Yagyu H, Kiguchi T, Matsuoka K. Sarcoidosis with multiple nodular shadows in bilateral lung fields. *Intern Med*. 1995;11:1144–5.
6. Onal E, Lopata M, Lourenco RV. Nodular pulmonary sarcoidosis. Clinical, roentgenographic, and physiologic course in five patients. *CHEST.* 1977;72(3):296–300.
7. Rose RM, Lee RG, Costello P. Solitary nodular sarcoidosis. *Clin Radiol*. 1985;36:589–92.
8. Kurogouchi F, Fujimori Y, Oharazawa A, Nagasaki M, Miyazawa K, Sano K, Kubo K, Kiyosawa K. Sarcoidosis associated with multiple large pulmonary nodules. *Respirology*. 1998;3:273–6.
9. McCullough PC, McCullough AE. Nodular sarcoidosis. *N Engl J Med*. 2002;346:1970
10. Nutting S, Carr I, Cole FM, Black LL. Solitary pulmonary nodules due to sarcoidosis. *Can J Surg*. 1979;22:584–6.
11. Romer FK. Sarcoidosis with large nodular lesions simulating pulmonary metastases. An analysis of 126 cases of intrathoracic sarcoidosis. *Scand J Respir Dis*. 1977;58(1):11–6.
12. Malaisamy S, Dalal B, Bimenyuy C, Soubani A. The clinical and radiologic features of nodular pulmonary sarcoidosis. *Lung*. 2009;187:9–15.



*Eyad Kawar, MD, is a Teaching Fellow in Pulmonary and Critical Care Medicine at the Warren Alpert School of Medicine at Brown University.*

*Sajid Y. Saraf, MD, is a PGY III Internal Medicine Resident at Memorial Hospital.*

*Sidney S. Braman, MD, FCCP, is Professor of Medicine at the Warren Alpert Medical School of Brown University, and Rhode Island Hospital.*

*Terrance T. Healey, MD, is Director of Thoracic Radiology at Rhode Island Hospital and Clinical Assistant Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University.*


### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


Eyad Kawar
593 Eddy St.
Providence RI 02903
e-mail: eyad_kawar@brown.edu

# Cardiac CT Angiography Findings In Paradoxical Coronary Artery Embolism

*Michael K. Atalay, MD, PhD, and Athena Poppas, MD*

**An active 33-year-old male with a** history of hyperlipidemia, hypertension, and Wolff-Parkinson-White syndrome, presented to the emergency department with diaphoresis and acute onset 3/10 substernal chest pain triggered during heavy lifting. The pain was relieved with nitroglycerin and morphine. EKG showed sinus bradycardia (58 bpm) and less than one mm ST elevation in an inferior lead. The first troponin value was normal, but acute coronary syndrome remained a concern and the patient was started on enoxaparin, clopidogrel, aspirin, a statin, and nitrates—the latter for possible vasospasm. A **cardiac CT angiogram (CCTA)** showed no evidence of coronary artery disease or pulmonary embolism. (Figure 1a) However, a small hypoperfusion defect was present in the distal inferior left ventricular wall. (Figure 1b) Moreover, because retrospective ECG-gating was employed, segmental wall motion analysis could be assessed. This demonstrated focal hypokinesis of the underperfused segment. (Figures 1c & 1d)

The second troponin value was elevated (3.0 ng/ml), and the patient underwent cardiac catheterization for definitive coronary artery assessment. While atherosclerotic stenosis was again excluded, angiography did reveal an abrupt cut-off of the 1 mm-diameter terminus of a long, apical **left anterior descending coronary artery (LAD)**, suggestive of embolism. (Figure 2) Due to the small vessel size no intervention was performed. However, based on these findings, a bubble echocardiogram was performed. This confirmed right to left blood flow across a **patent foramen ovale (PFO)** during Valsalva maneuver. Hypercoagulable work-up and toxicology screen were normal. Paradoxical embolus remained the presumptive diagnosis, and percutaneous closure of the PFO was subsequently performed. (Figure 3)

## Discussion

The foramen ovale is formed by the overlap of the primum and secundum membranes that constitute the interatrial septum. During fetal life, the opening permits shunting of oxygenated umbilical venous blood from the **right atrium (RA)** to the **left atrium (LA)**, bypassing the non-inflated lungs. In most individuals, the membranes fuse after birth. However, in 25-30% of adults the foramen ovale remains patent[1] and can serve as a potential right-to-left shunt and a source of paradoxical emboli (described below).

LA pressure usually slightly exceeds RA pressure and ensures that the PFO—when present—remains closed. Transient increases in RA pressure can occur in normal individuals (e.g. Valsalva maneuver, coughing), opening the PFO and briefly shunting blood to the LA. This provides a short-lived opportunity for a small embolus traveling in venous circulation—and otherwise destined for the lungs—to crossover into the arterial circulation where it will eventually lodge in a small, peripheral vessel and cause end-organ ischemia. This is the paradoxical embolus. While PFO has been associated with cryptogenic embolic stroke, transient ischemic attacks, and migraine headaches,[2] causation has not been proven. Its association with other systemic embolic events such as myocardial infarction is rare. In a series of 416 patients referred for evaluation of PFO related conditions, only eight (3.6%) had acute MI without evidence of obstructive CAD on angiography.[3]

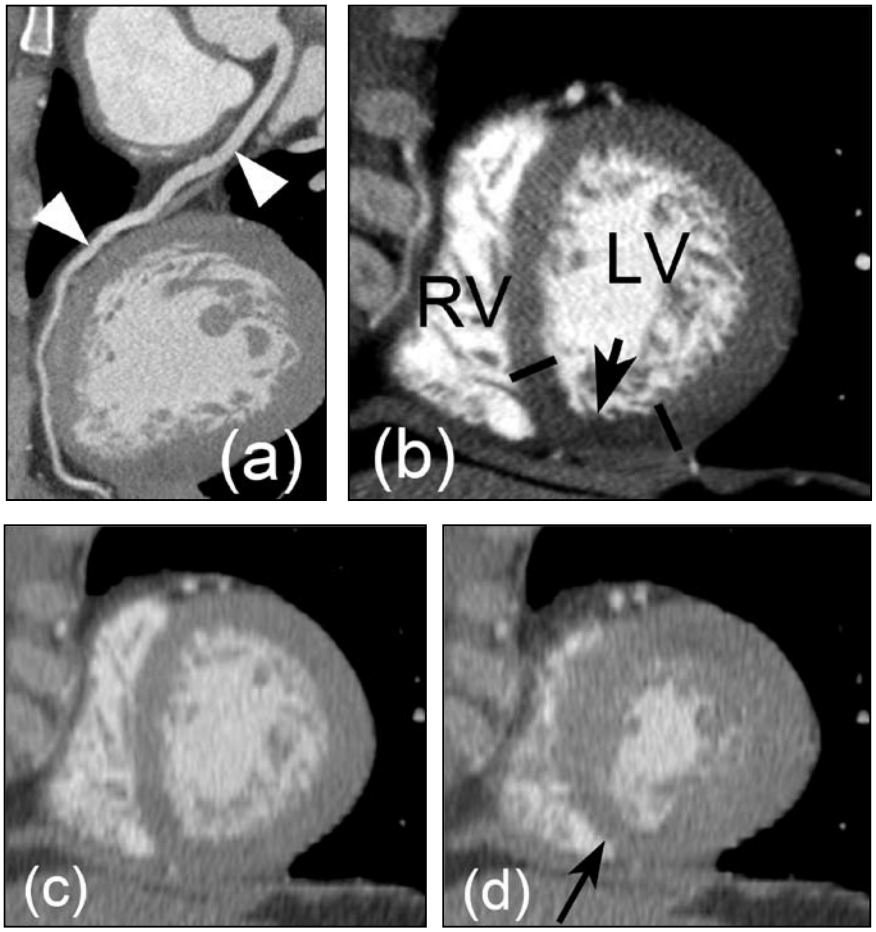



Figure 1. (a) Curved reconstructed image from the CCTA shows a normal LAD (arrowhead). (b) Short axis (SA) CT image shows a small focus of low density (relative darkness, arrow) in the inferior LV due to hypoperfusion. Small black bars delineate this segment. (c-d) End-diastolic and end-systolic phase SA CT images show lack of normal thickening of the underperfused segment (arrow).

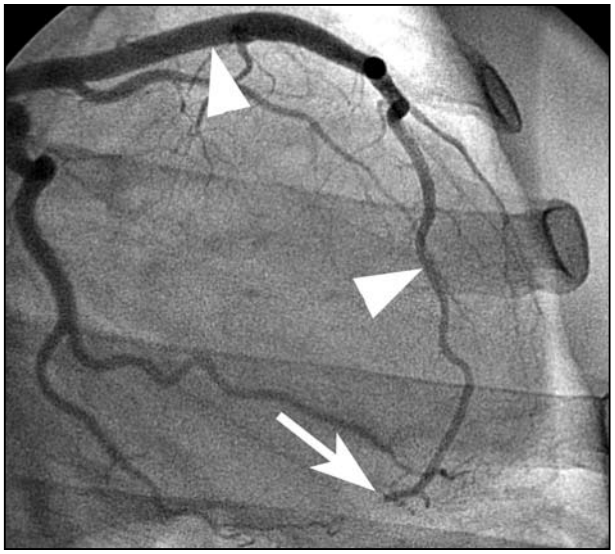


Figure 2. Single view from the conventional angiogram shows LAD (arrowheads) with an abrupt distal cut-off (arrow) suggestive of embolus.

Paradoxical embolism is a diagnosis of exclusion, invoked when other explanations for the observed systemic events are disproved. Such is our case where a young, healthy man with no CAD had a myocardial infarction, a PFO, and no other explanation. Three imaging modalities were employed to render this diagnosis and ultimately manage his treatment.

Echocardiography and conventional angiography remain the gold standard of anatomical and functional cardiac assessment. However, recent advances in computed tomography technology now permit non-invasive, highly detailed, stop-action imaging of the heart. With a spatial resolution of ~0.5 mm, CCTA now provides accurate imaging of the proximal coronary arteries. CCTA has a high negative predictive value for the detection of significant coronary artery disease in selected populations (~99%) and has emerged as a useful tool in the assessment of low to intermediate risk patients with chest pain or equivocal stress tests. Its use in the setting of coronary embolism is not established. In our case, the angiographic portion of the CTA examination was falsely negative owing to the small, distal nature of the affected segment, but the secondary signs of myocardial injury were evident on the perfusion and wall motion portions of the same examination.

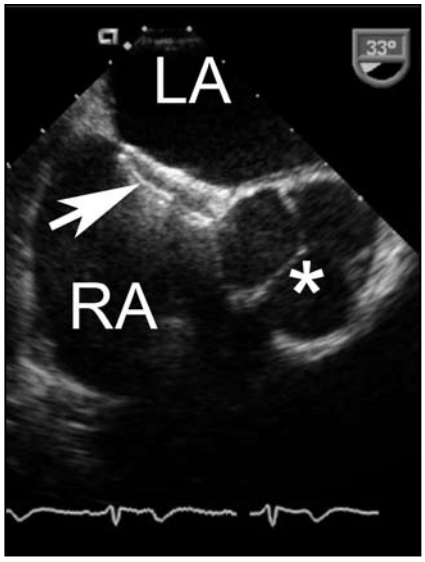



Figure 3. Transesophageal echocardiogram shows the echogenic (bright) closure device (arrow) along the interatrial septum occluding the PFO. Asterisk: aortic valve.

## References

1. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc.* 1984;59:17–20.
2. Lamy C, Giannesini C, Zuber M, et al. Clinical and imaging findings in cryptogenic stroke patients with and without patent foramen ovale: the PFO-ASA Study. Atrial Septal Aneurysm. *Stroke.* 2002;33:706–11.
3. Dao CN, Tobis JM. PFO and paradoxical embolism producing events other than stroke. *Catheter Cardiovasc Interv.* 2011.

*Michael K. Atalay, MD, PhD, is an Assistant Professor Department of Diagnostic imaging at the Warren Alpert School of Medicine of Brown University.*

*Athena Poppas, MD, is an Associate Professor Department of Medicine at the Warren Alpert School of Medicine of Brown University.*

### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence

Michael K. Atalay, MD, PhD, FSCCT
phone: (401) 444 - 5184
fax: (401) 444 - 5017
e-mail: atalay_99@yahoo.com

# Cardiac Magnetic Resonance Imaging of Burned-out Hypertrophic Cardiomyopathy

*Michael K. Atalay, MD, PhD, and Franklin Schneider, MD*

**A 52 YEAR-OLD MAN WITH CHRONIC** obstructive pulmonary disease and no prior cardiac history presented with new onset **congestive heart failure** (**CHF**). Echocardiography revealed severely depressed global **left ventricular** (**LV**) function and mildly reduced **right ventricular** (**RV**) function. His estimated pulmonary artery pressure was 80 mmHg, consistent with severe pulmonary hypertension. EKG revealed normal sinus rhythm with right bundle branch block. The patient was referred for **cardiac magnetic resonance imaging** (**CMR**) to evaluate the etiology of the **cardiomyopathy** (**CM**).

CMR confirmed severe global LV dysfunction with an ejection fraction of 15% and moderate RV dysfunction. While both the LV and RV free walls were mildly thickened, the interventricular septum was markedly thickened measuring 2.7 cm (normal ≤ 1.2 cm). (Figure 1a) Post-contrast imaging demonstrated patchy predominantly subepicardial enhancement throughout the LV, septum and apical RV consistent with fibrosis. (Figure 1b) This pattern of enhancement is non-specific, but excluded an ischemic CM where scar is sub*endo*cardial. The pattern is not typical for cardiac amyloid.[1] Though other non-ischemic etiologies such as cardiac sarcoid were considered, in light of the myocardial thickening, **hypertrophic CM** (**HCM**) was favored. Subsequent endomyocardial biopsy demonstrated myocyte disarray and hypertrophy, as well as areas of fibrosis, confirming HCM. (Figure 1c)

## Discussion

HCM is a genetic disease of the cardiac sarcomere with an autosomal dominant inheritance pattern typically showing a high degree of penetrance. It is a relatively common disease occurring in up to 0.29% of individuals[2] and is among the most common cardiac lesions found in athletes with sudden cardiac death. Morphologically, several variants have been described: septal hypertrophy with or without LV outflow tract obstruction, concentric hypertrophy, apical hypertrophy, LV free wall hypertrophy, and RV hypertrophy. At histology myocardial disarray may be seen. Additionally, myocardial fibrosis is considered a hallmark of HCM and a potential substrate for arrhythmias and heart failure.[3]

CMR has emerged as a powerful tool for evaluating a host of non-ischemic cardiomyopathies, including HCM.[4] In addition to providing accurate quantification of wall thickness, chamber volumes and ejection fraction, assessment of regional and global wall motion, and quantification of valve lesion severity, CMR has the ability to characterize tissue pathology. It permits detection of edema, infarction, inflammation, and fibrosis. In our case, substantial fibrosis was detected in a non-infarct distribution confirming a non-ischemic etiology. It is proposed that the extensive fibrosis contributed to the marked LV dysfunction and eventual CHF observed in this patient.

## References

1. Syed IS, Glockner JF, Feng D, et al. Role of cardiac magnetic resonance imaging in the detection of cardiac amyloidosis. *JACC Cardiovasc Imaging.* 2010;3:155–64.
2. Maron BJ, Mathenge R, Casey SA, Poliac LC, Longe TF. Clinical profile of hypertrophic cardiomyopathy identified de novo in rural communities. *J Am Coll Cardiol.* 1999;33:159–5.
3. Ho CY, Lopez B, Coelho-Filho OR, et al. Myocardial fibrosis as an early manifestation of hypertrophic cardiomyopathy. *N Engl J Med.* 2010;363:552–63.
4. Hendel RC, Patel MR, Kramer CM, et al. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 appropriateness criteria for cardiac computed tomography and cardiac magnetic resonance imaging. *J Am Coll Cardiol.* 2006;48:1475–97.

*Michael K. Atalay, MD, PhD, is an Assistant Professor of Diagnostic Imaging at the Warren Alpert School of Medicine of Brown University*

*Franklin Schneider, MD, is a Clinical Assistant Professor of Medicine at the Warren Alpert School of Medicine of Brown University.*

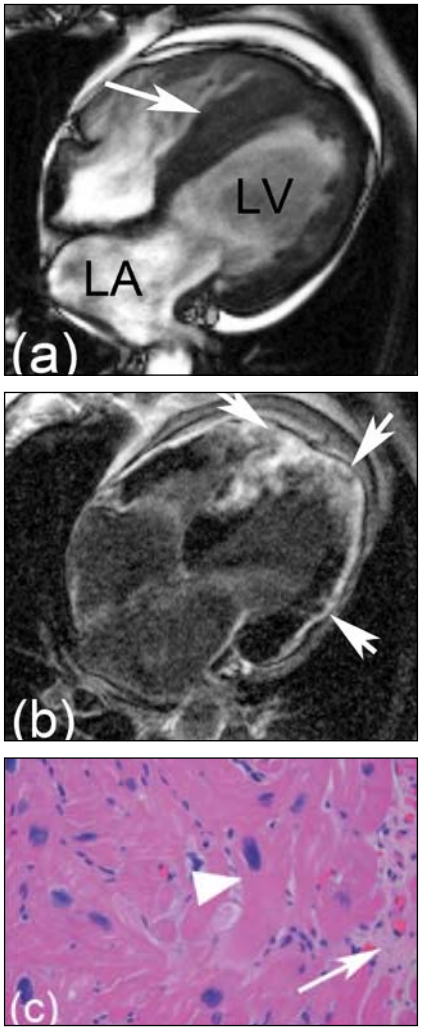



Figure 1. (a) Four-chamber bright-blood CMR image shows abnormal ventricular thickening, chiefly involving the interventricular septum (arrow). (b) Post-contrast imaging demonstrates extensive enhancement throughout the septum, distal right ventricle, and subepicardial left ventricular free wall (arrows). (c) Histologic specimen with H&E stain shows myocyte disarray, fibrosis (arrow), and hypertrophy (arrowhead). LA: Left atrium; LV: Left ventricle.

### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence

Michael K. Atalay, MD, PhD, FSCCT
phone: (401) 444 - 5184
fax: (401) 444 - 5017
e-mail: atalay_99@yahoo.com

# Non-small Cell Lung Cancer With Unsuspected Distant Metastasis To the Kidney Seen on PET/CT

*Marilyn Barry-Brooks, MD, Don C. Yoo, MD, Michael Chaump, MD, and Richard B. Noto, MD*

**A 72-year-old man with a 40-pack-**year history of smoking presented to his primary care physician with a three-week history of hoarseness. Physical examination at the time of presentation was remarkable only for hoarseness. The patient was treated with a trial of inhalers and continued to have symptoms.

## Diagnosis

A CXR was performed at an outside institution that reported no evidence of an infectious process in the lungs. The patient subsequently had a chest CT scan that showed a five cm left upper lobe mass invading the mediastinum and likely involving the recurrent laryngeal nerve resulting in the patient's hoarseness. (Figure 1) CT guided biopsy of the mass was performed which showed clusters of atypical cells on histology consistent with **non-small cell lung cancer (NSCLC)** with adenocarcinoma favored. (Figure 2) The patient then underwent a **positron emission tomography/computed tomography (PET/CT)** scan with **18F-fluorodeoxyglucose (F-18 FDG)** for staging. **Maximum intensity projection (MIP)** image (Figure 3) and axial images (Figure 4) from the PET/CT scan showed intense uptake in the lung mass. There was no evidence of metastatic disease in the mediastium or hilar regions. However, there was an intense focus of increased activity in the lower pole of the left kidney (MIP image in Figure 3 and coronal reformats in Figure 5) which was suspicious for metastatic disease and the patient had a contrast enhanced renal MRI for further characterization. MRI showed a solid mass in the lower pole of the left kidney (Figure 6) corresponding to the abnormality on the PET/CT scan. An ultrasound-guided biopsy was performed which showed glandular like clusters of cells on histology that were morphologically similar to those seen in the fine needle aspirate of the lung. In conjunction with the clinical scenario, the kidney mass was diagnosed as poorly differentiated adenocarcinoma consistent with metastatic lung adenocarcinoma. (Figure 7) The patient was treated with chemotherapy and also received radiation therapy to the lung mass but died within a year from his diagnosis.

## Discussion

This case highlights the utility of PET/CT in the staging of NSCLC. NSCLC accounts for 75-80% of lung cancer related deaths in the western hemisphere. Accurate staging is needed to make treatment decisions including whether or not the patient is a surgical candidate.[1] Staging of NSCLC is based on location and size of the tumor, the degree of nodal involvement, and the presence of intrathoracic and extrathoracic metastases. It is important to determine the presence or absence of mediastinal disease.[2]

CT is usually the initial imaging modality used for the staging of NSCLC. The limitation of CT is that it is based on morphologic criteria, i.e. suspicious lymph nodes are identified by size criteria of being greater than 1 cm in short axis. However, lymph nodes smaller than 1 cm can have metastatic involvement and enlarged lymph nodes may be due to etiologies other than neoplasm. One study demonstrated that up to 44% of metastatic lymph nodes in patients with NSCLC measured less than 1 cm.[3]

PET/CT has become an established and readily available imaging modality for staging and restaging of patients with NSCLC. PET/CT using F-18 FDG combines information regarding the physiologic activity of a tumor (as most cancer cells have higher metabolic activity and accumulate more F18-FDG compared to normal cells) with the anatomic finding on CT. PET/CT has a sensitivity of 74% and specificity of 85% for identifying

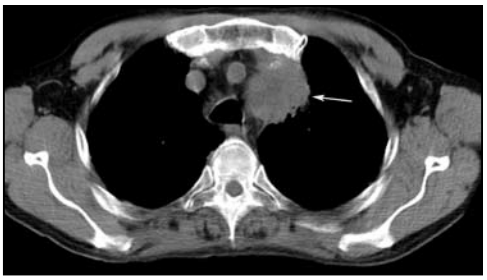


Figure 1. Noncontrast CT image shows a heterogeneous lung mass (white arrow) with direct extension into the anterior mediastinum and encasement of the left subclavian artery and left common carotid artery.

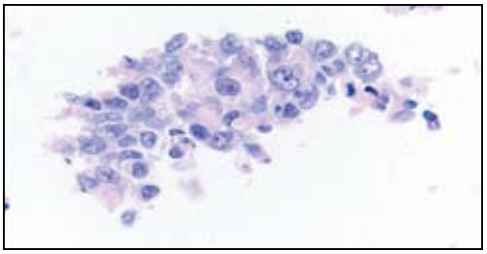


Figure 2. Microscopic image of the fine needle aspirate cell block from the left upper lobe lung mass, showing clusters of atypical cells (400x). Hematoxylin and Eosin stain.

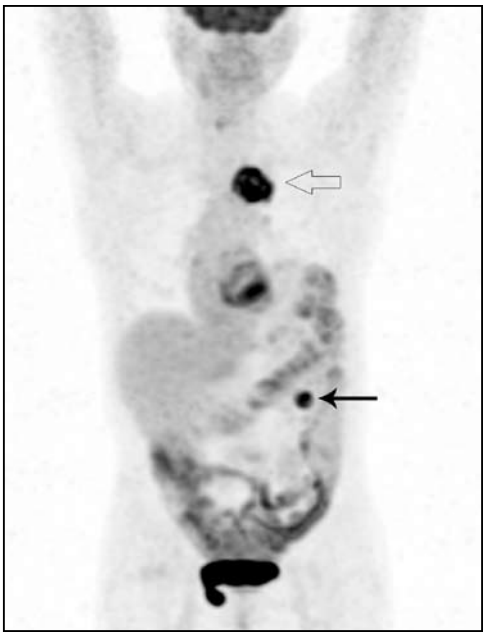


Figure 3. MIP image from the F18-FDG PET/CT scan shows intense uptake in the lung mass invading the mediastinum (open arrow) and a discrete round focus of intense uptake in the left kidney (closed arrow).

mediastinal metastases versus CT with a sensitivity of 51% and a specificity of 86% when compared with mediastinoscopy or surgical staging.[4] In addition, PET/CT can aid in the detection of intrathoracic and extrathoracic metastases. PET/CT has been shown to contribute to upstaging of patients primarily by the identification of extrathoracic metastases resulting in 20% fewer unnecessary thoracotomies.[5] A limitation of PET/CT is the low metabolic activity of some types of lung cancers such as adenocarcinoma in situ and primary lung carcinoid which can make the evaluation of metatastic lymph node involvement and distant metastatic disease more difficult.

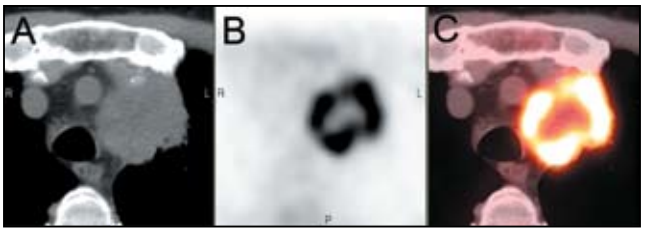



Figure 4. Axial CT (A), PET (B), and fused PET/CT (C) images show intense uptake around the periphery of the left upper lobe mass. The central region of the mass was photopenic consistent with central necrosis.

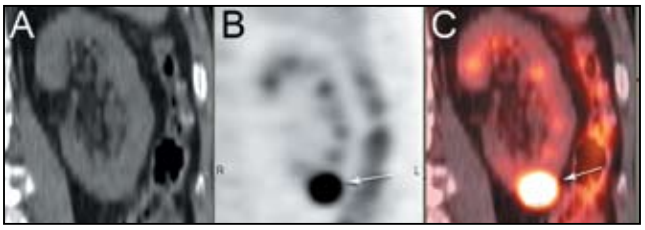



Figure 5. Axial CT (A), PET (B), and fused PET/CT (C) images show focal intense uptake in the lower pole of the left kidney (white arrows) without an unenhanced CT correlate.

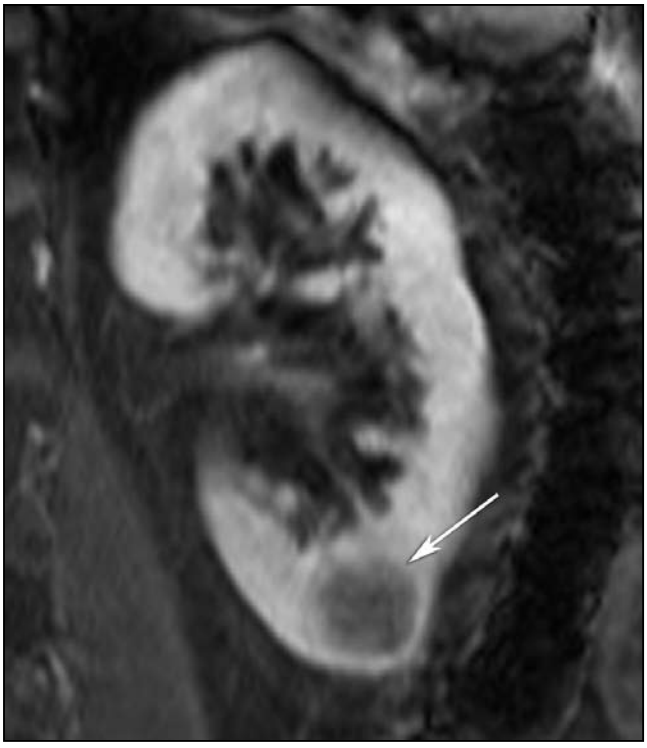


Figure 6. Postcontrast T1 weighted coronal MR image of the left kidney show a solid mass in the lower pole with mild peripheral nodular enhancement corresponding to the focus of intense uptake on the PET/CT scan.

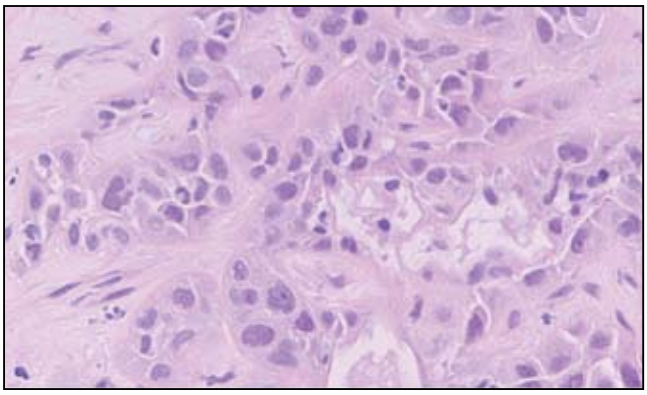


Figure 7. Microscopic image of the left lower pole kidney biopsy showing glandular like clusters of atypical cells (400x) morphologically similar to those seen in the fine needle aspirate of the lung. Hematoxylin and Eosin stain.

This case shows a very unusual unsuspected isolated distant site of metastatic disease to the left kidney detected on PET/CT in a patient with absent mediastinal or hilar metastatic adenopathy. The primary method of excretion of F18-FDG is through the kidneys with 40% of the administered dose excreted by the kidneys in the first two hours. Evaluation of primary renal malignancies on PET/CT is limited due to the high background activity in the kidneys that can obscure uptake in primary renal cell cancers especially as most renal cell cancers have relatively low metabolic activity on PET/CT. Metastatic disease to the kidneys from lung cancer is uncommon. However, when it occurs, it typically demonstrates intense activity on PET/CT.[6]

## Conclusion

This patient had a PET/CT for the staging of a left upper lobe NSCLC that did not show evidence of metastatic adenopathy in the chest but showed an unexpected suspicious focus of intense activity in the left kidney which on biopsy represented a site of distant metastatic disease. This patient was upstaged from a stage 3A to a stage 4 lung cancer based on the PET/CT, altering the patient's prognosis and treatment.

## References


1. Devaraj A, Cook GJR, et al. PET/CT in non-small cell lung cancer staging-promises and problems. *Clinical Radiology.* 2007;62: 97–108.
2. Nair A, Klussman MJ, et al. Revisions to the TNM staging of non-small cell lung cancer: rationale, clinicoradiologic implications, and persistent limitations. *RadioGraphics.* 31:215–38.
3. Prenzel KL, Monig SP, Sinning JM, et al. Lymph node size and metastatic infiltration in non–small cell lung cancer. *Chest.* 2003;123:46–7.
4. Silvestri, GA, Gould MK, et al. Noninvasive staging of non-small cell lung cancer. *Chest.* 2007;132:178S-201S.
5. Van Tinteren H, Hoekstra OS, et al. Effectiveness of positron emission tomography in the preoperative assessment of patients with suspected non-small cell lung cancer: the PLUS multicentre randomized trial. *Lancet.* 2002; 359:1388–92.
6. Kochhar R, Wong CO, et al. Role of FDG PET/CT in imaging of renal lesions. *J Med Imag Rad Oncol.* 2010;54:347–57.

*Marilyn Barry Brooks, MD, is a Diagnostic Radiology Resident at the Warren Alpert Medical School of Brown University.*

*Don C. Yoo, MD, is Assistant Professor of Diagnostic Imaging (Clinical) at the Warren Alpert Medical School of Brown University.*

*Michael Chaump, MD, is a Pathology Resident, The Warren Alpert Medical School of Brown University.*

*Richard B. Noto, MD, is Associate Professor of Diagnostic Imaging (Clinical), The Warren Alpert Medical School of Brown University.*

**Disclosure of Financial Interests**

The authors and/or significant others have no financial interests to disclose.

**Correspondence**

Marilyn Barry-Brooks, MD
Department of Diagnostic Imaging
Rhode Island Hospital
593 Eddy Street
Providence, RI 02903
e-mail: mbarry@lifespan.org

# Recurrence of Lung Cancer After Radiofrequency Ablation Detected by PET/CT and Contrast Enhanced CT Scan

***Albert A. Scappaticci, MD, PhD, and Don C. Yoo, MD***

**82-year-old female with a history of** cough for three months had a CT of the chest which revealed a 1.5 cm right upper lobe pulmonary nodule. (Figure 1) The patient underwent a CT guided percutaneous biopsy which on histology was consistent with adenocarcinoma of the lung. The patient then underwent a **positron emission tomography/computed tomography (PET/CT)** scan (Figure 2) with **18F-fluorodeoxyglucose (F-18 FDG)** for staging which showed intense uptake in the right upper lobe adenocarcinoma without evidence of metastatic disease. The patient was not a surgical candidate due to poor pulmonary reserve and subsequently underwent percutaneous **radiofrequency ablation (RFA)** of the right upper lobe adenocarcinoma. After RFA, a follow-up PET/CT (Figure 3) six months later showed curvilinear activity around the ablation site with slightly more intense activity anteriorly. A contrast-enhanced CT scan (Figure 4) showed focal enhancement in the anterior aspect of the ablation cavity corresponding to the area of more focal intense activity seen on the follow-up PET/CT. Percutaneous CT guided biopsy of this region confirmed suspicion of residual or recurrent adenocarcinoma. The patient was retreated with RFA. (Figure 5) Repeat CT scan (Figure 6) performed at four months after reablation showed no evidence of residual tumor (not shown).

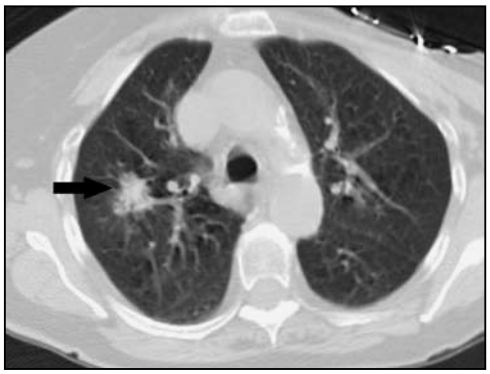


Figure 1. Noncontrast CT image show a spiculated 1.5 cm lung nodule in the right upper lobe (arrow).

## Discussion

Radiofrequency ablation of lung cancer is an important treatment option for patients who are medically inoperable with proven safety and efficacy. RF abla-

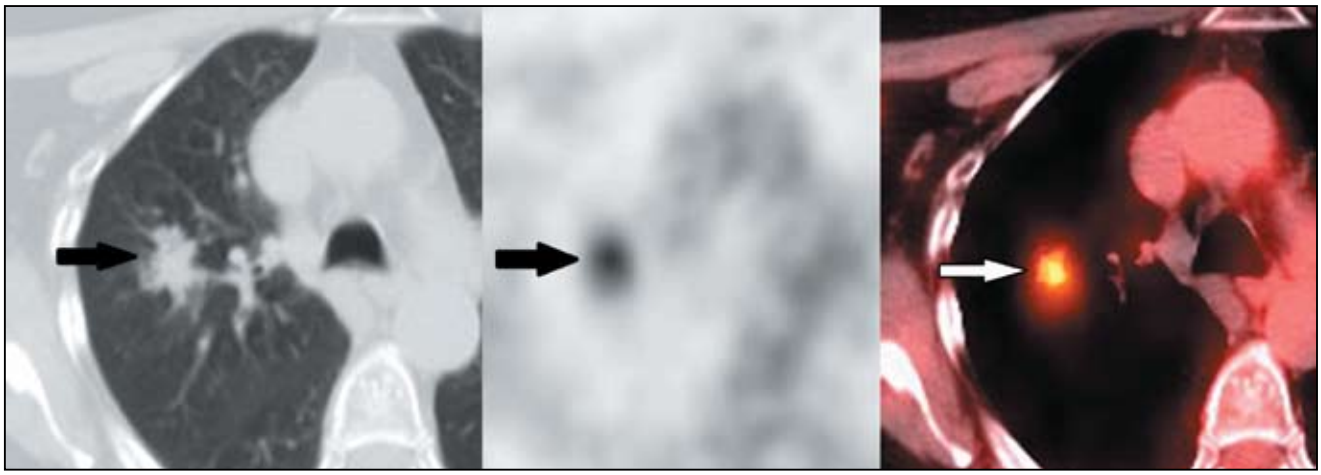


Figure 2. Preablation Axial CT (A), PET (B), and fused PET/CT (C) images show intense uptake in the right upper lobe nodule (arrows) which was histologically proven to represent an adenocarcinoma.

