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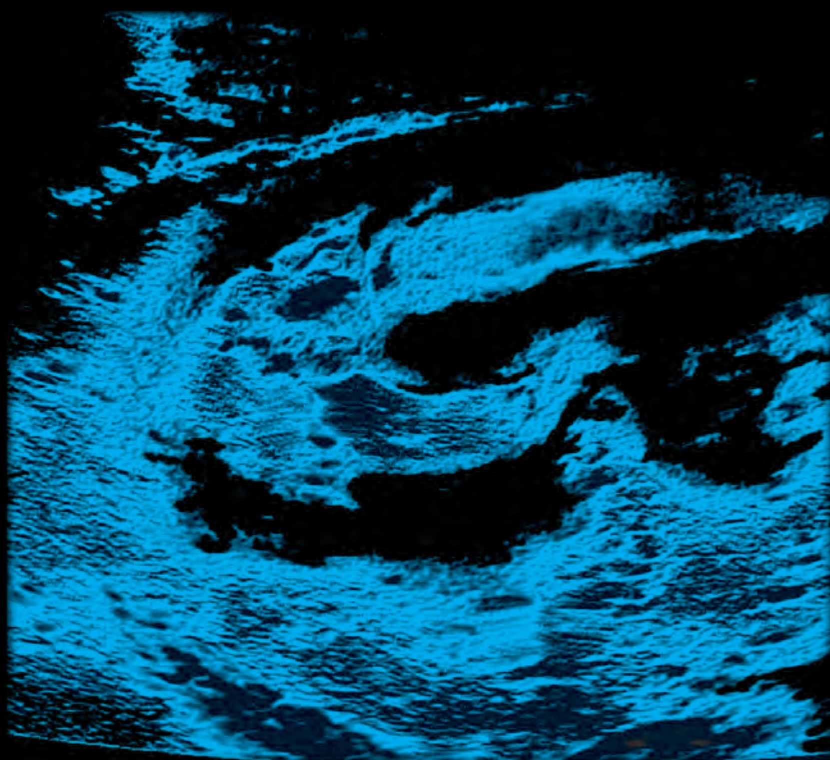

CME Pediatric Appendicitis US: Practical Considerations

Assessing MRI Facility Damage Post-Hurricane: Best Practices and Safety Considerations

Klippel-Feil Syndrome Essentials, Part 1: Embryological Development and Genetic Mechanisms

Transforming Radiology: The Role of AI, Automation, and Advanced Cloud Computing

Alzheimer Disease Imaging in the Age of Disease-Modifying Therapies

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'Tis the Season

Erin Simon Schwartz, MD

If it's late November, then it's RSNA! The annual gathering of radiology professionals from around the globe is almost upon us. It's one of my favorite meetings of the year. As a pediatric neuroradiologist, I tend to travel in a pretty small sphere, professionally speaking. One of the many things I love about RSNA – and my position with *Applied Radiology* – is the opportunity to interact with radiologists and other imaging professionals across all subspecialties.

It's fascinating to hear the latest developments throughout our entire field being presented by respective leaders and experts, not to mention learning how to optimize and automate those all-important noninterpretive tasks that affect everyone, regardless of your subspecialty.

This year's theme, *Building Intelligent Connections*, seems more relevant than ever, especially with the increasing offerings in artificial intelligence promising to help us work more efficiently and effectively under those crushing workloads. Also, at a time when folks in the United States and around the world are feeling less unified, fostering shared values and connections is greatly needed.

Please do stop by our booth just inside the entrance to the South Exhibit Hall (Booth 1500), if you will be attending in person. You might even walk away with a helpful item. We so enjoy meeting our readers and hope you enjoy putting faces to the names you've been reading in our pages over the years.

Digital Edition Greenlight

As mentioned in our previous issue, *Applied Radiology* is moving to an all-digital format in 2025, beginning with the January-February edition. We are excited to support the much-needed sustainability efforts in publishing (and beyond) while maintaining the high-quality content you've come to expect from *AR* over the decades. We look forward to embracing a greener future together.

New Editorial Advisory Board Member

Please help us welcome **Abass M. Noor, MD,** to the Global Health section of our Editorial Advisory Board. Dr Noor holds an Endowed Chair and serves as director of Pediatric Radiology Global Outreach and Education in the Department of Radiology at the Children's Hospital of Philadelphia. He is also the director of the Health Equity Leadership Track for the Radiology Residency program at the Perelman School of Medicine at the University of Pennsylvania as well as being the program manager for RAD-AID Botswana. Dr Noor brings a wealth of experience to the section, and we look forward to reading his contributions to our Global Health column.

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Pediatric Appendicitis US: Practical Considerations

Description

The techniques described in this article will aid the radiologist in performing and interpreting a US exam for appendicitis. The article details assessment criteria pertaining to the normal appendix and acute appendicitis, differential diagnosis and potential pitfalls, and considerations for the reporting of right lower quadrant US findings.

Learning Objectives

Upon completing this activity, the reader should be able to:

1. Apply US techniques to identify and characterize the pediatric appendix and guide sonographers who are less experienced in providing the examination.
2. Rule out differential diagnoses and understand potential pitfalls surrounding US applications for diagnosing pediatric appendicitis.

Target Audience

- Radiologists
- Related imaging professionals

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5. Complete the evaluation.
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Estimated Time for Completion

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CME DETAILS ON PAGE 5

Pediatric Appendicitis US: Practical Considerations

Artineh Hayrapetian, MD; Mathurika Jeyasingam, MD; Michael Francavilla, MD

Introduction

Appendicitis is a frequent pediatric surgical emergency in the United States, occurring at a rate exceeding 100 per 100,000 person-years.[1] It is more frequent in biological men, individuals of White ethnicity, and those ages 10-19.[2,3] Classic symptoms are absent in up to one-third of cases.[2] The main causes of inflammation include bacterial overgrowth, lumen obstruction, and ischemic mucosal damage.[3] Abdominal pain, especially in the right lower quadrant (RLQ), along with guarding, tenderness, and elevated inflammatory blood markers, including C-reactive protein and white blood cell count, raise clinical suspicion.[4-6]

While many sites perform CT and MRI for suspected appendicitis, US is often the initial imaging modality in staged clinical pathways that incorporate risk stratification owing to its cost-effectiveness, frequently more rapid availability and performance, absence of contrast requirement, and lack of radiation exposure.[2,3,7] US demonstrated a pooled sensitivity of 72.5% and specificity of 97.0% in one multicenter study.[7] However, US is highly operator dependent. Visualization of the appendix may be challenging owing to factors such as operator experience, patient body habitus, excessive bowel gas, and variable appendix location.[3,6] This article describes techniques for the sonographer and radiologist to identify and characterize the pediatric appendix. We focus on how to perform the US study so that the radiologist can do so personally or provide guidance to sonographers who are less experienced in US of the pediatric appendix.