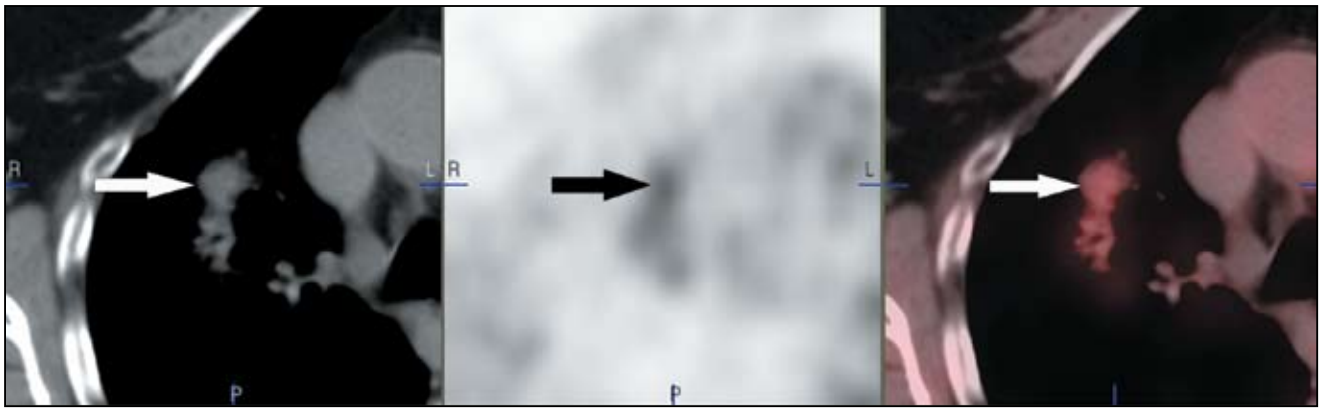


Figure 3. Axial CT (A), PET (B), and fused PET/CT (C) performed 6 months after RFA images show curvilinear activity around the ablation site with slightly more intense activity anteriorly (arrows).

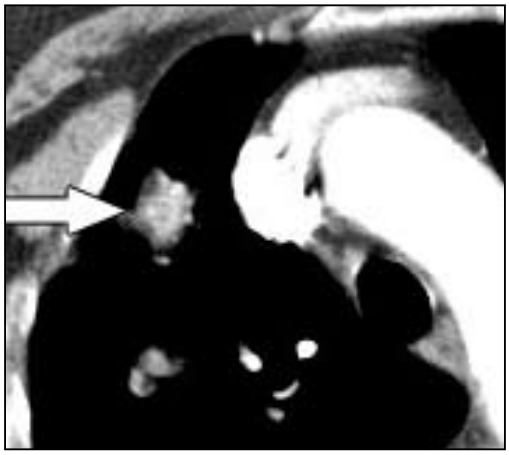


Figure 4. Contrast enhanced CT image performed 3 months after the postablation PET/CT scan shows focal enhancement in the anterior aspect of the ablation cavity (arrow) corresponding to the area of more focal intense activity seen on the prior PET/CT. This area on biopsy showed recurrence of adenocarcinoma.

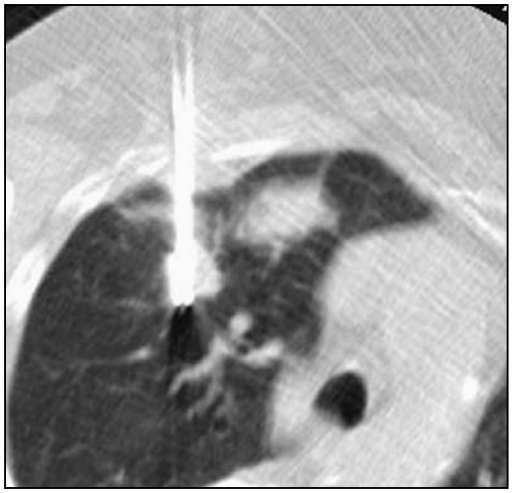


Figure 5. Noncontrast CT image shows a radiofrequency probe within the area of recurrence of adenocarcinoma.

tion of stage I **non-small cell lung cancer (NSCLC)** has a 78% one year survival and 27% five year survival as compared with 50% one year survival in patients undergoing observation alone.[1] Currently contrast enhanced CT imaging and more recently PET imaging with F18-FDG have been used to evaluate treatment success. With the increase in patients undergoing thermal ablation, appropriate imaging follow-up is needed in order to evaluate treatment response since early detection of residual tumor and recurrence can stratify patients into groups who may benefit from reablation or other therapy

PET/CT using F18-FDG is a molecular imaging modality that utilizes the radioisotope 18F coupled to glucose. When administered intravenously the radioisotope is transported into all cells of the body which actively use glucose. Cancer cells are typically more metabolically active than the native cells of the body and molecular imaging takes advantage of this differential.

PET-CT has become an invaluable modality in assessing for recurrence of patients treated with image guided percutaneous ablation of lung cancer. Recent articles demonstrate the important role of PET in following this patient population. In a recent retrospective five-year study which followed 68 patients with 94 pulmonary lesions, including metastases and primary lung cancers, researchers reviewed 18F-FDG PET/CT scans performed before and after RFA and were able to determine several indicators that could help predict local recurrence. Among pre-RFA scans, lesion size and type of tumor (primary or metastases) were factors in determining potential for local recurrence. In this study pulmonary metastases recurred less often than treated primary lung cancers and tumors smaller than three cm responded better to RFA. PET/CT scans conducted after RFA showed a variety of different uptake patterns in the ablation sites. Discretely increased focal activity along the periphery of the ablation cavity especially if this focus corresponded to the location of the original tumor was considered an unfavorable appearance and suspicious for recurrence.[2]

The optimal timing of obtaining the initial PET/CT after ablation is still being determined. In a recent prospective study of 34 lung lesions (five lesions representing primary lung cancer, 29 lesions representing metastases) follow-up post-ablation PET/CT scans were performed at one day, one month and three months after RFA. The authors concluded that PET/CT at three months after RFA may be a good time for the initial study to limit the false positive results from inflammatory uptake which occurred more frequently on the PET/CT scans performed at one day and one month after RFA.[3] More research will be necessary to determine the optimal imaging time of the initial PET/CT scan after RFA and the optimal image times for additional surveillance PET/CT scans.

CT scans performed prior to and after administration of IV contrast are also important for follow-up of patients after RFA. More recently CT densitometry evaluation for focal enhancement at the ablation site at multiple timepoints have been used for to improve the accuracy on CT for detecting recurrence.[4]

The findings in this case show the complementary role of PET/CT and contrast enhanced CT scans in the detection of cancer recurrence post treatment. The initial follow-up PET/CT scan in this patient demonstrated periphery activity with more intense uptake anteriorly which on a follow-up CT scan showed focal enhancement highly suspicious for recurrent disease which resulted in reablation of the area of recurrence. Follow-up of lung cancers after RFA with PET/CT scans and contrast enhanced CT scans can increase the accuracy of early recurrence detection allowing for reablation or other treatment leading to improved patient outcomes.

## References


1. Simon CJ, Dupuy DE, DiPetrillo TA, et al. Pulmonary radiofrequency ablation: longterm safety and efficacy in 153 patients. *Radiology*. 2007;243(1):268–75.
2. Singnurkar A, Solomon SB, Gönen M, et al. 18F-FDG PET/CT for the prediction and detection of local recurrence after radiofrequency ablation of malignant lung lesions. *J Nucl Med*. 2010;51:1833-40.
3. Deandreis D, Leboulleux S, Dromain C, et al. Role of FDG PET/CT and chest CT in the follow-up of lung lesions treated with radiofrequency ablation. *Radiology*. 2011;258:270–6.
4. Suh RD. Unresectable pulmonary malignancies: CT-guided percutaneous radiofrequency ablation—preliminary results. *Radiology*. 2003;229(3):821–9.



*Albert A. Scappaticci, MD,PhD is a Diagnostic Radiology Resident, The Warren Alpert Medical School of Brown University.*

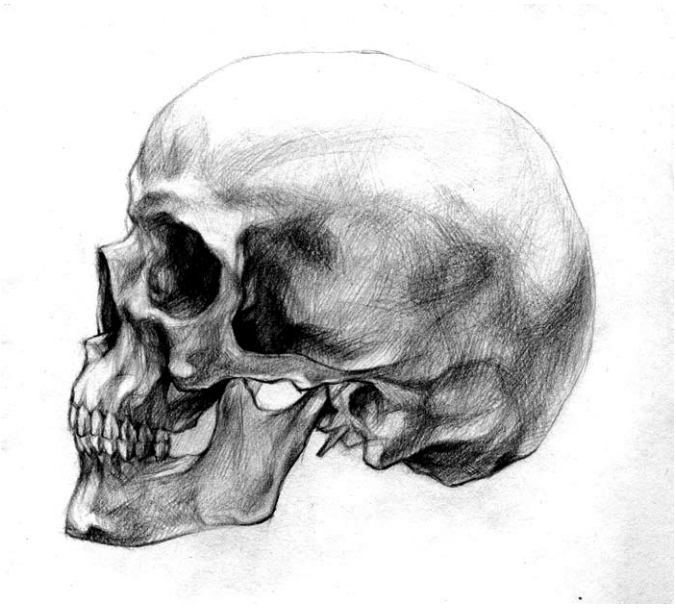
*Don C. Yoo, MD, is Assistant Professor of Diagnostic Imaging (Clinical), The Warren Alpert Medical School of Brown University.*


### Disclosure of Financial Interests

The authors and/or significant others have no financial interests to disclose.

## Correspondence


Albert A. Scappaticci, MD, PhD
Department of Diagnostic Imaging
Rhode Island Hospital
593 Eddy Street
Providence, RI 02903
e-mail: ascappaticci@lifespan.org

# Rapidly Involuting Congenital Hemangioma

*Lawrence J. Keating, MD, Gregory M. Soares, MD, and Christopher S. Muratore, MD*

**A NEWBORN MALE, DELIVERED BY CAESAREAN** section after induction at 41 weeks of gestation for failure to progress, was admitted to the **Women and Infants' Hospital (WIH) Neonatal Intensive Care Unit (NICU)** with a large vascular mass on his left knee (Figure 1). Routine prenatal US had been normal and the pregnancy had been uneventful. Physical examination at delivery was otherwise unremarkable. In addition to the WIH NICU team, the baby was followed clinically by pediatric surgical and interventional members of the Hasbro Children's **Hospital Vascular Anomalies Clinic (HVAC)**. On day two in the NICU, the patient developed signs of congestive heart failure. A chest radiograph demonstrated pulmonary edema and mild cardiomegaly (not shown). On day 14, the lesion began to bleed briskly.

## Diagnosis

**Rapidly Involuting Congenital Hemangioma (RICH)** causing high-output congestive heart failure and hemorrhage.

## Discussion

Pediatric vascular masses are classified into two basic groups: tumors, which are lesions exhibiting endothelial proliferation, and malformations, which demonstrate normal cellular and macroscopic growth with the child. A large vascular mass in a newborn is suggestive of a **congenital hemangioma (CH)**, of which two additional subtypes are described: RICH, and the less common, non-involuting congenital hemangioma, or NICH. Unlike the more common **infantile hemangioma (IH)**, which generally becomes evident at a median age of two weeks, CHs are fully developed at birth and are often preceded by abnormalities on prenatal ultrasound. They have an equal sex predilection and are most often present on the head or on a limb near a joint.[4] RICH subtype begins to involute promptly after birth and are usually completely resolved by 12–14 months of age whereas NICH will never disappear.[1, 3]

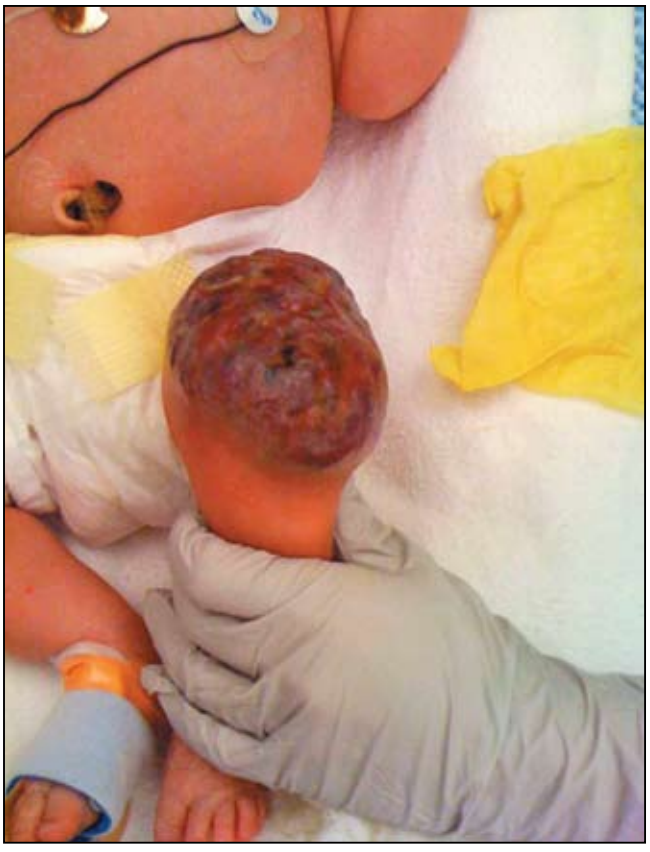


Figure 1. Left knee vascular lesion.

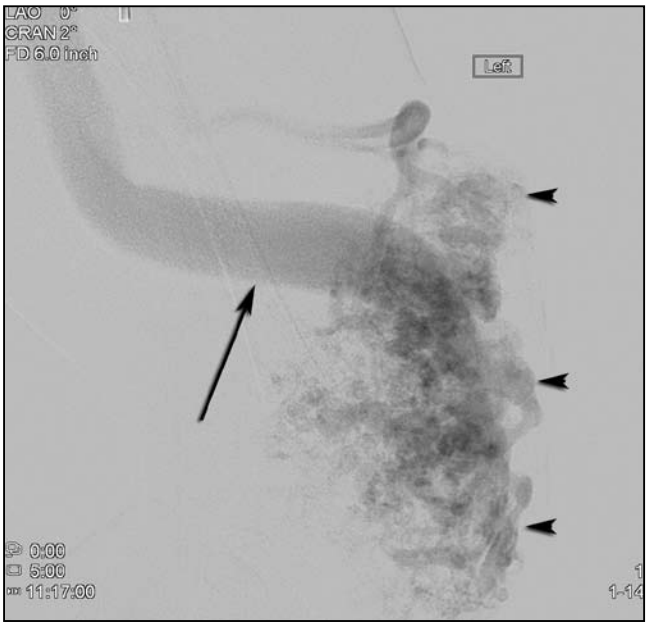



Figure 2. Tangled vessels with early drainage into dilated vein.

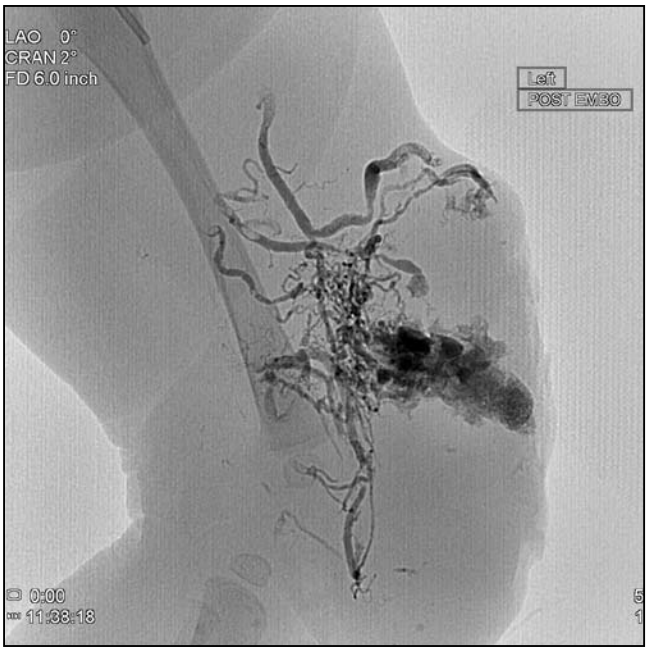
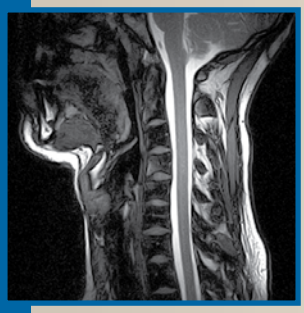



Figure 3. Post embolization; glue "casting" of the central vessels.