Technique

The examination begins by positioning the child supine and asking the patient to indicate the location of maximum pain or tenderness to help identify the inflamed appendix's position. If a specific point is mentioned, initiate the examination from that spot.[2,3] Distraction techniques, such as using electronic tablets, toys, or pointing to images during the US examination, can be beneficial to help the child remain distracted from any pain and help them keep still. High-frequency transducers (eg, 6-15 MHz linear transducers) are ideal,[2,5] while low-frequency transducers (eg, 1-6 MHz curved array) are suitable for patients with a large body habitus or a deep-seated appendix in whom greater penetration depth is necessary.[2,3] Harmonic imaging should be used if available. An excessively deep field of view decreases axial resolution and the ability to visualize small caliber structures such as the normal appendix (3-5 mm). To facilitate visualization of a normal appendix, initially set the depth of the scan to include the RLQ bowel, but not deeper structures such as the posterior aspect of the iliac wing and, in younger children, the vertebrae. Start scanning with light to moderate pressure, applying additional pressure on expiration.[3] Normal bowel can be displaced by a technique called graded compression. This technique involves applying light pressure with the transducer during the initial anterior abdominal sweep.[8] The goal is to displace and compress bowel gas and fluid from the cecum and the

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Figure 1. The iliac vessels (red and blue) and psoas major muscle are helpful landmarks. This transverse image shows the posterior iliac bone. The depth of field should be reduced to better visualize small structures such as the appendix.

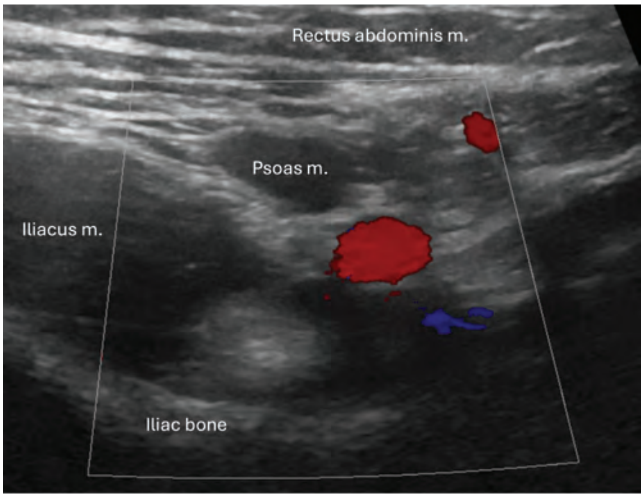


Figure 2. Grayscale image of the right lower quadrant shows the cecum in the transverse plane. The colon (seen here in cross-section) is located laterally, contains air with "dirty" shadowing, and is larger caliber than the adjacent small bowel (shown longitudinally).

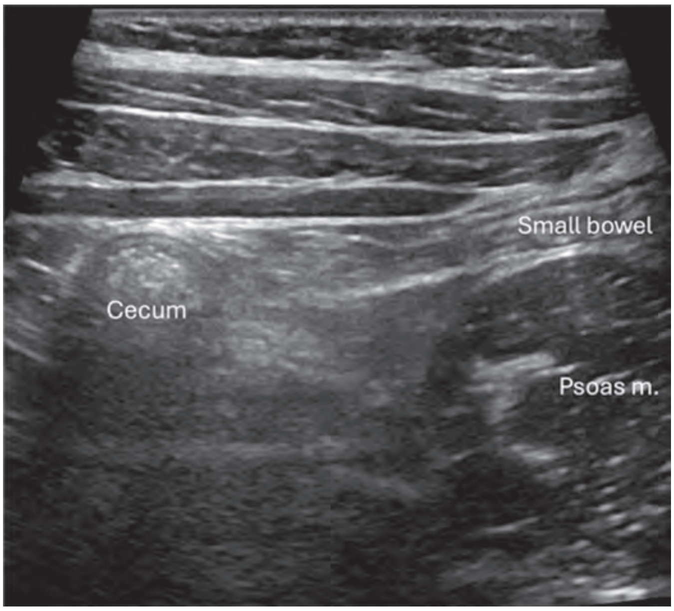
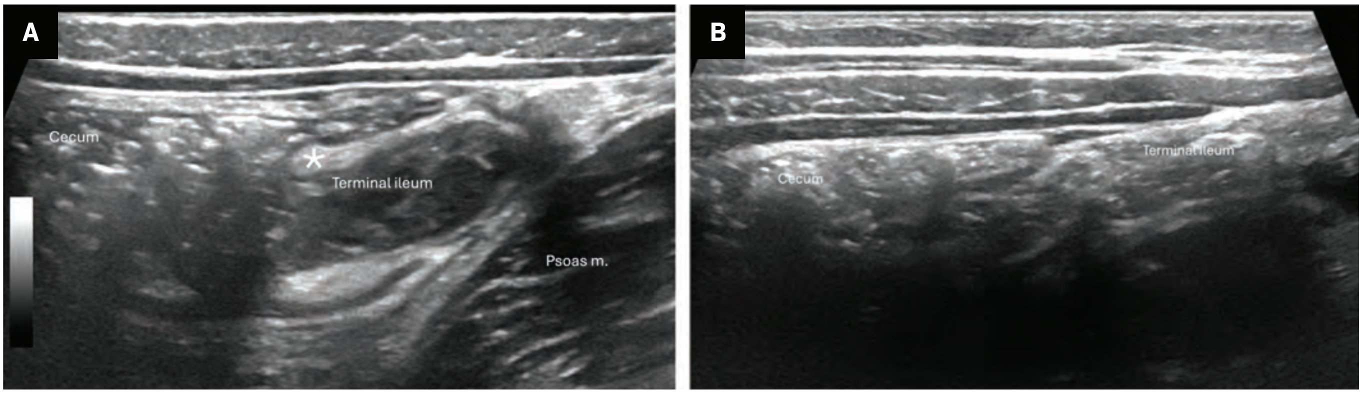


Figure 3. When possible, radiologists and sonographers should locate the terminal ileum (TI) and cecum. The ileocecal valve (ICV) (*) is variably identified and visible in (A) but not (B).



terminal ileum (TI) with improved visualization of the noncompressible inflamed appendix with gradually increasing pressure.[8] Important anatomic landmarks include the cecum, TI, psoas muscle, and iliac vessels (Figure 1). The appendix is typically found inferior to the cecum and TI, and anterior to the psoas muscle and iliac vessels.