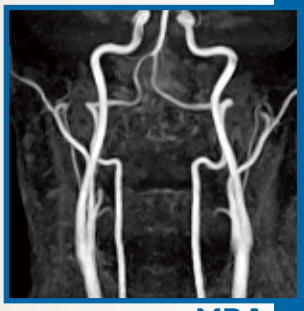
# THE IMAGING INSTITUTE

## OPEN MRI • MEDICAL IMAGING

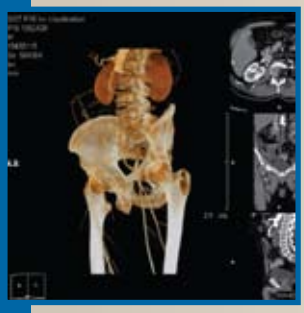


High Field MRI

- **Offering both 1.5T High Field & Higher Field OPEN MRI Systems**
- **Advanced CT with multi-slice technology, 3D reconstruction**
- **Digital Ultrasound with enhanced 3D/4D technology**
- **Digital Mammography with CAD (computer assisted diagnosis)**



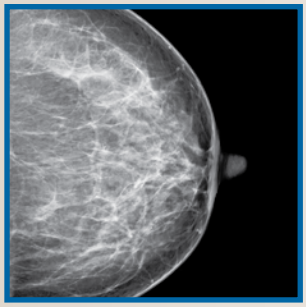
MRA



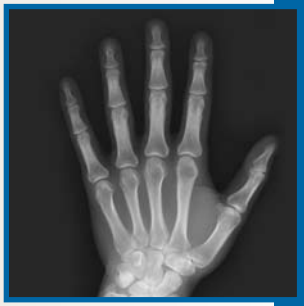
CT • 3D CT

CTA

3D Ultrasound



Digital Mammography



Digital X-Ray & DEXA

- Electronic Medical Record (EMR) Interfaces now available
- Preauthorization Department for obtaining all insurance preauthorizations
- Fellowship, sub-specialty trained radiologists
- Friendly, efficient staff and convenient, beautiful office settings
- Transportation Service for patients

*Higher Field OPEN MRI*

**WARWICK**
250 Toll Gate Rd.
TEL 401.921.2900

**CRANSTON**
1301 Reservoir Ave.
TEL 401.490.0040

**CRANSTON**
1500 Pontiac Ave.
TEL 401.228.7901

**N. PROVIDENCE**
1500 Mineral Spring
TEL 401.533.9300

**E. PROVIDENCE**
450 Vets. Mem. Pkwy. #8
TEL 401.431.0080

***A Clearer Vision of Health™***

***theimaginginstitute.com***

Although similar to IH, CH has unique imaging characteristics, and ultrasound and MRI are the most useful modalities. In contrast to IH, calcifications and large tubular structures representing outflow veins in the context of significant intratumoral arteriovenous shunting are more frequently observed in CH with grayscale US.[1] With MRI, large flow voids are present and in post-contrast images there is inhomogeneous enhancement, unlike the typical uniform enhancement seen in IH. Surrounding edema on T2 images, characteristic of IH, is absent in CH. Arteriography may be indicated in cases where fibrosarcoma or arteriovenous malformation are suspected.

In larger tumors, which frequently ulcerate and hemorrhage as involution proceeds, interventional radiological embolization may be required to staunch severe bleeding and help to resolve high output heart failure. The risks of this procedure in a newborn, including a high probability of ispilateral or contralateral limb loss, obviate its use in all but the most extreme situations. This was the case with our patient. He was brought to interventional radiology at Rhode Island Hospital where an angiogram demonstrated large disorganized feeding arteries and massively dilated outflow veins. (Figure 2) The central portion of the lesion was embolized with **n-butyl cyanoacrylate (NBCA)**, a glue embolic agent. (Figure 3) There were no complications, the bleeding abated, and the patient's heart failure improved.

## References


1. Gorincour G, Kotka V, Rypens F, et al. Imaging characteristics of two subtypes of congenital hemangiomas: rapidly involuting congenital hemangiomas and non-involuting congenital hemangiomas. *Pediar Radiol*. 2005;35:1178–85.
2. Konez O, Burrows P, Mulliken JB, et al. Angiographic features of rapidly involuting congenital hemangioma. *Pediar Radiol*. 2003;33:15–9.
3. Krol A, MacArthur CJ. Congenital Hemangiomas. *Arch Facial Plast Surg*. 2005;7:307–11.
4. Mulliken JB, Enjolras O. Congenital hemangiomas and infantile hemangioma: Missing links. *J Am Dermatol*. 2004;50:875–82.



*Lawrence J. Keating, M.D. is currently a fellow in Vascular and Interventional Radiology at Rhode Island Hospital.*

*Gregory M. Soares, M.D. is Assistant Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University and Director of Vascular and Interventional Radiology at Rhode Island Hospital.*

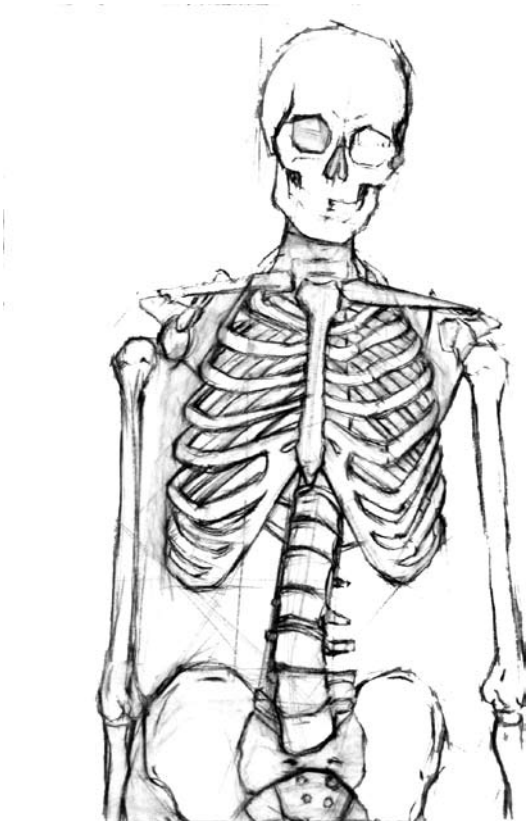
*Christopher S. Muratore, M.D. is Assistant Professor of Surgery and Pediatrics at Warren Alpert Medical School of Brown University and a pediatric surgeon at Hasbro Children's Hospital.*


### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


Gregory M Soares, MD, FSIR
e-mail: gsoares@lifespan.org

# Femoroacetabular Impingement

*Peter T. Evangelista, MD, and Holly C. Gil, MD*

**A 33 year-old avid male runner** presented to an orthopedic surgeon with complaints of worsening left hip pain interfering with activities of daily living and restricted motion. His examination was remarkable for a positive anterior impingement sign, reproducible pain with forced internal rotation and adduction in 90° of flexion.

Magnetic resonance arthrogram (Figure 1) of the left hip demonstrated a focal tear/detachment at the acetabulolabral junction in the anterosuperior quadrant with a focal osseous "bump" along the anterosuperior femoral neck. There was adjacent marrow edema and synovial herniation pits. No chondral injury was noted. Corresponding **computed tomography (CT)** of the left hip was performed with 3D volume rendering (Figure 2) which more clearly depicts the focal area of osseous ridging along the anterosuperior femoral neck. The patient subsequently underwent hip arthroscopy with debridement of the labral tear and femoral osteoplasty of the osseous "bump."

## Diagnosis: Femoroacetabular impingement (FAI) with labral tear.

FAI and hip dysplasia comprise two opposing ends of the spectrum of hip morphologies associated with early hip osteoarthritis in young active patients. The farther one is from center (normal) the more likely one is to have symptoms. Morphology alone, however, does not directly equate to symptomatology as the prevalence of cam morphology was found to be 14% in asymptomatic volunteers.[1] The level and type of activity are equally, if not more, important to the clinical expression of disease with increasing levels of activity and those activities requiring more extremes in range of motion having higher likelihood of symptoms.

FAI is characterized by pathologic contact during hip joint motion between skeletal prominences of the acetabulum and the femur that limits the physiologic hip range of motion, typically flexion and internal rotation.[2] FAI is further divided into pincer type (acetabular causes), cam type (femoral causes), and mixed type (both acetabular and femoral causes).

The typical clinical presentation in FAI is groin pain with hip rotation, in the sitting position, or both during and after sports activities in young active patients. On clinical exam, patients demonstrate restricted range of motion, particularly with flexion and internal rotation. A positive impingement sign should be present. Patients may also have a "Drehmann's" sign if there is unavoidable passive external rotation while performing hip flexion.[3]

Imaging of FAI starts with radiographs that can evaluate for focal or general acetabular overcoverage as well as for osseous "bumps" or loss of concavity at the femoral head/neck junction. They can also exclude alternate pathology. Ultimately, advanced imaging such MR arthrography is typically performed to evaluate for labral tears and chondral lesions (typically anterosuperior) associated with the disorder. Although MR imaging may demonstrate the osseous bumps on the femoral neck, CT, with 3D reconstructions, is often helpful in preoperatively defining the bony anatomy.

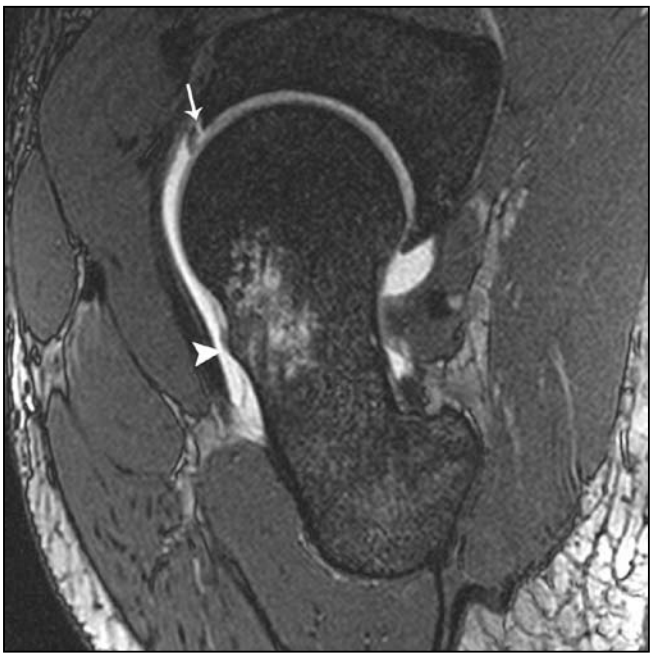


Figure 1: Axial oblique 3D water excitation true FISP MR image of left hip with intra-articular gadolinium demonstrates focal detachment of the anterosuperior labrum (arrow) and osseous bump (arrowhead) on the anterosuperior femoral neck with associated marrow edema.

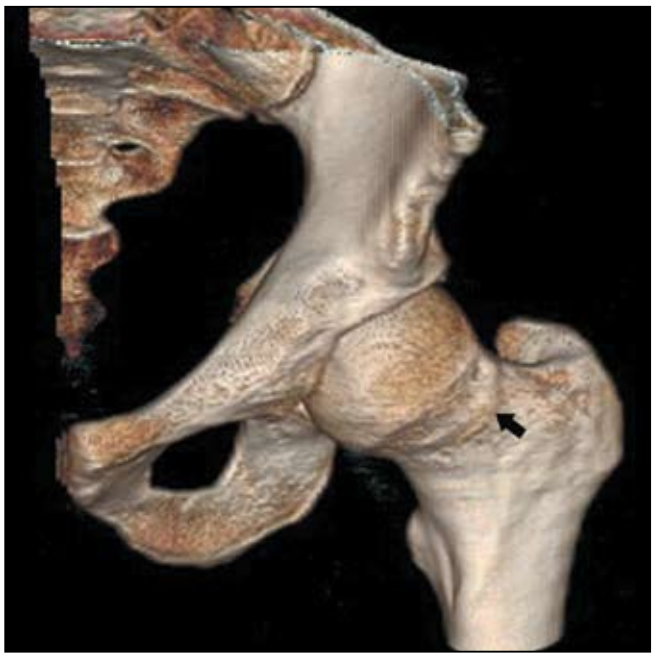


Figure 2: 3D volume rendered image from a CT of left hip demonstrates focal osseous ridge (arrow) along the anterosuperior femoral neck.

Surgical treatment of FAI focuses on alleviating femoral abutment by either femoral osteoplasty or trimming the acetabular rim. Labroplasty, chondroplasty, and focal synovectomy also play a role in specific cases. Rarely, more extensive surgery including periacetabular osteotomy may be indicated.

In conclusion, FAI is a clinical entity with an associated pattern of imaging findings which physicians should be aware of in active young patients presenting with hip pain. One must be cautious in making the diagnosis based on imaging findings alone as asymptomatic patients may have hip morphologies that fall within the FAI end of the spectrum.[4] Long-term data is needed to determine whether early surgical intervention will ultimately delay or prevent future osteoarthritis.

## References


1. Hack K, DiPrimio G, Rakhra K, Beaule PE. Prevalence of cam-type femoroacetabular impingment morphology in asymptomatic volunteers. *J Bone Joint Surg Am*. 2010;92:2436–44.
2. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: Radiographic diagnosis-what the radiologist should know. *AJR*. 2007;188:1540–2.
3. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M. Comparison of six radiographic projections to assess femoral head/neck asphericity. *Clin Orthop Relat Res*. 2006;445:181–5.
4. Anderson SE, Siebenrock KA, Tannast M. Femoroacetabular impingment: Evidence of an established hip abnormality. *Radiology*. 2010;257:8–13.



*Peter T. Evangelista, MD, is Director of Musculoskeletal Radiology at Rhode Island Hospital, and Assistant Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown Universty.*

*Holly C. Gil, MD, is an Assistant Professor of Diagnostic Imaging (Clinical) at the Warren Alpert Medical School of Brown Universty.*

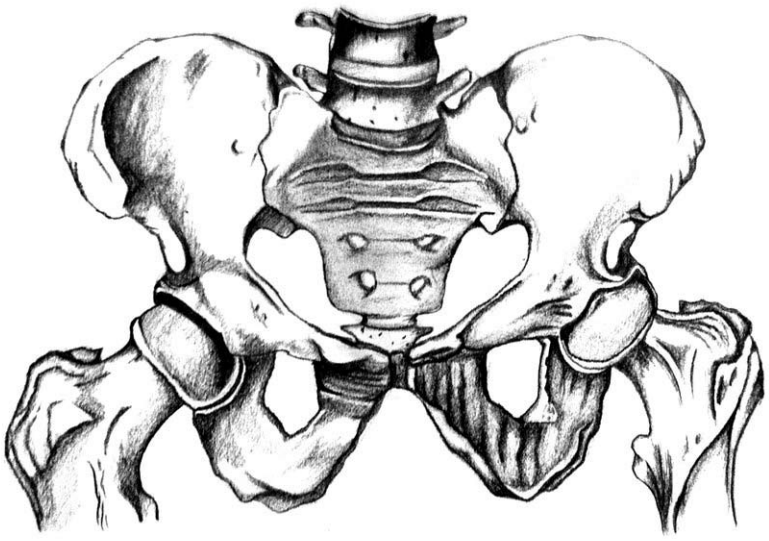

### Disclosure of Financial Interests

Peter T. Evangelista, MD, is a consultant fpr BioMetric Therapeutics, Inc.

Holly C. Gil, MD, and/or their spouse/significant other has no financial interests to disclose.

## Correspondence


Peter T. Evangelista, MD
e-mail: PEvangelista@Lifespan.org

# Imaging Diagnosis of Uterine Anomalies

*Ana P. Lourenco, MD, and David Swenson, MD*

**Clinical Presentation: We illustrate the benefits of** imaging for diagnosis of uterine anomalies with two clinical cases recently evaluated at our hospital.

The first patient (A) is a 21-year-old female, who is being evaluated for possible uterine anomaly. A pelvic **ultrasound (US)** three years prior had suggested a uterine anomaly.

The second patient (B) is a 34-year-old female presenting for evaluation of uterine anomaly following multiple pregnancy losses in the second trimester.

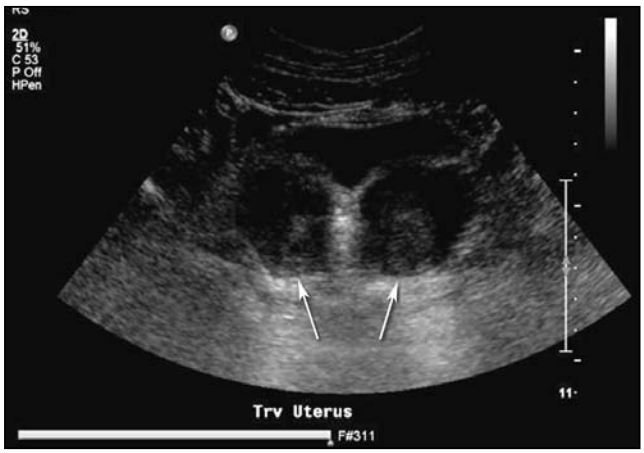



Figure 1.

## Imaging Findings:

Figure 1 shows the 2D US images from Patient A, obtained three years prior for evaluation of pelvic pain. Two endometrial cavities are seen and the diagnosis of uterus didelphys was suggested (white arrows).

Figure 2 shows the MRI images from Patient A, which confirm the diagnosis of uterus didelphys, and show two separate uterine horns, widely separated (white arrows), as well as two cervices (white arrowheads) and two vaginal canals (white arrows).

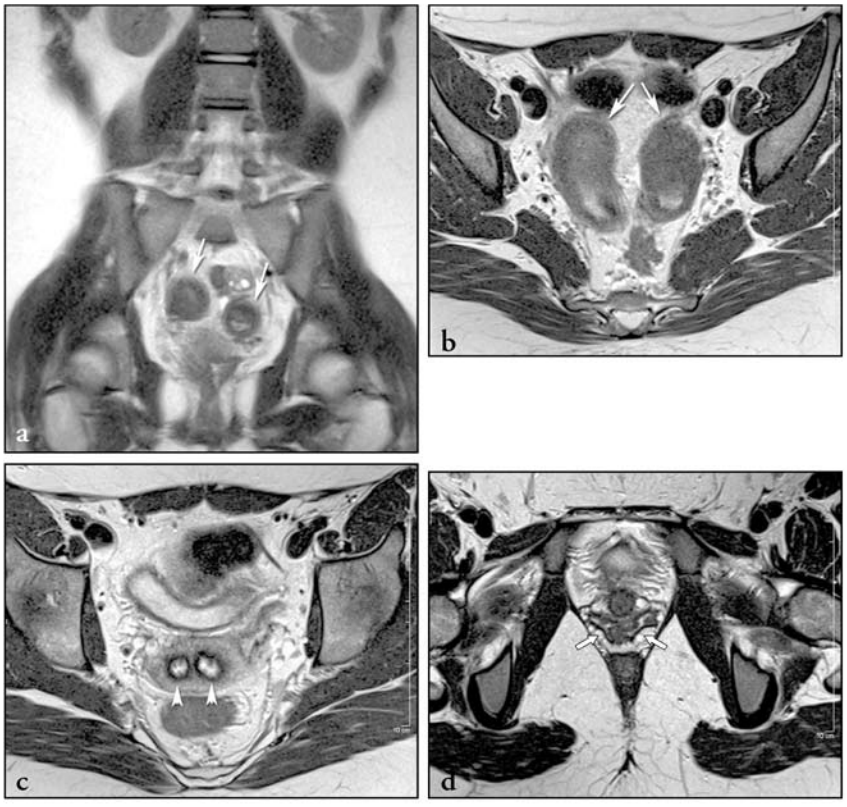
Figure 3 shows the 3D US image from Patient B showing a septate uterus, with two endometrial cavities seen (white arrows) and a flat outer contour of the uterus (white arrowhead).

Figure 4 shows the hysterosalpingogram (HSG) from Patient B showing two endometrial cavities (white arrows). This appearance can be seen with septate uterus or bicornuate uterus. Evaluation of the outer uterine myometrial contour is necessary to make an accurate diagnosis.

Figure 5 shows multiplanar MRI images showing two endometrial cavities (white arrowheads), with a septum extending to the cervix (white arrow). The outer myometrial contour is well seen and is flat. This establishes the diagnosis of septate uterus.

## Discussion:

Congenital uterine anomalies are rare in women with normal reproductive outcomes, but affect up to 25% of women with recurrent pregnancy losses.[1] The diagnosis can be difficult to establish. However, precise diagnosis of the type of anomaly is critical, as treatment and reproductive outcomes vary significantly depending on the type of anomaly. The American Fertility Society classification[2] is the most commonly used in describing congenital uterine anomalies, which include agenesis/hypoplasia, unicornuate, didelphys, bicornuate, septate, arcuate, and **diethylstilbestrol (DES)** drug related.




Figures 2a-d.

Uterus didelphys results from failure of fusion of the two mullerian ducts during embyrogenesis. The duplication often involves the uterus and cervix, but can include duplication of the vaginal canal as well, as seen in this case.

Septate uterus is the most common congenital uterine anomaly, and has the worst reproductive outcomes.[3] It results from failure of resorption of the midline septum between the two mullerian ducts during embryogenesis. The septum can be partial, or can extend to the cervix, and possibly into the vagina as well. Septa can be muscular or fibrous, and are often surgically resected in an attempt to improve pregnancy outcome.

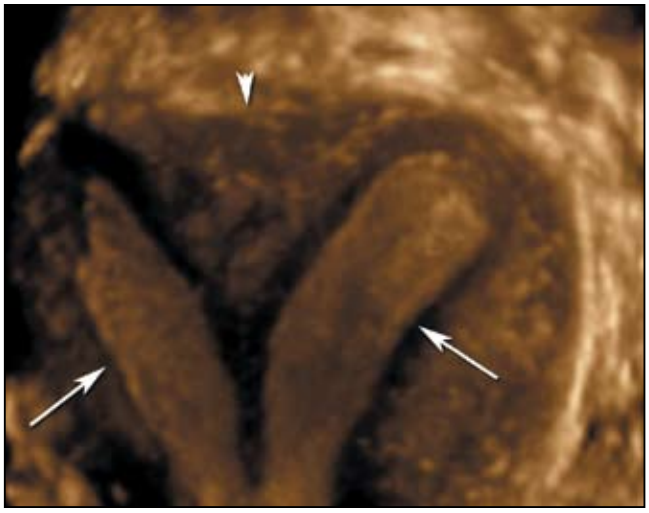


Figure 3.

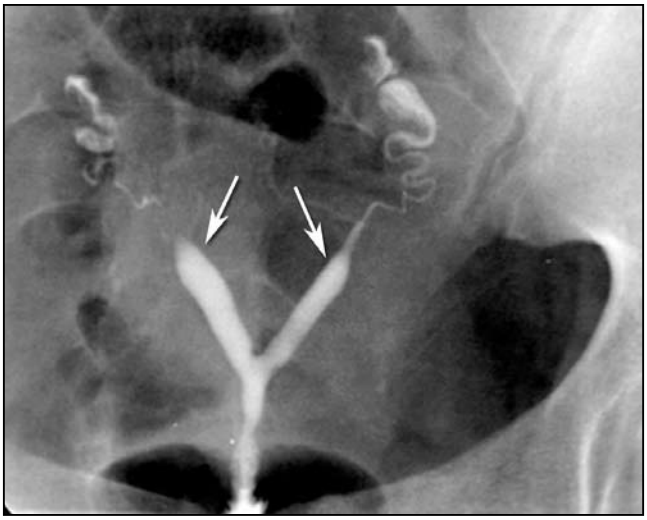


Figure 4.

MRI is the most sensitive, specific, and least invasive imaging test to definitively characterize uterine congenital anomalies.[4] Additional imaging studies are available, and include 3D US, HSG, and sonohysterography. Renal anomalies can be associated with uterine anomalies, and evaluation of the kidneys should be standard in the imaging work-up of such anomalies.

## References:


1. Acien P. Incidence of Mullerian defects in fertile and infertile women. *Hum Reprod*. 1997; 12:2277.
2. The American Fertility Society classifications of adnexal adhesions, distal tubal occlusion, tubal occlusion secondary to tubal ligation, tubal pregnancies, mullerian anomalies and intrauterine adhesions. *Fertil Steril*. 1988;49:944.
3. Homer HA, Li TC, Cooke ID. The septate uterus: a review of management and reproductive outcome. *Fertil Steril.* 2000;73:1–14.
4. Troiano RN, McCarthy SM. Mullerian duct anomalies: imaging and clinical issues. *Radiol.* 2004;233:19–34.



*Ana P Lourenco MD is a Staff Radiologist with Rhode Island Medical Imaging, and an Assistant Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University.*

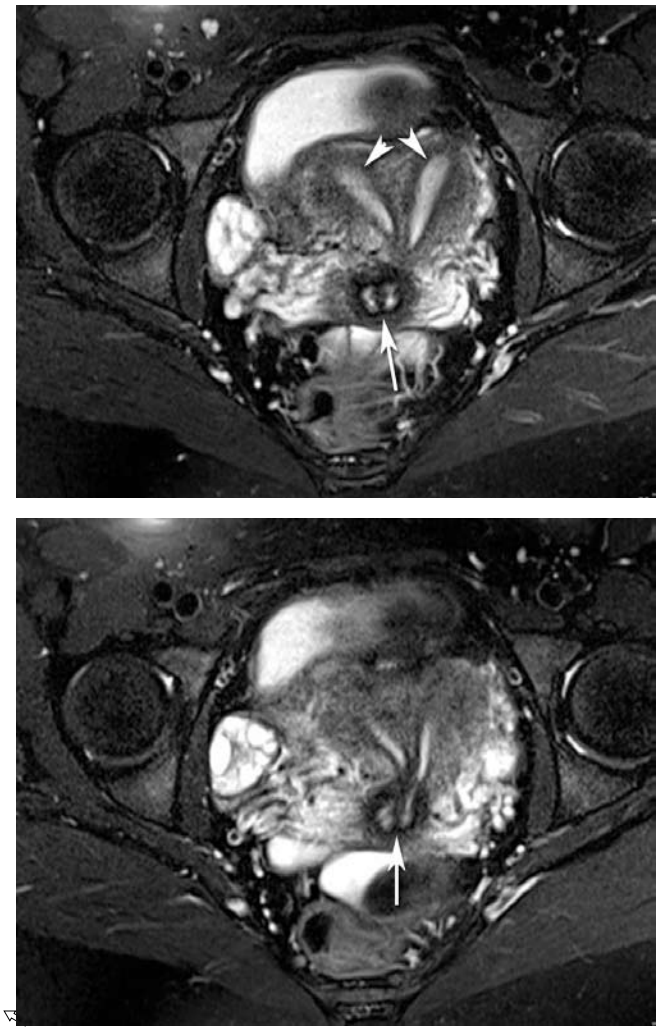
*David W. Swenson, MD is a Diagnostic Radiology resident at the Warren Alpert Medical School of Brown University.*


### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


Ana P Lourenco, MD
RIH Dept Diagnostic Imaging
593 Eddy Street
Providence, RI 02903
phone: (401) 444-5184
fax: (401) 444-5017
e-mail: alourenco@lifespan.org

Figures 5a and b.

# 4D CT – A Diagnostic Tool to Localize an Occult Parathyroid Adenoma In a Patient With Primary Hyperparathyroidism

*Michael D. Beland, MD, and Jack M. Monchik, MD*

**A 64 YEAR-OLD FEMALE PRESENTED WITH** persistent primary hyperparathyroidism after undergoing two previous neck explorations for a suspected parathyroid adenoma. Twelve days after her most recent neck exploration, her parathyroid hormone level remained elevated and a **four-dimensional computed tomography** (**4D CT**) of the neck and upper chest was performed. The CT examination demonstrated early dense arterial phase enhancement and contrast washout within an 8 x 8 x 10 mm nodule immediately posterolateral to the left aspect of the esophagus at the level of the T3 vertebral body highly suspicious for an ectopic parathyroid adenoma. (Figures 1 and 2) Following recovery from the most recent surgery and confirmation of a persistently elevated parathyroid hormone level, the patient was taken back to the operating room for targeted exploration of the area seen on 4D CT.

An extensive dissection in the left paratracheal tissues behind the common carotid artery revealed a soft tissue mass that was removed. Frozen section confirmed parathyroid adenoma. The estimated weight of the adenoma was 389 mg. Parathyroid hormone levels were monitored during the case and demonstrated an appropriate response after resection. One third of the adenoma was used for autotransplantation in the forearm. The autologous autotransplanted parathyroid tissue was then removed approximately six weeks later after her calcium and parathyroid hormone levels remained normal.

## Discussion

Improvements in imaging localization of parathyroid adenomas in patients with **primary hyperparathyroidism** (**PHPT**) prior to surgical excision have allowed the transition from a traditional bilateral neck exploration to selective excision of an adenoma localized pre-operatively. The newest techniques of minimally invasive parathyroidectomy require precise pre-operative localization to be successful.[1] The current modalities used for routine pre-operative imaging localization of a parathyroid adenoma primarily consist of **nuclear medicine** (NM) sestamibi scans with or without cervical **ultrasound** (US). The two modalities are complimentary as the nuclear medicine scan offers functional information while US demonstrates more detailed anatomic information.

Despite improvements in US and NM technology, neither modality will detect all parathyroid adenomas. A recent meta-analysis demonstrated Tc-99m sestamibi scanning outperformed ultrasound with respective sensitivities of 88% versus 78% for single adenomas. Sensitivity for detection of multiple adenomas was sub-

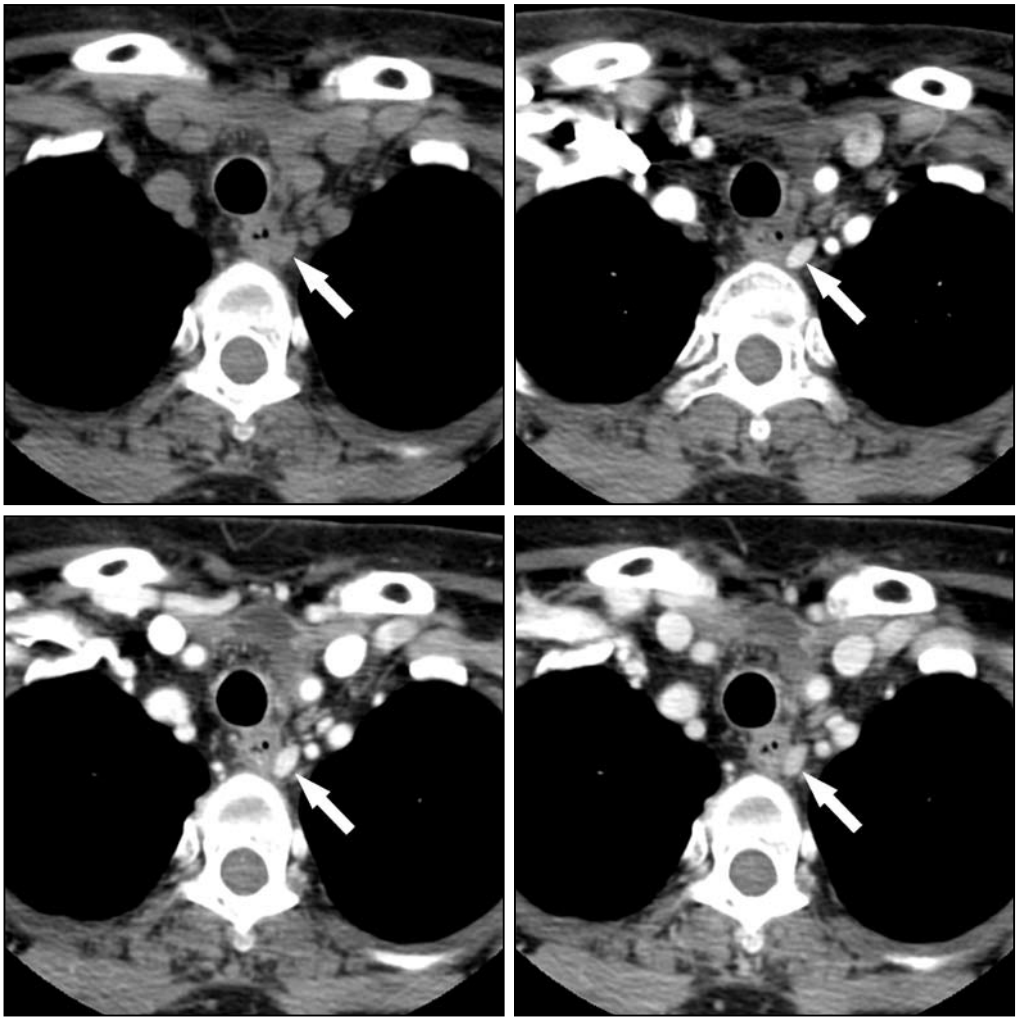


Figure 1. Axial CT examination (a. upper left) before, (b. upper right) 30 seconds, (c. lower left) 60 seconds and (d. lower right) 90 seconds after intravenous contrast administration demonstrate early arterial phase enhancement and washout of the ectopic parathyroid adenoma (arrows).

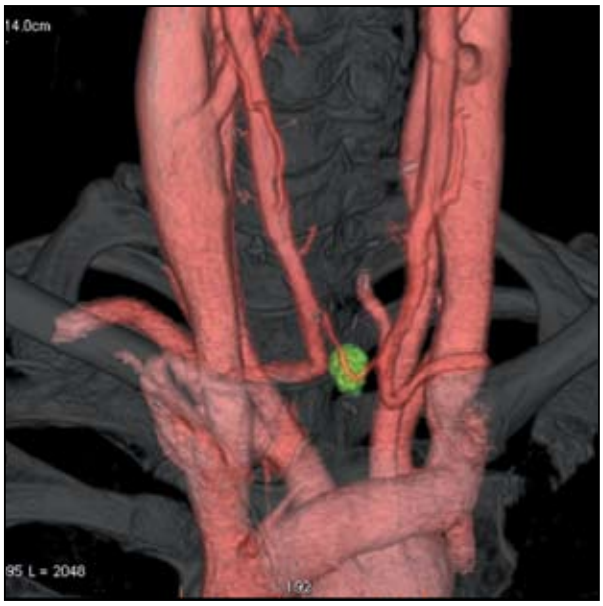



**Figure 2.** Volume rendered post-contrast CT image demonstrating the parathyroid adenoma (green) relative to the adjacent cervical and upper thoracic vascular and skeletal anatomy.

stantially less being 30% for sestamibi and 16% for US.[2] Some institutions are now routinely using SPECT/CT for localization as a single test that can offer both functional and anatomic information although the literature has shown conflicting evidence of additional clinical value.[3, 4]

Localization is especially important in the setting of persistent or recurrent PHPT after surgical exploration. Before proceeding with reoperation, the current consensus is that two concordant preoperative imaging studies should localize hyperfunctioning parathyroid tissue to the same anatomic region of the neck.[5] The goal of accurate localization is to minimize morbidity and maximize the likelihood of success.[6] There are recent reports in the surgical literature describing the use of single phase contrast-enhanced CT to localize parathyroid adenomas, particularly in patients who have already undergone a surgical neck exploration.[7–9] 4D CT, a technique which employs precontrast and multiphase post contrast images through the neck typically at 30, 60 and 90 seconds, has been described as a potentially more robust method of localization exploiting the differential early enhancement of parathyroid adenomas.[1, 6, 10]

Our group at Rhode Island Hospital has reported one of the largest series in the literature using 4D CT, demonstrating a mean sensitivity and specificity of 82% and 92% respectively for precise localization of occult parathyroid adenomas when prior surgery or localization with US and sestamibi NM scans have been unsuccessful.[10] We showed fair to excellent interobserver reliability across all pairs with an overall excellent reliability. In addition, we demonstrated that adenomas demonstrate a characteristic contrast enhancement pattern of early enhancement. This enhancement was significantly different at the 30 and 60 second phases compared with the progressive enhancement pattern demonstrated in normal lymph nodes.