Our practice is to first identify the ascending colon and cecum in the transverse plane (Figure 2). Compared with the small bowel, the colon is located more laterally, contains more air, often resulting in "dirty" shadowing,[9] and is generally of a larger caliber, making it relatively easy to identify in many patients. Second, we move the transducer inferiorly to attempt to locate the TI and ileocecal valve (ICV) (Figure 3). The appendix usually arises from the cecum on the same side as the ICV and 2-3 cm inferior to the ICV, making visualization of the ICV a helpful landmark.[2,3] Identifying the TI is additionally useful to exclude potentially confounding diagnoses (eg, inflammatory bowel disease) and ensure that the TI is not mistaken for the appendix. The TI should be documented with grayscale and color Doppler imaging to evaluate for hyperemia. The appendix is variably positioned and, in more than 50% of cases, it is retrocecal.[10] In addition to graded compression to help identify the appendix, posterior manual compression aids in reducing the distance from the transducer to the bowel, facilitating visualization, especially in patients with larger

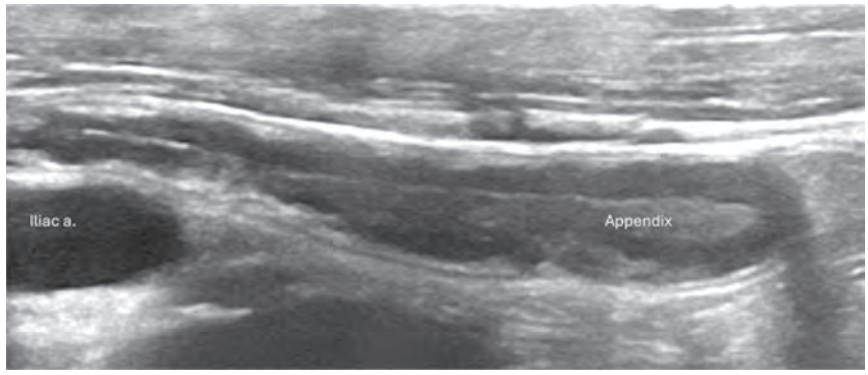
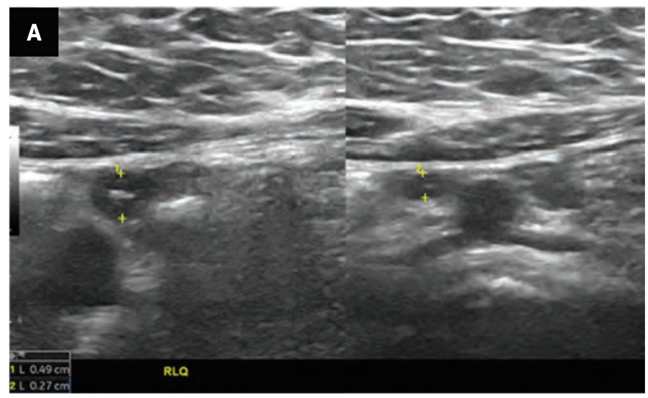


Figure 4. Longitudinal image of a normal appendix (calipers) with expected wall structure and echogenic luminal contents



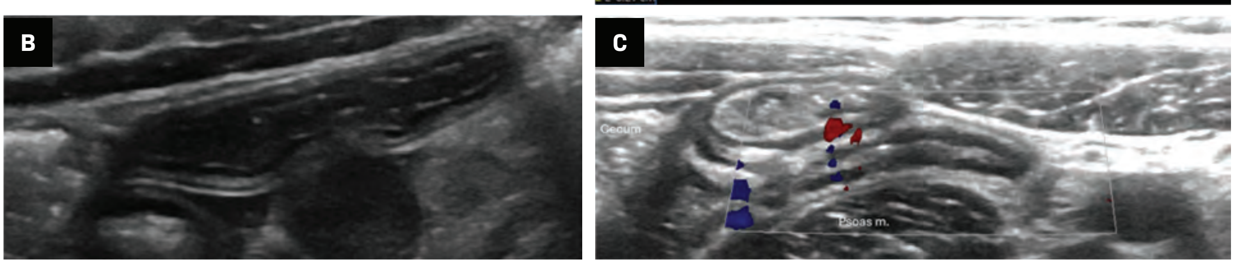


Figure 5. Transverse images (A). Compressibility should be assessed by measuring the diameter of the appendix, ideally in a side-by-side grayscale view (left, without compression; right, with compression). Longitudinal images showing the cecum origin in grayscale (B) and the blind-ending tip on color Doppler (C). Note that the appendix is not displaced from the adjacent structures by inflamed fat or edema; there is no fat stranding.

abdomens.[2,3] Placing the patient in a left lateral decubitus can help position the cecum and TI medially, improving access to the retrocecal region.[3] If the appendix remains occult, repositioning the patient may cause bowel gas movement, enhancing visualization.[2] Some have suggested adding a posterior approach.[11]

Once identified, the appendix should be documented in grayscale and color Doppler images. The Doppler pulse repetition frequency should be very low. Longitudinal images showing the blind-ending tip (Figure 4) and, if possible, origin from the cecum will prove that the structure identified is the appendix and not another piece of bowel (Figure 5). The presence of hyperemia is best demonstrated in longitudinal images. Compression images showing transverse luminal diameter should be obtained side-by-side (Figure 5).

Morrison pouch and the pelvis are commonly examined with low-frequency transducers and a deeper field of view to evaluate for free fluid or abscess.[3] The appendix may be seen suspended within any free fluid.[2]

Normal Appendix

The normal appendix is a compressible, blind-ending, tubular structure featuring 5 distinct layers in the wall, although only 3 may be visible. The innermost layer is a hyperechoic mucosal linear structure containing lymphoid tissue.[2] Appendiceal diameter is typically less than 6 mm and does not change with age.[12] The maximal mural thickness, however, does vary with age and a maximum of 3 mm should be considered normal for those under 6 years old.[12,13] There should be minimal color Doppler signal in the appendix wall (Figure 5). Gas in the appendix typically indicates the absence of acute appendicitis.[14] The appendix is often enlarged in patients with cystic fibrosis in the absence of

Figure 6. Primary signs of appendicitis (A). Calipers are placed on the outer walls of the dilated, fluid-filled, hyperemic appendix. Note the displacement of the appendix away from adjacent structures by the thickened, echogenic surrounding fat stranding, a secondary sign of appendicitis. Free fluid (*) is also present. Color Doppler (B) shows hyperemia within the appendiceal wall, a primary sign of appendicitis. Free fluid is also demonstrated.