Given that the majority of parathyroid adenomas will be found using a combined US and NM imaging algorithm, 4D CT should not be used in the standard evaluation. However, 4D CT will localize a majority of adenomas successfully in patients where conventional imaging is unsuccessful. While 4D CT may offer improved localization in the routine work-up of primary hyperparathyroidism,[1] the increased radiation dose should limit 4D CT to problem-solving cases.

In summary, parathyroid adenomas in patients with unsuccessful localization on conventional imaging can be accurately demonstrated pre-operatively on 4D CT. Parathyroid adenomas demonstrate characteristic rapid enhancement which can help differentiate these lesions from lymph nodes. Precise localization is critical to minimizing surgical morbidity, particularly in patients who have undergone prior surgery.

## References

1. Rodgers SE, Hunter GJ, Hamberg LM et al. Improved preoperative planning for directed parathyroidectomy with 4-dimensional computed tomography. *Surgery.* 2006;140:932–40.
2. Ruda JM, Hollenbeak C, Stack BC Jr. A systematic review of the diagnosis and treatment of primary hyperparathyroidism from 1995 to 2003. *Otolaryngol Head Neck Surg.* 2005;132:359–72.
3. Lavely WC, Goetze S, Friedman KP et al.. Comparison of SPECT/CT, SPECT, and planar imaging with single- and dual-phase (99m)Tc-sestamibi parathyroid scintigraphy. *J Nucl Med.* 2007;48:1084–9.
4. Gayed IW, Kim EE, Broussard WF et al. The value of 99mTc-sestamibi SPECT/CT over conventional SPECT in the evaluation of parathyroid adenomas or hyperplasia. *J Nucl Med.* 2005;46:248–52.
5. Caron NR, Sturgeon C, Clark OH. Persistent and recurrent hyperparathyroidism. *Curr Treat Options Oncol.* 2004;5:335–45.
6. Mortenson MM, Evans DB, Lee JE et al. Parathyroid exploration in the reoperative neck: improved pre-operative localization with 4D- computed tomography. *J Am Coll Surg.* 2008;206:888–95.
7. Harari A, Zarnegar R, Lee J, Kazam E, Inabnet WB 3rd, Fahey TJ 3rd. Computed tomography can guide focused exploration in select patients with primary hyperparathyroidism and negative sestamibi scanning. *Surgery.* 2008;144:97–6.
8. Zald PB, Hamilton BE, Larsen ML, Cohen JI. The role of computed tomography for localization of parathyroid adenomas. *Laryngoscope.* 2008;118:1405–10.
9. Randall GJ, Zald PB, Cohen JI, Hamilton BE. Contrast-enhanced MDCT characteristics of parathyroid adenomas. *AJR.* 2009;193:W139–W143.
10. Beland MD, Grand DJ, Mayo-Smith WW, Machan JT, Monchik JM. 4D CT for localization of difficult to find parathyroid adenomas in patients with hyperparathyroidism. *AJR.* 2011;196:61–6.

*Michael D. Beland, MD, is Director of Ultrasound at Rhode Island Hospital and Assistant Professor of Diagnostic Imaging, Warren Alpert Medical School of Brown University.*

*Jack M. Monchik, MD, is Chief of Endocrine Surgery at Rhode Island Hospital and Clinical Professor of Surgery, Warren Alpert Medical School of Brown University.*

### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence

Michael D. Beland, MD
Department of Diagnostic Imaging
Rhode Island Hospital
593 Eddy Street
Providence, RI 02903
phone: (401) 444-5184
fax: (401) 444-5017

# Radiation Necrosis of a High-grade Glioma

*Deepak Raghavan, MD, Jerrold Boxerman, MD, PhD, Suriya Jeyapalan, MD, MPH, and Jeffrey Rogg, MD*

**A 64 YEAR OLD MALE WAS REFERRED TO THE NEUROSURGICAL SERVICE** at Rhode Island Hospital for evaluation of a left frontal mass. Given proximity of the lesion to eloquent cortex, resection was not performed and a histopathologic diagnosis of grade III anaplastic oligoastrocytoma was made through biopsy. The patient was enrolled in an experimental treatment protocol that included Paclitaxel Poliglumex (PPX; a microtubule stabilizer and mitotic inhibitor with a radiosensitization index of 4-8), temozolomide, and radiation. The patient also underwent two cycles of maintenance temozolomide therapy and a third cycle of dose-intensive temozolomide therapy. Serial MR imaging with perfusion-weighted imaging was performed at multiple time points throughout treatment.

## Imaging Findings

Initial pre-biopsy brain MRI (Figure 1a) demonstrated a 2.7cm heterogeneously enhancing mass in the left posterior frontal operculum (white arrow), and a second smaller enhancing nodular lesion (Figure 1b) in the left frontal lobe (white arrowhead). Both of these enhancing lesions demonstrated significantly elevated **relative cerebral blood volume** (**rCBV**) on perfusion-weighted imaging (Figures 1c and 1d). Follow-up MRI at three months following completion of PPX therapy demonstrated substantial response to treatment at the site of the left frontal opercular mass (not shown), with minimal residual enhancement and reduced mass effect. The left frontal nodular lesion was shown to have slightly increased enhancement with persistent but stable, elevated rCBV. (Figures 2a and 2c)

Follow-up imaging at 8 months following completion of PPX therapy demonstrated significantly increased enhancement associated with the left frontal satellite lesion. (Figure 2b, white arrowhead) However, the corresponding rCBV was substantially smaller, approximating that of normal appearing contralateral white matter (Figure 2d), consistent with radiation necrosis and not tumor progression. The patient underwent a brain biopsy nine days after the MRI, and histopathology confirmed the imaging findings, demonstrating

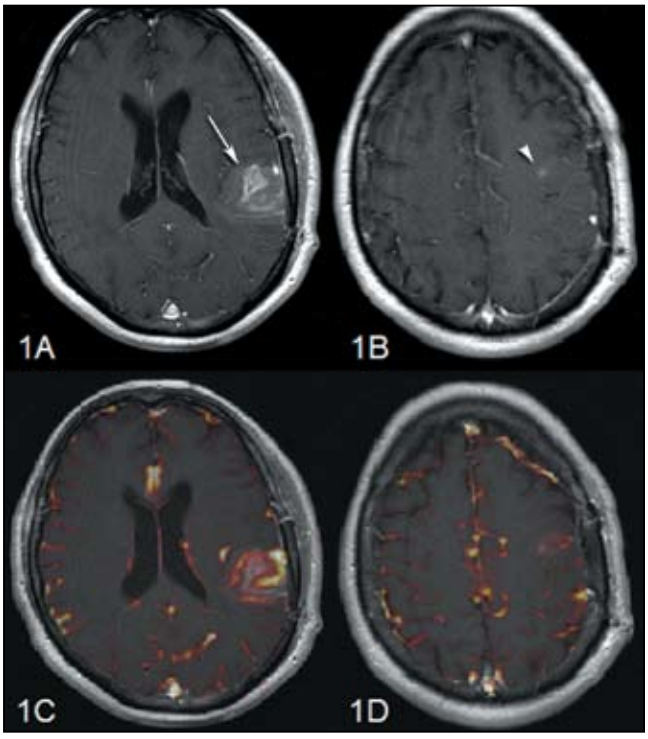



**Figure 1**: Preoperative contrast-enhanced T1-weighted MRI imaging was obtained with additional corresponding MR perfusion-weighted images. A heterogeneously enhancing left frontal opercular lesion is demonstrated **(A, white arrow)** with an additional enhancing satellite lesion in the left frontal lobe **(B, white arrowhead)**. Both of these lesions demonstrate increased rCBV on the corresponding perfusion-weighted images **(C and D)**.

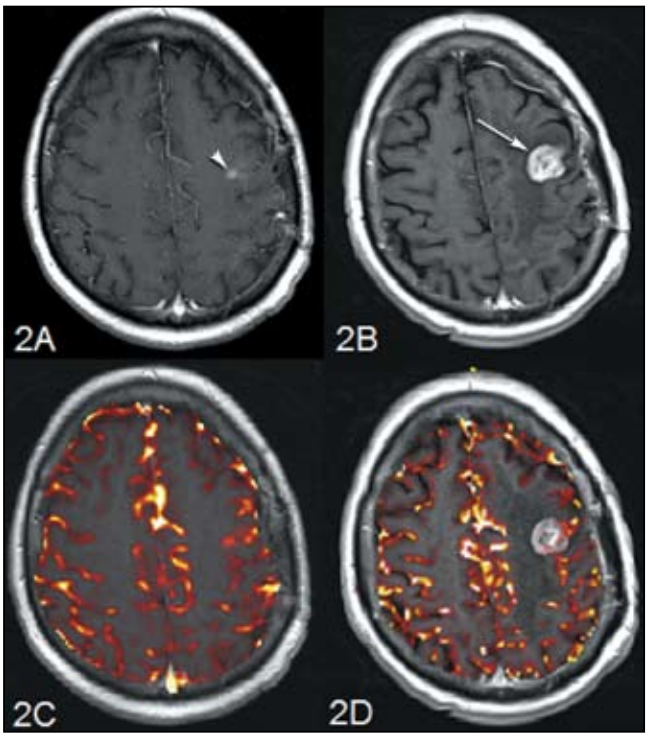



**Figure 2**: Serial follow-up contrast-enhanced T1-weighted MRI imaging as well as corresponding perfusion-weighted imaging was obtained at 3 months as well as 8 months after resection and initiation of chemotherapy and radiation. 3 month follow-up imaging demonstrates slightly increased enhancement of the left frontal opercular lesion **(A, white arrowhead)** with stable rCBV **(C, white arrow)**. Follow-up imaging at 8 months demonstrates significantly increased enhancement of the left frontal satellite lesion **(B)**, however the associated rCBV had significantly decreased, approximating that of normal contralateral white matter **(D)**. These findings were most suggestive of radiation necrosis, which was confirmed by histology.

"quiescent" tumor with foci of necrosis and positive Ki-67 (a marker of cellular proliferation) in only approximately one percent of tumor cells.

## Discussion

Radiation therapy and chemotherapeutic agents that act as radiation sensitizers are often used in the treatment of patients with high-grade gliomas. Following treatment, patients may deteriorate clinically with progression of enhancement on follow-up brain MRIs that looks similar for both radiation necrosis and true progression of tumor.[5] Recurrent tumor demonstrates angiogenesis and microvascular proliferation, and indicates treatment failure. Radiation necrosis is a sign of treatment response manifested as endothelial cell damage and decreased capillary perfusion and may actually be associated with longer survival than in cases without radiation necrosis.[6] Therefore, distinguishing between these two entities carries significant clinical implications. Traditionally, serial MRI and stereotactic biopsy have been the primary methods of diagnosing radiation necrosis. MR **perfusion-weighted imaging** (**PWI**) may help differentiate radiation necrosis and recurrent or residual neoplasm, and this remains an active area of investigation.[1-3]

PWI measures cerebral perfusion by utilizing the transient microscopic magnetic field disturbances that are induced by a bolus injection of exogenous paramagnetic contrast material.[4] Rapid MRI acquisition permits estimation of contrast concentration-time curves utilizing modified tracer kinetic principles.[4] A variety of hemodynamic parameters can subsequently be computed. Of these parameters, rCBV has been the most widely used and has been demonstrated to correlate with tumor grade and tumor microvascular density.[3] Preliminary investigations have demonstrated that decreasing rCBV values over time is indicative of response to treatment, whereas increasing rCBV values suggests tumor progression and treatment failure. In the case above, the enlarging enhancing lesion had decreasing rCBV, consistent with treatment effect or "pseudoprogression," and this was confirmed on biopsy.

## References


1. Barajas RF, Chang JS, Sneed PK, Segal MR, et al. Distinguishing recurrent intra-axial metastatic tumor from radiation necrosis following gamma knife radiosurgery using dynamic susceptibility-weighted contrast-enhanced perfusion MR imaging. *Am J Neurorad*. Feb2009;30:367–72.
2. Boxerman JL, Schmainda KM, Weisskoff RM. Relative cerebral blood volume maps corrected for contrast agent extravasation significantly correlate with glioma tumor grade, whereas uncorrected maps do not. *Am J Neurorad*. Apr2006;27:859–67.
3. Hu LS, Baxter LC, Smith KA, Feuerstein BG, et al. Relative cerebral blood volume values to differentiate high-grade glioma recurrence from posttreatment radiation effect: direct correlation between imaging-guided tissue histopathology and localized dynamic susceptibility-weighted contrast-enhanced perfusion MR imaging measurements. *Am J Neurorad*. Mar2009;30:552–8.
4. Zaharchuk G. Theoretical basis of hemodynamic MR imaging techniques to measure cerebral blood volume, cerebral blood flow, and permeability. *Am J Neurorad*. Nov/Dec2007;28:1850–8.
5. Kumar AJ, Leeds NE, Fuller GN, Van Tassel P, et al. Malignant gliomas: MR imaging spectrum of radiation therapy – and chemotherapy – induced necrosis of the brain after treatment. *Radiology*. Nov2000;217:377–84.
6. Remler MP, Marcussen WH, Tiller-Borsich J. The late effects of radiation on the blood brain barrier. *Int J Radiation Oncology, Biology, Physics.* Nov1986;12:1965–9.



*Deepak Raghavan, MD is a Diagnostic Radiology resident, Warren Alpert Medical School, Brown University.*

*Jerrold Boxerman, MD, PhD is Assistant Professor of Diagnostic Imaging, Warren Alpert Medical School of Brown University.*

*Suriya Jeyapalan, MD, MPH is Assistant Professor of Neurology, Warren Alpert Medical School of Brown University.*

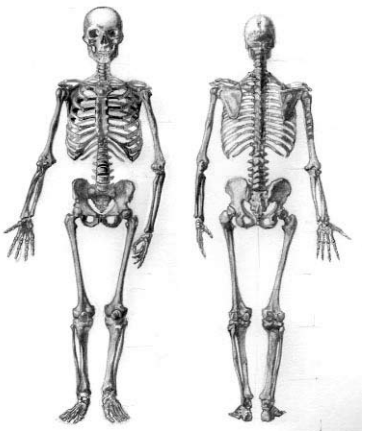
*Jeffrey Rogg, MD is Section Chief of MRI and Associate Professor of Diagnostic Imaging, Warren Alpert Medical School of Brown University.*


### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


Deepak Raghavan, MD
Dept. of Diagnostic Imaging
Rhode Island Hospital
593 Eddy St.
Providence, RI 02903
phone: (401) 444-5184
e-mail: draghavan@lifespan.org

# 14 Year-Old Female With Splenic Torsion

*David W. Swenson, MD, and Thaddeus W. Herliczek, MD*

**A 14-YEAR-OLD FEMALE WITH HIV DEVELOPED** intermittent left-sided abdominal pain one day prior to admission. Her pain progressed rapidly over 24 hours, becoming constant and severe, inducing nausea and vomiting. Her physical exam was notable for left abdominal pain and apparent splenomegaly. Vital signs on admission were as follows: blood pressure 101/62 mmHg, heart rate 112 bpm, respiratory rate of 20, and temperature of 98.7 F°.

Diagnostic imaging was pursued. Ultrasound examination demonstrated a prominent, ectopic spleen positioned anterior to the left kidney and pancreas. Doppler assessment of the splenic hilum was performed. While arterial flow was demonstrated in the splenic artery, no discernable flow was obtained in the splenic vein. (Figure 1) The sonographic findings were concerning for wandering spleen complicated by splenic torsion.

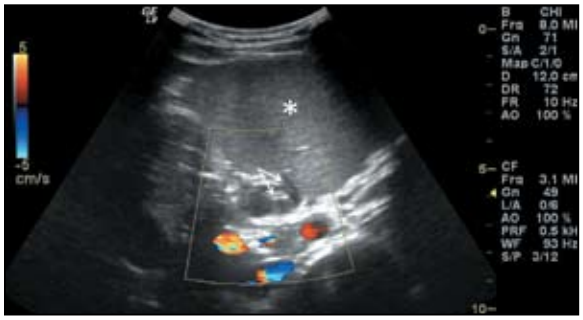



Figure 1. Transverse Doppler US of splenic hilum. Absent flow in splenic vein (arrows).

**Computed tomography (CT)** with intravenous contrast was performed immediately prior to surgical intervention. CT revealed an enlarged, ectopic spleen without parenchymal enhancement. (Figure 2)

Congenital absence of the splenorenal and gastrosplenic ligaments, with 270-360 degrees of splenic torsion was confirmed surgically. Splenectomy was performed given surgical evidence of splenic infarction. An accessory spleen showing preserved perfusion was spared.

## Discussion

Splenic torsion is a rare cause of abdominal pain. In a surgical series of 1,413 splenectomies, pathology revealed splenic torsion as the underlying etiology for the patient's symptoms in only 0.3% of cases.[1] Splenic torsion occurs in the setting of a "wandering spleen," related to congenital absence or laxity of the gastrosplenic and splenorenal ligaments. Absence or laxity of these ligaments results in elongation of the splenic vascular pedicle and splenic "wandering" or hypermobility.[2–4] Several cases have also been reported in association with splenomegaly or during the post partum period. In these cases, ligamentous laxity may be due to the enlarged spleen or hormonal effects.[5]

In patients with wandering spleen, torsion can be intermittent, causing symptoms of mild abdominal pain related to splenic congestion, or it can be acute and severe, leading to rapid splenic necrosis and associated symptoms of acute abdomen.[5] While physical exam often reveals a painful abdominal mass, the diagnosis is generally made by cross-sectional imaging.[3–5]

Ultrasound often demonstrates an enlarged, ectopic spleen. Splenic echotexture varies by the degree of parenchymal congestion and/or infarction.[3,4,6] Similar findings can be seen with contrast en-

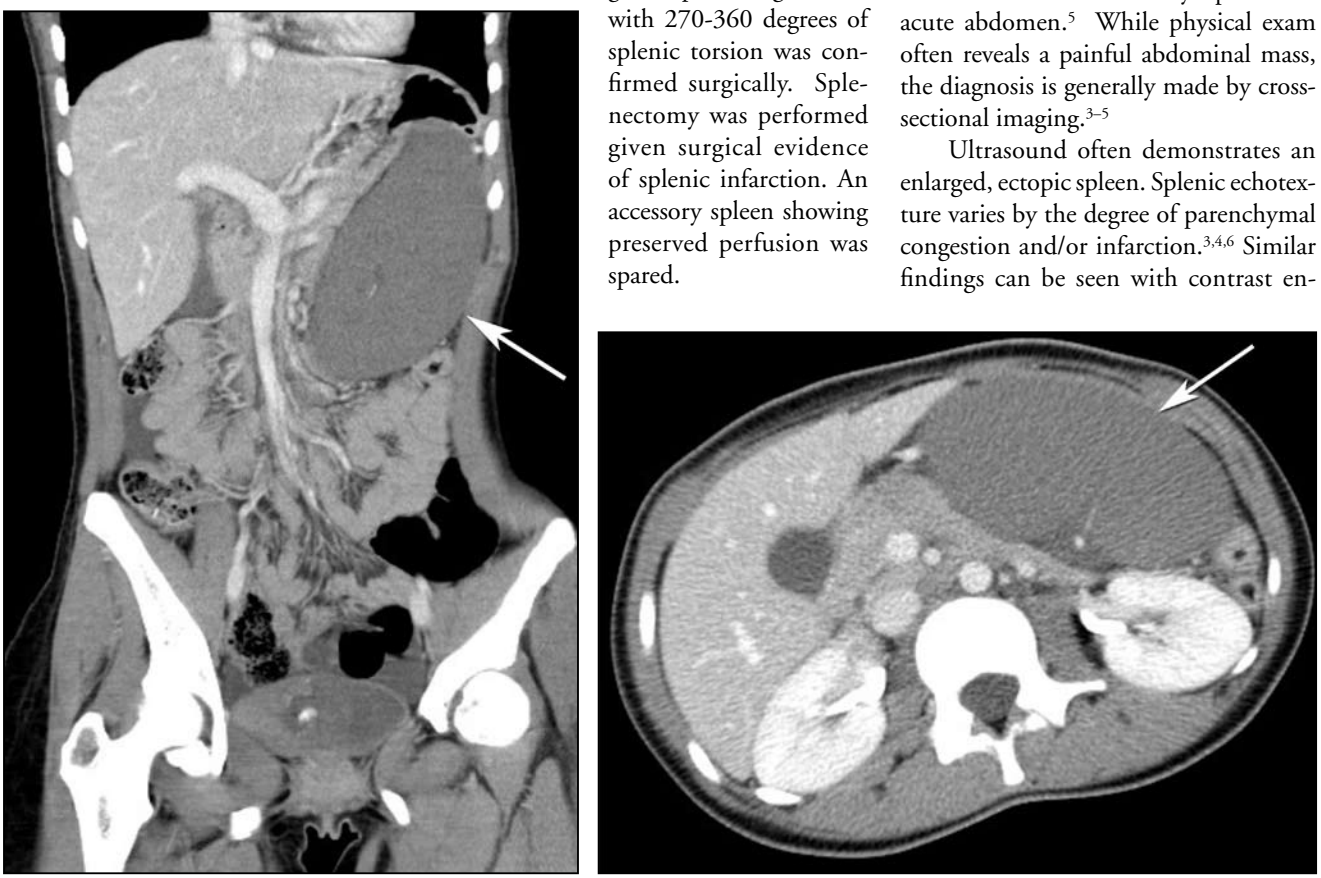


Figure 2. Computed Tomography. Coronal image. (left) Ectopic spleen (arrow) without parenchymal enhancement. Axial image. (right) Ectopic spleen (arrow) anterior to left kidney and pancreas.

hanced CT, including an enlarged, ectopic spleen with relative hypoattenuation, suggesting poor perfusion.[3,4,6] In some cases, twisting of the vascular pedicle may demonstrate a "whorled" appearance.[3]

Splenic torsion is treated surgically, with detorsing of the vascular pedicle and splenopexy if the spleen is found to be viable, or with splenectomy if the spleen is already necrotic. In patients who undergo splenectomy, vaccination for Hemophilus influenzae B, meningococcus, and pneumococcus has become standard prophylaxis.[5,6]

## References


1. Eraklis AJ, Filler RM. Splenectomy in childhood: a review of 1,413 cases. *J Pediatr Surg*. 1972:382–8.
2. Peitgen K, Schweden K. Management of intermittent splenic torsion ("wandering spleen"): a review. *Eur J Surg*. 1995;161:49–52.
3. Swischuk LE, Williams JB, John SD. Torsion of wandering spleen: the whorled appearance of the splenic pedicle on CT. *Pediatr Radiol*. 1993;23:476–7.
4. Brocker FHL, Fellows K, Treves S. Wandering spleen in three children. *Pediatr Radiol*. 1978;6:211–4.
5. Soleimani M, Mehrabi A, Kashifi A, Fonouni H, Buchler M, Krau TW. Surgical treatment of patients with wandering spleen: Report of six cases with a review of the literature. *Surgery Today*. 2007;37:261–9.
6. Chawla SC, Boal DKB, Dillon PW, Grenko RT. Best cases of AFIP: splenic torsion. *Radiographics*. 2003;23:305–8.



*David W. Swenson, MD is a Diagnostic Radiology resident at the Warren Alpert Medical School of Brown University.*

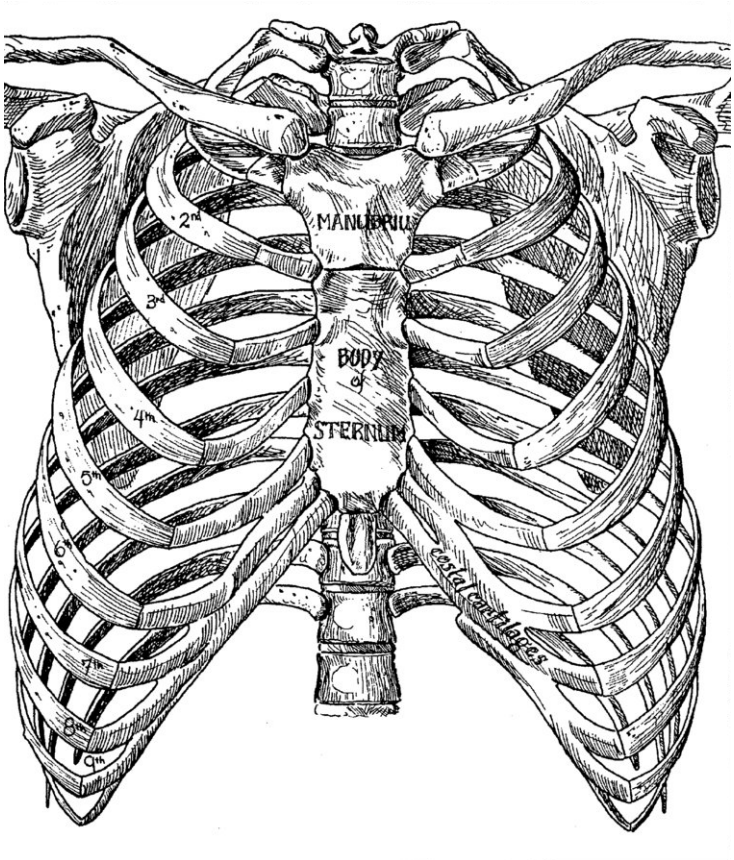
*Thaddeus W. Herliczek, MD, is Director of Pediatric Imaging at Hasbro Children's Hospital, and an Assistant Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University.*


### Financial Disclosures

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


David W. Swenson, MD
Dept. of Diagnostic Imaging
Rhode Island Hospital
593 Eddy St.
Providence, RI 02903
phone: (401) 444-5184
e-mail: dswenson@lifespan.org

Health By Numbers
Rhode Island Department of Health • Michael Fine, MD, Director of Health
Edited by Samara Viner-Brown, MS

# Poisonings and Opportunities for Prevention in Rhode Island

*Robert R. Vanderslice, PhD, and Edward F. Donnelly, RN, MPH*

A competent and efficient medical care system is an essential component of any prosperous society. Rhode Island's medical providers often take advantage of the clinical expertise at our **Regional Center for Poison Control** (**RCPC**) serving Massachusetts and RI, but RCPC's resources for preventing poisonings remain underutilized. Calls to the RCPC are declining at a time when both emergency department visits and fatalities due to poisoning in RI are increasing.

Rhode Island is currently experiencing a prescription drug poisoning epidemic. Over 14,000 hospitalizations resulted from accidental drug overdoses in Rhode Island between 2005-2009.[1] This sobering statistic tells only half the story; drugs are not the only agents to cause poisonings. Both local data from the RCPC [2] and data collected from the nation's network of poison control centers [3] show that exposures to non-drug agents account for more than 40% of poison center calls (23,347 of 52,911 in MA and RI; 1,232,413 of 2,759,287 calls nationally in 2010). This study evaluates the public health significance of poisonings in Rhode Island, both with respect to the impacts on health and the opportunities for prevention.

## Methods

Populations most vulnerable to poisoning in RI, and agents most commonly involved in human exposures were identified using data from death certificates, emergency department discharges and calls to the RCPC.

### Poison Deaths

Poison deaths are those that include poisoning as the underlying cause of death. All causes of death are coded in categories that are part of the **International Classification of Diseases tenth revision** (**ICD-10**) based on information provided on the death certificate. Data from 2000-2009 were selected to show trends over time, with 2009 being the latest year for which data were available.

### Poison Visits to Emergency Departments

Emergency department records are reported to the Rhode Island Department of Health by hospitals licensed in Rhode Island. All diagnoses are coded in accordance with the **ICD ninth revision, clinical modification** (**ICD-9-CM**). Records of persons one to four years of age on the day of the visit with a poison code for any diagnosis were included as cases of poisoning. External causes of injury (Ecodes), also part of ICD-9-CM, are grouped by the first four characters listed and name categories of poisoning for those cases.

### Exposure and information calls to the RCPC

The RCPC toll-free call center responds 24/7/365 with translation services available in over 100 languages. Data from calls made from RI in 2011 were used for this analysis. RCPC collects data on the nature of the call (information, human exposure, animal-related), the agent (AAPCC classification of agents), the person exposed (age, gender, severity of exposure), the caller and location (calls from general public or medical provider, exposures at home, work). Data from accredited centers are combined and published annually by the American Association of Poison Control Centers.

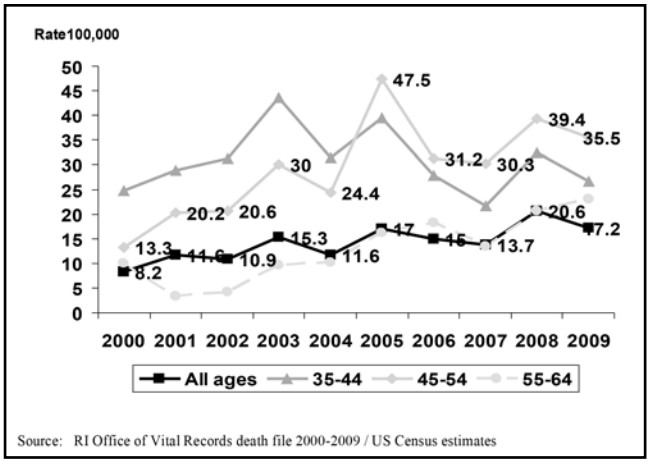



Figure 1. Rate of deaths per 100,000 population due to poisoning by year, Rhode Island, 2000-2009.

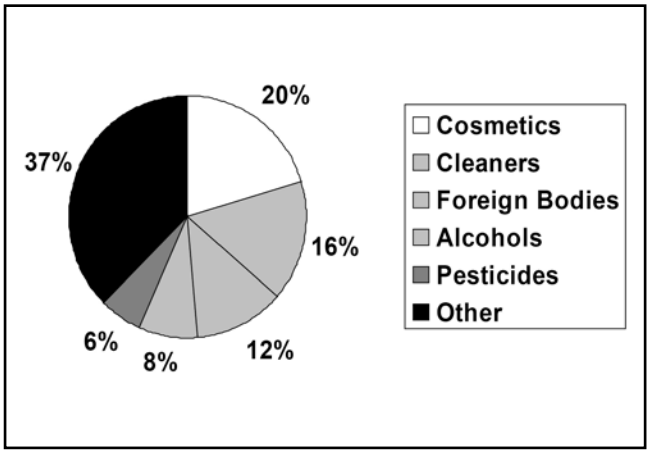



Figure 2. MARI 2011 Poison Calls.

**Table 1. Most Common Categories of poison Ecode (first four digit categories) in Emergency Department persons aged 1 to 4 years, Rhode Island, 2005-2009.**

| Ecode | Freq | % | Description |
|---|---|---|---|
| E858 | 430 | 27.4 | Accidental poisoning by other drugs |
| E850 | 208 | 13.3 | Accidental poisoning by analgesics, antipyretics, and antirheumatics |
| E905 | 113 | 7.2 | Venomous animals and plants as the cause of poisoning and toxic reaction |
| E854 | 82 | 5.2 | Other psychotropic agents |
| E853 | 72 | 4.6 | By tranquilizers |
| E861 | 62 | 4.0 | Cleansing and polishing agents, disinfectants, paints, & varnishes |
| E866 | 62 | 4.0 | Accidental poisoning by other and unspecified solid and liquids |
| E864 | 57 | 3.6 | Corrosives and caustics |
| E869 | 49 | 3.1 | Accidental poisoning by other gases and vapors |
| E865 | 47 | 3.0 | Foodstuffs and poisonous plants |
| E868 | 47 | 3.0 | Other utility gases and carbon monoxide |

## Results

Deaths due to poisoning trended upward in the years 2000-2009. Most of these were drug overdose deaths, and the increase was seen in persons aged 45-54 and to a lesser extent, in persons aged 55-64. Only two poisoning deaths in RI were among those aged less than 15 during this ten year period.

In contrast, pre-school children aged one to four years have the highest injury rates due to non-fatal poisonings and comprised nearly ten percent of the visits to Rhode Island emergency departments during the period 2005-2009. Over 40% of exposure calls to poison centers are for children aged one to four.[2,3] This is the age in which children display hand-to-mouth behaviors that make them vulnerable to poisonings, of which over 85% are associated with ingestion exposures.[3]

Emergency Department discharge data external codes (e-codes) were used to determine the agents responsible for poisonings of children aged one to four years. (Table 1) Drugs, including over-the-counter and prescription medications, accounted for the majority (70%) of poisonings. The classification scheme used by poison centers provides a more detailed description of the agents most commonly associated with poisonings. For non-drug agents, the majority of poisonings fall into a few broad categories that include cosmetics, household cleaners, foreign objects like desiccants and toys like glow sticks, alcohols and pesticides. Figure 2 displays RCPC data for 2011 exposures. Fumes (including carbon monoxide), food products, chemicals, plants, and bites/venoms each accounted for less than five percent of the non-drug calls for 2011 and make up the bulk of the category labeled "other" in Figure 2.

### Origin of calls to the RCPC

Most of the calls to the RCPC (74%) originate with the general public and can be managed on-site without a visit to a medical facility. This capacity to assist people who would otherwise seek treatment for an exposure is an important element of an effective health care system. The cost savings associated with this service more than justify the modest $200,000 contribution from the RI Department of Health.

About 20% of calls to the RCPC originate from health care facilities and medical offices. Despite this awareness of the Regional Center's clinical management expertise, the RCPC is still an under-utilized resource for poisoning prevention.

## Discussion

### Prevention of exposures

The Regional Center has experience responding to concerns from the public that medical providers may find difficult or impossible to address. For example:

- I'm worried about exposure to West Nile Virus but my skin breaks out when I use products containing DEET. Is there another product I can use that is less irritating?
- I am tortured by bed bugs every night, but I am worried about the chemicals my landlord wants to spray in my apartment.
- Rompi una bombilla. Es peligroso?
- As an employer, I know I need to inform my employees of the potential hazardous chemicals in their workplace, and that the information I provide has to conform to international standards for the new Globally Harmonized system (GHS). Where can I get this safety information?