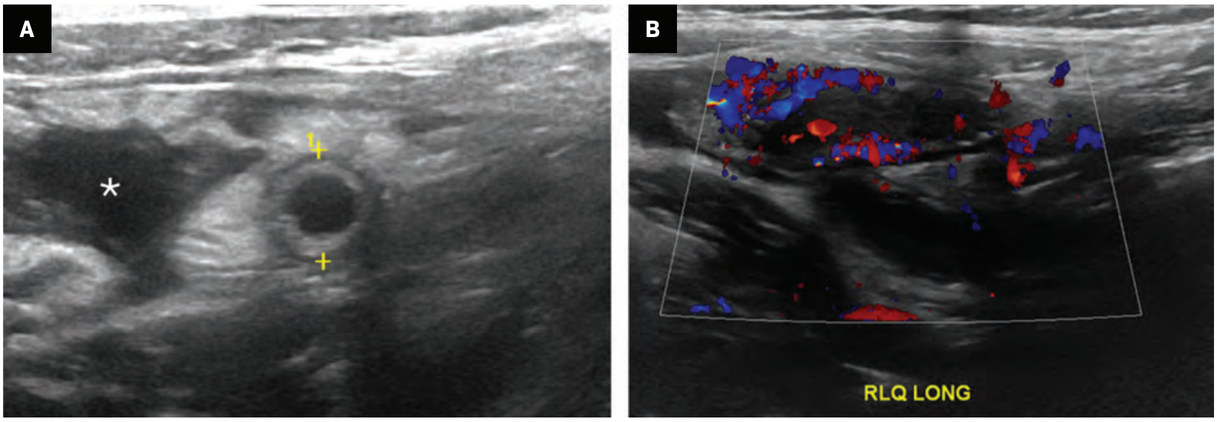
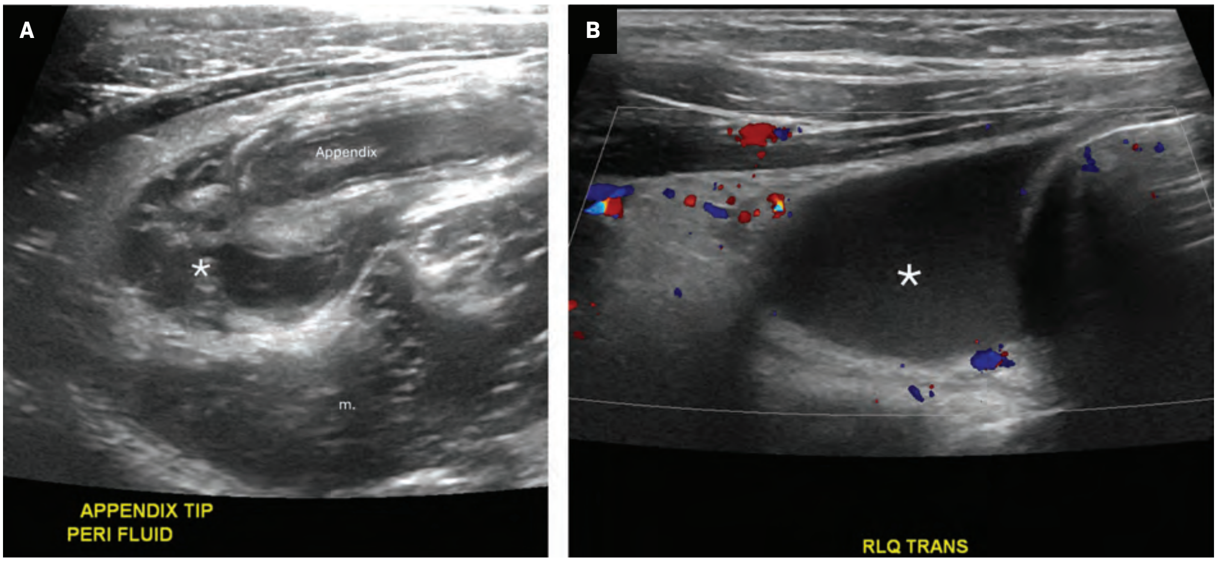


Figure 7. Grayscale US image (A) of the right lower quadrant (RLQ) shows a dilated, inflamed appendix with surrounding fat stranding and an adjacent complex fluid collection (*) in a patient with a perforated appendicitis and abscess. Color Doppler image (B) of the RLQ shows complex free fluid (*), hyperemia, and fat stranding.



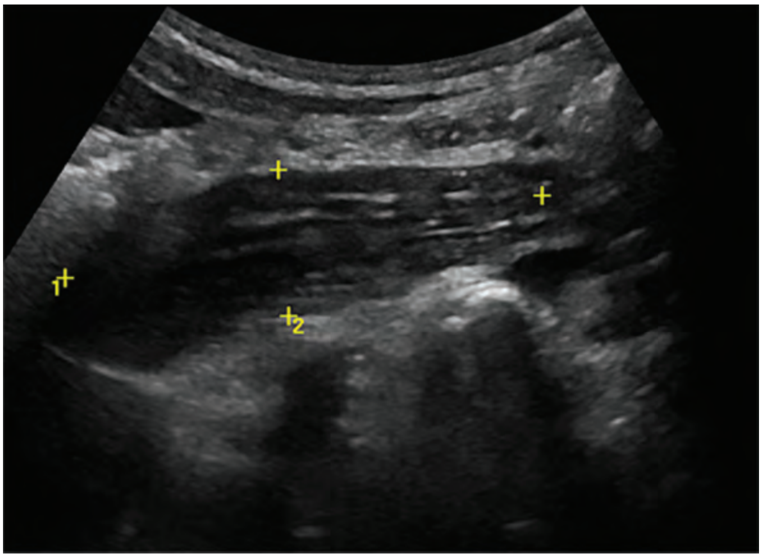
appendicitis (mean diameter of 8.3 mm).[15] Visualization of the normal appendix in its entire length, including its tip, definitively excludes appendicitis.[16] However, an appendiceal US in which the appendix is not visualized and no inflammatory findings are present in the RLQ has been shown to have a high negative predictive value.[7]

Acute Appendicitis

Appendix thickening, maximal tenderness over the thickened appendix, noncompressibility, (large) appendicolith, and hyperemia (Figure 6) are the primary US indicators of appendicitis.[16] While 6 mm is the conventional cut-off for appendiceal diameter for US, it has been suggested that specificity may be improved with a cut-off of 7 mm.[17] Note that this cut-off value does not apply to CT or MRI.

Secondary signs of appendicitis include RLQ mesenteric fat stranding, the presence of a complex fluid collection, mesenteric lymphadenopathy, and/or periappendiceal fluid.[16] Fat stranding appears as mesenteric thickening and hyperechogenicity (Figure 7). Hyperechoic, thickened fat is highly specific for inflammatory disease in the RLQ.[18,19] An appendicolith has been variably associated with acute appendicitis.[20] An additional concerning finding is the presence of an excessive volume of free abdominopelvic fluid. A small amount of simple free fluid has low specificity for appendicitis in both boys and girls, but a moderate to large amount has been reported to be highly

Figure 8. Grayscale US image shows calipers placed around the iliopsoas muscle, which can be mistaken for an inflamed appendix



specific.[19] Complex fluid is highly specific for perforated appendicitis.[20] Focal, complex fluid suggests the presence of an abscess (Figure 7).

Differential Diagnosis and Pitfalls

Various conditions can mimic appendicitis, including enterocolitis, inflammatory bowel disease, appendiceal lymphoid hypertrophy, mesenteric adenitis, Meckel diverticulum, and tumor. Enterocolitis and inflammatory bowel disease are causes of RLQ pain that can mimic appendicitis. It is crucial to recognize the cecum and ilium and differentiate them from the appendix. The TI, ending at the ICV, is not blind-ending and exhibits peristalsis. There may be reactive inflammation of the cecum and ileum in acute appendicitis, but the amount of inflammation tends to be relatively minor. Conversely, inflammatory bowel disease is often most severe at the TI. Involvement of the appendix can be seen relatively frequently in Crohn disease.[16]

Lymphoid hyperplasia is identified by clusters of more than 10 lymphoid nodules with follicles exceeding 2 mm in size. This condition can reduce the compliance of the appendiceal wall, leading to noncompressibility on US and increasing the appendix's maximum diameter beyond 6 mm, which may result in a false-positive diagnosis of appendicitis.[21] Distinguishing between lymphoid hyperplasia and appendicitis is aided by identifying periappendiceal fluid collection and a lamina propria thickness of 1 mm or less, which are considered the most reliable indicators.[21]

Mesenteric adenitis can be identified by reactive mesenteric lymph nodes, with a short axis of more than 5 mm as well as more than 3 lymph nodes in the small bowel mesentery without acute inflammation.[3,22,23] Meckel diverticulum is a blind-ending structure arising from the distal ileum, often associated with rectal bleeding. Meckel diverticulitis should be suspected if a cyst-like structure with gut US signature is identified and the structure has anomalous vessels and signs of wall inflammation on color Doppler.[24] Origin of the inflamed structure from the cecum excludes a diagnosis of Meckel diverticulum.[25]

Other tubular-appearing structures in the RLQ can mimic an inflamed appendix. The psoas muscle, when imaged obliquely, can be mistaken for an inflamed appendix (Figure 8).[16] Tuboovarian abscess could be mistaken for a perforated appendicitis.[26]

Reporting Right Lower Quadrant US

After obtaining the patient's history and employing the appropriate technique, the report should assess the appendix, detailing its diameter, compressibility, Doppler flow, presence of appendicolith, and any secondary signs. Additionally, the report should describe findings such as other bowel wall thickening or enlarged lymph nodes. Structured reporting may decrease the use of CT and reduce the rate of negative appendectomies.[27] If the appendix is not identified and any secondary signs are present, CT or MRI should be performed. In cases where clinical suspicion remains high despite a negative or inconclusive US, referrers may opt to perform further imaging studies, such as CT or MRI, before arriving at a definitive diagnosis or ruling out appendicitis.[16,28]

Conclusion

Appendicitis is the most prevalent surgical emergency in children. The techniques described in this article should aid the radiologist and sonographer in performing an US examination for appendicitis and the radiologist in interpreting the findings.

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REVIEW

Assessing MRI Facility Damage Post-Hurricane: Best Practices and Safety Considerations

Laura P. Del Rey-Vasquez, PhD, RVT(ARDMS), RT(R)(MR)(ARRT), MRSO(MRSC)

Introduction

While direct testimonials from MRI technologists regarding post-hurricane damage to MRI scanners may not be widely published, several reports and studies from health care facilities have documented the significant impact of hurricanes on medical imaging equipment, particularly MRI systems (Table 1). Hurricanes Milton and Helene recently inflicted substantial damage along the Gulf Coast, creating challenges for health care facilities equipped with MRI scanners. These events underscore the importance of immediate damage assessments and the implementation of safety protocols to protect MRI equipment, personnel, and patients.[1]

MRI presents unique safety challenges, largely due to the presence and interaction of 3 distinct magnetic fields integral to the imaging process.[2] These fields are:

Table 1. Issues MRI Centers Commonly Face After Hurricane Damage

CHALLENGE	DESCRIPTION
Water damage	Flooding and water intrusion are major risks, especially in coastal areas. Water can cause humidity issues, leading to condensation that damages the MRI scanner's electrical and mechanical components.
Power outages and quenching	MRI scanners are highly sensitive to power disruptions. Extended power outages can cause quenching, where the magnet loses superconductivity due to helium evaporation, resulting in downtime and costly repairs.
Quench pipe blockages	Wind-driven rain during hurricanes can block or obstruct quench pipes, which are essential for safely venting helium gas. Blockages can create risks when restarting the system after the storm.
Structural damage and delays	Structural damage to the facility can lead to delays in restarting MRI scanners as inspections and repairs must be completed to ensure safe operation.

1. **Static magnetic field (B0).** This primary and constant field, generated by the MRI scanner, creates a powerful magnetic environment that affects all ferromagnetic objects and materials within its vicinity. The high-strength static field poses risks such as projectile accidents and interference with electronic implants or devices.
2. **Radiofrequency magnetic field (B1).** This field is essential for generating MRI signals and is time-varying during scans. However, it can lead to patient heating and potential tissue burns if not appropriately managed. The B1 field interacts with human tissues and implanted devices, necessitating rigorous safety protocols to prevent adverse effects.
3. **Gradient magnetic fields (dB/dt).** These rapidly varying