Answers to these and other questions will be provided by highly trained RCPC specialists by calling 1-800-222-1222. For medical providers who are also employers, the RCPC can provide the information needed to meet **Occupational Safety and Health Administration (OSHA)** requirements for informing staff of hazards in the workplace.

### Support for the RCPC

RCPC services are free to the general public. Hospitals can access these services as well. Rhode Island's hospitals place over 1,000 calls to the RCPC each year. The RCPC tries to recoup some of its expenses by sending hospitals invoices based on the level of services the Center has provided that hospital. In 2010, invoices ranged from $2,000 to $38,000, with RI hospitals contributing about 40% of the total invoice amount. This year, contributions from hospitals have fallen to just 13% of the amount invoiced. With cuts to both federal and State funding to the Center, greater support from hospitals is essential to sustain a regional poison center.

## References


1. Green T, Donnelly E. Preventable death: accidental drug overdose in RI. *MHRI*. 2011;94(11):341–3.
2. Regional Center for Poison Control and Prevention Serving Massachusetts and Rhode Island. Annual Report. 2010. Available online at www.maripoisoncenter.org.
3. Bronstein AC, Spyker DA, Cantilena LR Jr, Green JL, Rumack BH, Dart RC. 2010 Annual Report of the American Association of Poison Control Center's National Poison Data System (NPDS): 28th Annual Report. *Clin Toxicol (Phila)*. Dec2011;49(10):910–41.



*Robert R. Vanderslice, PhD, is the Healthy Homes and Environment Team Lead in the Division of Community Family Health and Equity at the Rhode Island Department of Health.*

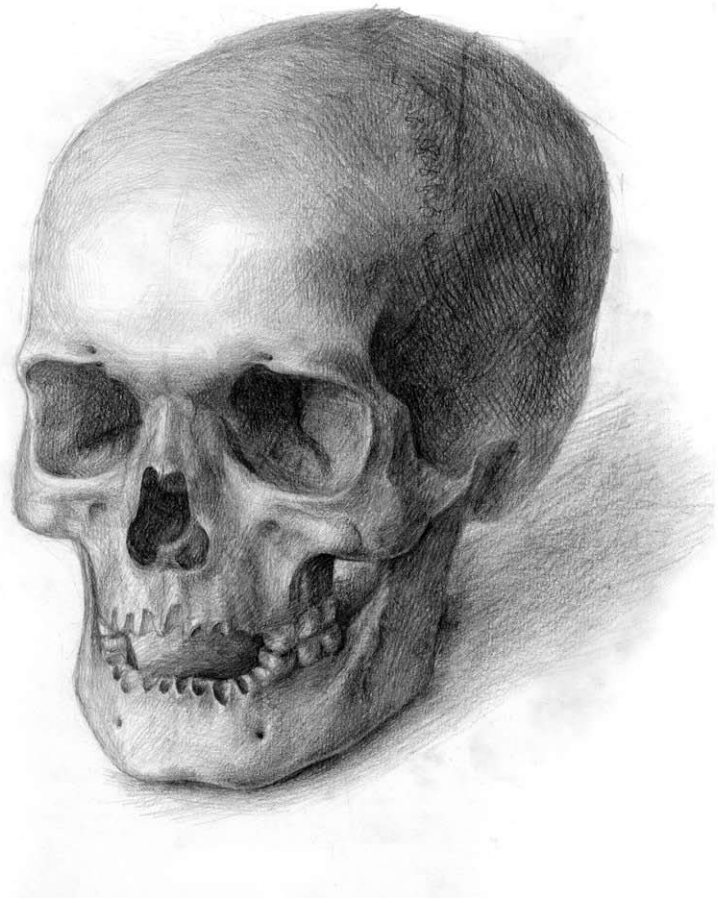
*Edward F. Donnelly, RN, MPH, is Senior Public Health Epidemiologist in the Center for Health Data and Analysis at the Rhode Island Department of Health.*


### Disclosure of Financial Interests

The authors and/or their spouses/significant others have no financial interests to disclose.

## Correspondence


Robert R. Vanderslice, PhD
e-mail: Robert.Vanderslice@health.ri.gov

# Information for Contributors

*Medicine & Health/Rhode Island* is peer-reviewed, and listed in the *Index Medicus*. We welcome submissions in the following categories:

### Contributions
Contributions report on an issue of interest to clinicians in Rhode Island: new research, treatment options, collaborative interventions, review of controversies. Maximum length: 2500 words. Maximum number of references: 15. Tables, charts and figures should be submitted as separate electronic files ( jpeg, tif, or pdf). Each submission should also be accompanied by a short (100-150 words) abstract.

### Creative Clinician
Clinicians are invited to describe cases that defy textbook analysis. Maximum length: 1200 words. Maximum number of references: 6. Photographs, charts and figures may accompany the case.

### Point of View
Readers share their perspective on any issue facing clinicians (e.g., ethics, health care policy, relationships with patients). Maximum length: 1200 words.

### Advances in Pharmacology
Authors discuss new treatments. Maximum length: 1200 words.

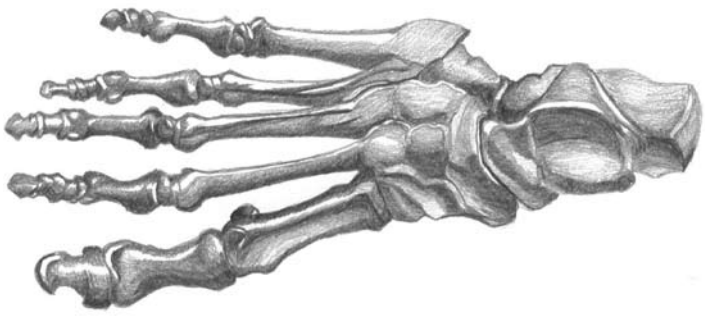
### Advances in Laboratory Medicine
Authors discuss a new laboratory technique. Maximum length: 1200 words.

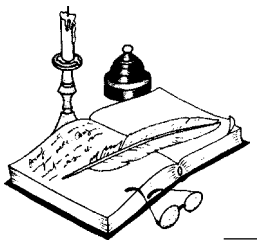
### Images in Medicine
Authors submit an interesng Image, with a 300-400 word explanation.

For the above articles: Please submit an electronic version (Microsoft Word or Text) with the author's name, mailing address, phone, fax, e-mail address, and clinical and/or academic positions to the managing editor, John Teehan, e-mail: jdteehan@rimed.org. For additional information, phone: (631) 903-3389. Faxes may be sent to (401) 826-1926.

Please be sure to provide complete and up-to-date contact information in order to facilitate communication during the editing process.







# Physician's Lexicon

## The Vocabulary of Medical School Administration

**Physicians concern themselves with the** the intricacies of medical word origins principally when the words in question define the name of a disease, a therapeutic agent, an anatomic structure or its physiological function. Once beyond the rigors of medical school, rarely do physicians contemplate the etymologies of the given names of those who supervise or are otherwise instrumental in their professional education.

The Roman legions were structured in multiples of ten and a tenth of a Legion was called a cohort (from the Latin, *cohors*, an enclosed place; a courtyard typically, where legionnaires underwent their training.) A unit of one hundred legionnaires was called a century (Latin, *centum*, meaning one hundred) and its commander, a centurion. A military leader of ten, accordingly, was called a decanus (Latin, *decem*, meaning ten; and hence, *decanus*, a leader of ten; and earlier, from the Greek, *deca*, also meaning ten.) Decanus, was the title given to monastery clerics who oversaw ten monks in training, and his office was called the decanate.

Many of the early European universities were outgrowths of religious centers devoted to scholarly inquiry; and so the administrative words first applied to the Roman military transferred readily to monastery administration and then, centuries later, to the supervisory structure of the universities and their professional colleges.

Decanus (and decanate) corrupted into words such as dean and deanery; and yet another lexical corruption of decanus, doyen, came to mean an authority, a specialist, the senior member of an assembly. And if a woman, then doyenne.

An administrator (Latin, past participle of *administrare,* meaning a manager) supervises an unspecified number of subordinates. The Latin prefix, *ad-*, generally means in the direction of or toward. The root, minister, comes from the Latin *minus-teros*, meaning a servant; and as a verb, to minister means to serve or perhaps even to heal and is the antonym of the Latin, *magister*. To minister, in contemporary English, has come to mean to care for.

Provost is from the Latin, *praepositus*, meaning to put forth, to preside. And chancellor, again from the Latin, *cancellarius*, is the title given to a high church official who by custom is seated near the latticed enclosure, called the chancel, in front of the altar. To cancel meant, originally, to be enclosed by a lattice but gradually it took on the meaning of something to be crossed out, to be cancelled. And cancellous, as an adjective to describe spongy bone, is from the same source.

– Stanley M. Aronson, MD

Rhode Island Department of Health
Michael Fine, MD
Director of Health

# Vital Statistics

Edited by Colleen Fontana, State Registrar

## Rhode Island Monthly Vital Statistics Report Provisional Occurrence Data from the Division of Vital Records

| Underlying Cause of Death | Reporting Period | | | |
|---|---|---|---|---|
| | May 2011 | 12 Months Ending with May 2011 | | |
| | Number (a) | Number (a) | Rates (b) | YPLL (c) |
| Diseases of the Heart | 228 | 2,388 | 226.7 | 3,434.5 |
| Malignant Neoplasms | 174 | 2,255 | 214.1 | 5,611.5 |
| Cerebrovascular Diseases | 27 | 440 | 41.8 | 737.0 |
| Injuries (Accidents/Suicide/Homicide) | 56 | 627 | 59.5 | 9,503.0 |
| COPD | 46 | 546 | 51.8 | 397.5 |

| Vital Events | Reporting Period | | |
|---|---|---|---|
| | November 2011 | 12 Months Ending with November 2011 | |
| | Number | Number | Rates |
| Live Births | 912 | 11,769 | 11.2* |
| Deaths | 756 | 9,782 | 9.3* |
| *Infant Deaths* | (2) | (77) | 6.5# |
| *Neonatal Deaths* | (2) | (60) | 5.1# |
| Marriages | 412 | 6,266 | 5.9* |
| Divorces | 308 | 3,330 | 3.2* |
| Induced Terminations | 289 | 4,096 | 348.0# |
| Spontaneous Fetal Deaths | 47 | 637 | 54.1# |
| *Under 20 weeks gestation* | (41) | (545) | 55.7# |
| *20+ weeks gestation* | (6) | (90) | 7.6# |

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,053,209. (www.census.gov)

(c) Years of Potential Life Lost (YPLL).

***Note:*** *Totals represent vital events that occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.*

* Rates per 1,000 estimated population
\# Rates per 1,000 live births

THE RHODE ISLAND MEDICAL JOURNAL
The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publications Committee
VOLUME 1
NUMBER 1
PROVIDENCE, R.I., JANUARY, 1917
PER YEAR $2.00
SINGLE COPY, 25 CENTS

## Ninety Years Ago, April, 1922

H.P. Lowell, MD, opens the May issue with a talk on medicinal plants native to Rhode Island. He discusses over 300 plants within the state known to have some medicinal use, citing particularly the lore and traditions of the native American population. He also notes a 1783 study by Johan David Schopf in which, as part of a study of American plants, he learned much on the subject from Indian tribes in Rhode Island and Connecticut. The author goes on to provide a number of highlights (including uses) of Rhode Island plants such as: golden club, star grass, adder's tongue, Amrican hellebore, wild spikenard, leatherwood, cranesbill, blood-root, black snake-root, poke-weed, red-root, American senna, and pipissewa. He also notes the presence of poison hemlock which is native to Europe and Asia, but has become naturalized in Rhode Island.

Chiropractics comes under fire again in an editorial decrying the public's faith in the practice and its lies. Serious medical conditions stand the risk of going undiagnosed by the practices and assurances of chiropractors and, thus, it is important to educate the public on the dangers involved.

In a related editorial, the topic of specialization comes up. When a general practitioner sends a patient to a specialist it is assumed that the GP has good knowledge of the specialist's abilities and knowledge in order to best help the patient. While the state of Rhode Island licenses general practitioners, there is no system in place for monitoring the quality of specialists and no system to keep someone from advertising themselves as a specialist. The editorial states: "Young men are flocking into specialties, some are properly trained, many are not. Likewise, older men are turning to them because the work is not so arduous or the remuneration is better. The result is that much poor work is being done in the name of surgery and will, if unchecked, lessen the repute in which doctors are now held. Some such is necessary in justice to the public and properly trained specialists, and works no hardships on physicians in general, for it does not prevent them from treating any disease they feel themselves qualified to treat.

## Fifty Years Ago, April, 1962

In the President's Message, noting the 150th anniversary of the Medical Society, Arthur E. Hardy, MD, touches on both the past and future. He writes, "During the next twelve months the answers to questions concerning medicine's continued freedom in this country will further unfold. Will we be caught asleep in a blanket of indifference, or will we be wide awake, keeping abreast of the times and truly advancing the art and science of medicine and the health and welfare of our people? If we can learn from some of our mistakes and take positive action where we have been inactive or neutral; become informed instead of remaining uninformed; if through individual example we restore the image of the physician with the public, we will defeat the present challenge to our freedom."

Thomas McOsker, MD, looks at neck injuries and rear-end collisions, noting that in the violent work of traffic today, such injuries are bcoming much more common. He discusses types of injuries as well as related injuries to the head and arms. In an overall study, the author mentions that about three quarters of patients who are injured in a rear-end collision have symptoms referable to the neck, but an extremely low percentage of these individuals will have any X-ray evidene of injury to the bony structure of the neck.

A study from the Public Health Service makes mention that hospitals in the United States are becoming increasingly dependent upon graduates of foreign medical schools to help fill out their house staff positions. In 1951, 2,100 graduates of medical schools outside the United States were serving as hospital interns and residents; by 1960, this figure had reached 9,500.

## Twenty-five Years Ago, April, 1987

John M. Wiecha brings up the topic of holisitic health and its implications for physicians in a changing world. He suggests that the rise in interest in holistic health may come from a growing dissatisfaction with conventional medicine, and that two-thirds of Americans are losing faith in doctors who they believe are too interested in simply making money. Less than half Americans feel that doctors explain things well. While not encouraging or even listing the practices of holistic helath specialists, the author does underscore one particular concept—that of encouraging, when appropriate, a patient's independence and responsibility. In this context, the physician is a guide, a facilitator, and, most importantly, an educator. Many reports encourage physicians to pay particular attention to their communiction skills as this correlates with patient satisfaction and compliance. The author recommends *The Holistic Health Handbook* from the Berkeley Holistic Health Center as an excellent overview on holisitc health.

H. Denman Scott, MD, MPH, addresses the need for long-term care solutions in relation to the baby boomer generation which has entered middle age and which, in thirty years time, will become senior citizens. He points out a need to develop a wide array of long-term care services which can be provided for the patient at the right time and the right place. Currently, the long-term care options, such as home care and nursing home care, are too narrow. Dr. Scott also notes that 75 percent of the people in nursing homes are women. Thus, the issue of long-term care is primarily a women's health issue. He states, "We can only hope that organized women's groups will take an active interest in the long-term care issue. If this happens, long-term care is more likely to receive the social attention which it deserves."

# The Name of Choice in MRI

'OASIS' 1.2 Tesla open-sided scanner

## Open MRI
### of New England, Inc.

- High Field Open-Sided and Short-Bore Systems
- Fast appointments and reports
- Insurance authorization services, physician web portal and EMR system interfaces

Open MRI
of
New England, Inc.

## ADVANCED
### Radiology, Inc.

- Low dose Multislice CT systems
- Digital xray, bone density and ultrasound
- Insurance authorization services, physician web portal and EMR system interfaces


Brightspeed low dose CT System

ADVANCED Radiology, Inc.

525 Broad St. • Cumberland
T 725-OPEN (6736) F 726-2536

1002 Waterman Ave • East Providence
T 431-5200 F 431-5205

148 West River St • Providence
T 621-5800 F 621-8300

501 Great Road • North Smithfield
T 766-3900 F 766-3906

335 Centerville Rd • Warwick
T 732-3205 F 732-3276

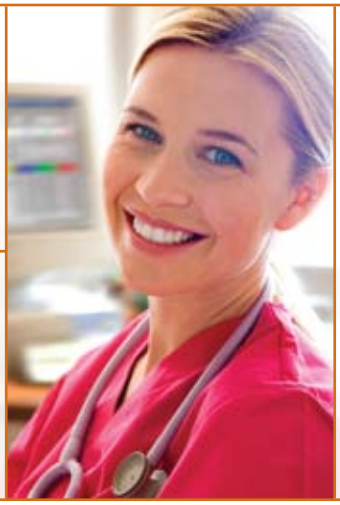
101 Airport Rd • Westerly
T 315-0095 F 315-0092

# NUMBERS THAT WORK AS HARD AS YOU DO

**22,800** CALLERS ASSISTED IN 2011

**1** PHONE CALL IS ALL IT TAKES

**24/7** PHONE ACCESS TO LIVE EXPERTS



**25,000** CREDENTIALING LETTERS ISSUED

What is great service? For NORCAL Mutual insureds, just 1 phone call is all it takes for great service. That means calling during business hours and immediately reaching a live, knowledgeable, friendly expert. After hours, it means promptly receiving a call back from a professional qualified to help with your issue. No automated telephone tango. Questions are answered and issues resolved — quickly. We're on call 24 hours a day, every day of the year. Great service brings you peace of mind. To purchase your NORCAL Mutual coverage call RIMS Insurance Brokerage at 401-272-1050. **Great service 24/7. Hard-working numbers you can count on.**

CALL 1-800-652-1051 OR VISIT NORCALMUTUAL.COM

Proud to be endorsed by the Rhode Island Medical Society.

NORCAL Mutual Insurance Company®

*Our passion protects your practice*