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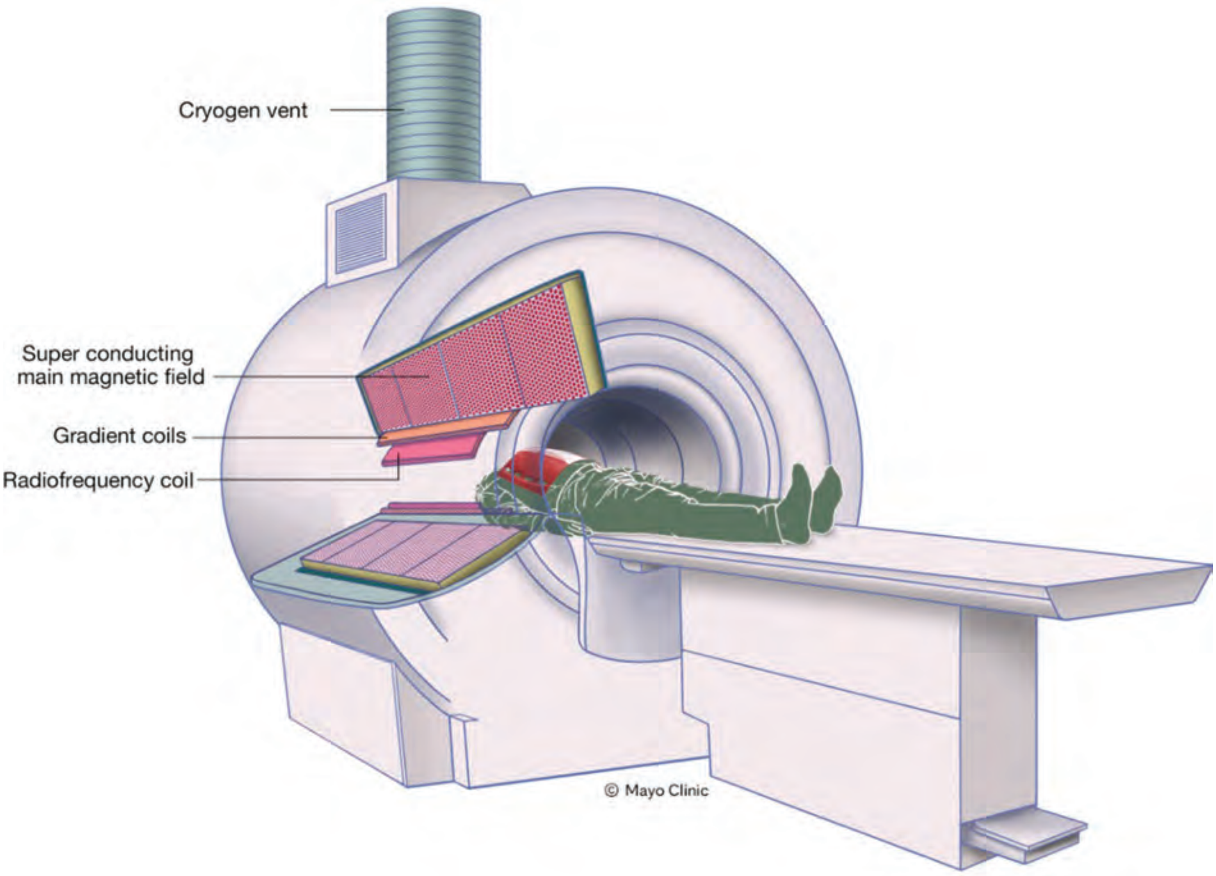
Figure 1. Schematic diagram of a superconducting magnet MRI system. Closest to the bore wall, adjacent to the patient, is the radiofrequency (RF) coil, which plays a critical role in transmitting and receiving signals. Proximity to this coil can pose a risk of patient heating or burns if safety protocols are not observed. Surrounding the RF coil are the gradient coils, which generate rapidly changing currents responsible for the characteristic acoustic noise during imaging and may also induce peripheral nerve stimulation. The outermost ring represents the static magnetic field (B0), which generates strong translational and rotational forces on ferromagnetic objects. Each of these electromagnetic fields can interact with metallic objects or implanted medical devices, emphasizing the importance of stringent safety measures within the MR environment. From: ACR Committee on MR Safety. ACR Manual on MR Safety (manual). Reston, VA: American College of Radiology, 2024, p. 10; used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.[2]



fields, responsible for spatial encoding in MRI, create risks of peripheral nerve stimulation. While they contribute to the production of clear and detailed images, they can induce electrical currents in the body, leading to discomfort or even unintended muscular contractions.

The complex interplay of these 3 magnetic fields (Figure 1) is at the heart of MRI technology. However, their presence also introduces distinct safety challenges, necessitating stringent protocols to safeguard both patients and health care personnel. Recognizing and mitigating the risks associated with these fields is crucial to maintaining safe MRI practices and ensuring the integrity of diagnostic outcomes.

In addition to these inherent risks, MR systems rely heavily on maintaining superconductivity within the magnet coils. A critical failure, known as a helium quench, occurs when liquid helium, essential for cooling the superconducting magnets, rapidly transitions to a gaseous state due to an equipment malfunction, power disruption, or operational error. This sudden vaporization can compromise the magnetic field, damage sensitive components, and pose serious safety hazards. To mitigate such risks, facilities must implement robust safety measures, including pressure relief systems and emergency ventilation protocols (Figure 2). Proper training with

Figure 2. A quench event in a research MRI magnet (left) shows the release of gaseous helium. Such events necessitate stringent safety protocols, including appropriate ventilation systems and emergency procedures to protect personnel from cryogenic hazards and asphyxiation risks. In an installation of a quench tube in an MRI (right), the blue arrow points to the tube and the green arrow points to the vent in the ceiling. Quench tubes serve a crucial role in venting helium gas from the MRI scanner during a quench event. Proper attachment and secure connections between the quench tube and vent are vital to maintaining safety within the MRI suite, especially during quench scenarios. *Printed with permission from MRIquestions.com.*

Table 2. American College of Radiology Guidelines

KEY POINTS	AMERICAN COLLEGE OF RADIOLOGY GUIDELINES REGARDING MRI SAFETY *BEST PRACTICES FOR MRI FACILITY SAFETY*
Environmental controls	Implement dehumidifiers to maintain 30-50% relative humidity levels in MRI suites to protect against condensation.
Electrical power management	Equip MRI systems with uninterruptible power supplies and backup generators to mitigate risks of quenching.
Quench pipe inspections	Inspect quench pipes for blockages, especially after hurricanes, to prevent obstructions that could cause equipment damage.

regular maintenance is essential to managing quench events effectively while sustaining MRI safety and performance.[3]

In the context of disaster preparedness, health care facilities must place a high priority on establishing comprehensive safety and recovery strategies. These strategies should encompass thorough risk assessments, environmental controls, and routine technical inspections. The following guidelines, grounded in research and expert recommendations, provide a framework for addressing hurricane-related threats to MRI systems. Implementing these best practices is vital for minimizing risks, ensuring a safe recovery, and preventing long-term damage to critical equipment.

Primary Threats to MRI Facilities: Wind and Water

Hurricanes pose 2 primary threats to MRI facilities: wind and water. Wind-driven debris can cause structural damage, leading to water intrusion. Heavy rainfall, which often exceeds 20 to 40 inches during hurricanes, poses additional risks by overwhelming drainage systems, particularly in low-lying areas. This increases the likelihood of water entering the facility, which can affect MRI equipment by increasing humidity levels and causing condensation on sensitive components.[4]

The American College of Radiology (ACR) recommends (Table 2) maintaining relative humidity levels of 30% to 50% within MRI suites using dehumidifiers. Such environmental controls help protect MRI systems from damage caused by condensation. Proper inspections should be conducted following severe storms to identify any water intrusion that may threaten equipment functionality.[2]

Impact on MRI Quench Pipes

Quench pipes, essential safety components for the controlled release of helium gas during a magnet quench, are particularly vulnerable to wind-driven rain. If water or debris obstructs these pipes, it can prevent proper venting during a quench, leading to significant equipment and structural damage.[5] It is critical to inspect

Table 3. MRI Chiller Features, Troubleshooting, and Key Practices

ASPECT	KEY POINTS	DESCRIPTION
Chiller failures	No cooling	Loss of effective cooling results in uncomfortable temperatures within the facility.
	Error codes	Modern chillers have built-in diagnostics that display error codes on the control panel to help identify issues.
	Automatic shut-off	Many chillers include safety features that automatically shut down the unit in case of critical malfunctions.
	Maintenance required	Professional service is typically needed to diagnose and repair chiller issues.
Possible causes	Low refrigerant levels	A common cause of reduced cooling capacity.
	Faulty compressors	Malfunctioning compressors often lead to chiller failures.
	Clogged condensers	Dirty condenser coils can hinder heat transfer and decrease cooling efficiency.
	Water flow problems	Insufficient water flow within the chiller can result in poor cooling performance.
	Electrical issues	Power supply problems or faulty electrical components are common triggers of chiller malfunctions.
Additional troubleshooting practices recommended by the American College of Radiology	Routine inspections and maintenance	Regularly inspect chiller components, including condensers, compressors, and electrical connections to prevent failures.
	Environmental controls	Maintain optimal humidity levels (30-50%) within the MRI suites using dehumidifiers to protect both the chiller and MRI system components.
	Backup power testing	Perform routine tests on uninterruptible power supplies and backup generators to prevent cooling system failures during power outages.

Table 4. Critical Steps and Safety Precautions for MRI Facilities Following a Hurricane

ACTION	DESCRIPTION
Inspect for wind and rain damage	If severe wind and rain have occurred, assume potential quench pipe blockage and arrange inspection by qualified service personnel. Risk of quench pipe blockage is reduced if weather was mild.[1]
Assess quench status	If the MRI has quenched, contact service personnel to reseal the magnet and assess each magnet individually. If no quench occurred, keep untrained personnel away from the discharge point. If power remains off, the risk of quenching increases.[1]
Evaluate building's power status	If power is available, exercise caution regarding electrical hazards, especially in cases of water infiltration. If power is off, quickly restore it and be aware that it may still pose magnetic field hazards.[5]
Personnel safety	Prevent untrained personnel from approaching the quench pipe discharge area. Inform staff of risks related to magnetic fields and quench events during cleanup or repairs.[5]

Table 5. Additional Recovery Tips and Best Practices Following a Hurricane

BEST PRACTICE	DESCRIPTION
Infrastructure modifications	Install preventive measures like flood barriers and sump pumps, and elevate electrical systems. Maintain roofing, windows, drainage systems, and quench pipe weatherheads to minimize water intrusion risks.[16]
Quench simulation drills	Regularly conduct quench simulation drills to train staff in emergency procedures, including safe evacuation and management of quench pipe blockages. Establish detailed emergency shutdown protocols.
Communication with emergency services	Develop an emergency response plan that includes communication protocols with local emergency services and first responders to ensure coordinated recovery efforts.[17]
Risk assessment and insurance	Conduct risk assessments with insurance providers to identify potential financial losses and ensure comprehensive insurance coverage for equipment damage and business interruption costs.[18]

Figure 3. Example of a zone layout in MRI facilities. This layout is adapted from Figure 1 in the Medicine and Healthcare products Regulatory Agency Safety Guidelines for Magnetic Resonance Imaging Equipment in Clinical Use. Note depictions of the MR-controlled access area, MR environment, and projectile area as they relate to the 4-zone model. *Source: Medicines and Healthcare Products Regulatory Agency. Safety guidelines for magnetic resonance imaging equipment in clinical use. Published November 7, 2014. Accessed November 7, 2024. Magnetic resonance imaging equipment in clinical use: safety guidelines - GOV.UK. www.gov.uk/government/publications/safety-guidelines-for-magnetic-resonance-imaging-equipment-in-clinical-use.*

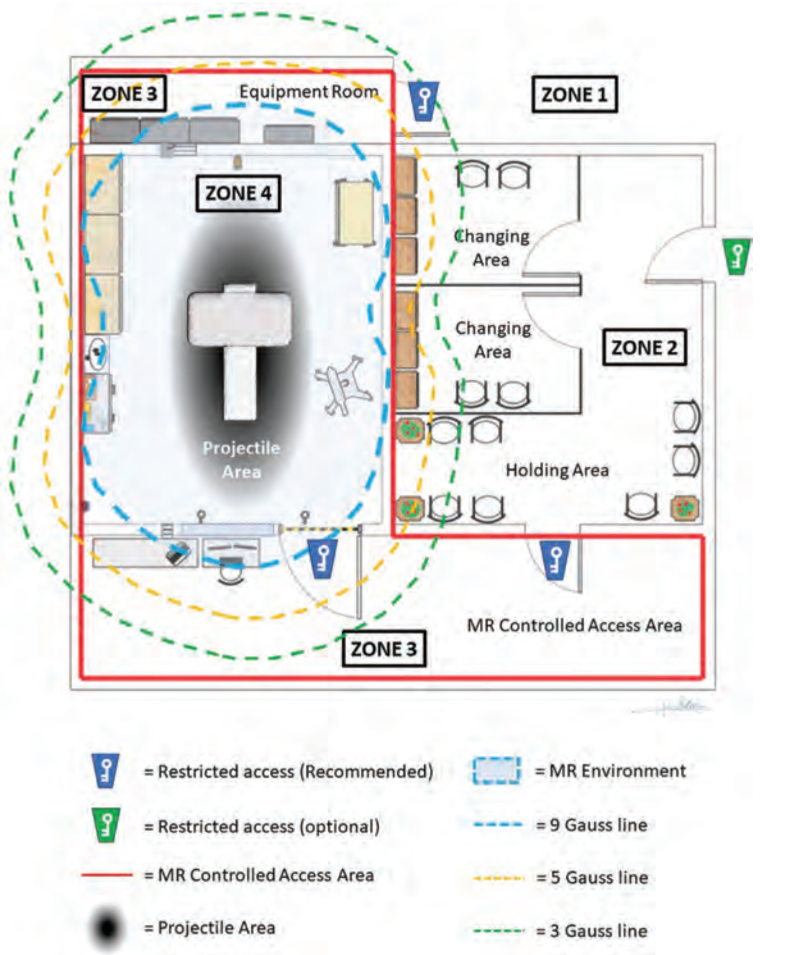


Table 6. Key Considerations for Post-Hurricane Safety and Recovery Protocols

KEY POINTS	RECOMMENDATIONS FOR POST-HURRICANE RESPONSE
Keep MRI service personnel on standby	Ensure MRI service personnel are available after a storm to provide guidance on safe operations and assist with recovery procedures.
Maintain MRI refrigeration system	If water is suspected in the quench pipe, maintain the MRI's refrigeration system to prevent catastrophic quenching.
Avoid metal tools during cleanup	During cleanup, avoid using tools with internal metal components as these can become dangerous projectiles near strong magnetic fields.[6]

quench pipes after a hurricane to ensure their proper function.

Power Outages and MRI Safety

Power outages pose a substantial risk to MRI systems. Unlike most imaging equipment, MRI scanners maintain their magnetic field even during power loss. The cooling system relies on a continuous power supply to prevent helium from warming and evaporating. A key component of this cooling system is the chiller unit, which maintains the superconductive magnet coils in a stable, cold state using liquid helium. When a chiller unit fails, the most immediate consequence is a loss of cooling capacity (Table 3). This interruption causes the air-conditioned space to gradually warm and, depending on the MRI scanner model, error codes may display on the control panel indicating a need for professional service.[6]

To mitigate these risks, health care facilities should equip MRI systems with uninterruptible power supplies (UPSs) and dedicated backup generators. Routine testing of backup systems is crucial, particularly before hurricane season, to ensure their functionality.[7]

Safety Precautions for MRI Facilities After a Hurricane

Following a hurricane, immediate action is essential to assess damage and minimize risks. The steps in Table 4 outline critical safety precautions.

Frank G. Shellock, PhD, has made significant contributions to the field of MRI safety, particularly under extreme conditions such as hurricanes. His work offers valuable insights into magnetic field safety, management of helium quenching, and training for MRI personnel during disaster recovery. Dr Shellock emphasizes the critical importance of adhering to stringent safety protocols in MRI facilities after a hurricane, including inspections for magnetic containment, appropriate system shutdowns, and comprehensive staff training.[8] His guidelines on magnetic field safety and emergency protocols, especially in post-hurricane MRI recovery, contain valuable details and recommendations.[8-12] The MR projectile area, MR environment, and MR-controlled access area and their relationship to the 4 ACR MR safety zones are illustrated in Figure 3.

Key considerations for post-hurricane safety and recovery protocols vary across the MRI vendors for their systems. MRI center personnel should be familiar with the common practices and vendor-specific protocols following major disasters.[13-15] Table 5 shows additional recovery tips and Table 6 summarizes considerations for safety and recovery protocols after a hurricane.

Conclusions

Hurricanes pose significant risks to MRI facilities, including wind, water damage, and power outages. By understanding these threats and implementing best practices — such as environmental control, electrical power management, and proper personnel training — health care facilities can effectively reduce the risk of equipment damage and ensure a safer recovery process. Incorporating preventive infrastructure modifications, communication strategies, and comprehensive risk assessments into emergency preparedness plans will further enhance the resilience of MRI facilities against hurricane impacts.

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Klippel-Feil Syndrome Essentials, Part 1: Embryological Development and Genetic Mechanisms

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Editor's note: This is Part 1 of a 2-part series. Part 2 will be featured in the January/February 2025 edition of Applied Radiology.

Introduction

Klippel-Feil syndrome (KFS) is a rare congenital fusion of the cervical spinal column, sometimes with concurrent involvement of the thoracic and lumbar vertebrae (Figure 1).[1-5] It was first described by Maurice Klippel and Andre Feil in 1912, with the presence of 3 classical clinical signs. The triad, which is seen in some but not all patients, consists of a short neck, limited neck movement, and low posterior hairline.[2]

The lack of robust population screening studies performed for KFS makes it difficult to present an exact incidence for the disease;[2] however, an incidence between 1 in 40,000 to 42,000 is commonly cited.[2,6,7] A 3:2 ratio has been reported in favor of a female predominance,[8] yet these statistics are difficult to determine because KFS often goes undetected and has a variety of clinical presentations.[2] As such, more recent data suggest that the occurrence of KFS in the general population is much higher. A large retrospective review of cervical trauma CT scans found that the prevalence of KFS was 0.0058% (1 in 172) with a slight male predominance,[6] while a prospective multicenter study of patients undergoing symptomatic cervical spinal myelopathy surgery found that 5 of 131 (~3.81%) patients had KFS.[7]

To assist in diagnosing KFS, classification systems based on morphology and etiology have been proposed.[1] The original classification model by Maurice Klippel and Andre Feil divided KFS into 3 subtypes based on fusion extent. Advancements in genetics have greatly improved our understanding of associated molecular changes and form the basis of a new genetic subtype classification presented by Clark et al.[1,2]

In the following review, we place particular emphasis on the embryology, molecular pathology, imaging diagnosis, clinical associations, and treatment of KFS.

Spinal Embryology

Understanding the embryological development of the spine is crucial for diagnosing developmental disorders of the spine, including KFS. The embryogenesis of the vertebrae begins during gastrulation.[9] The paraxial mesoderm forms around the notochord and, as neurulation progresses, matures and segments into somites on both sides of the neural tube.[10] Local signals cause the somites to assume a patterned and segmented appearance. From there, the dorsal somites become the dermomyotome, while the ventral somites differentiate into the sclerotome.[10]

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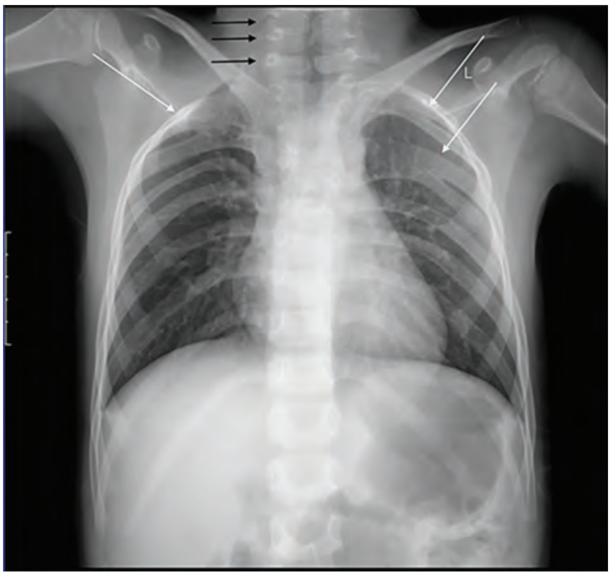
Figure 1. Vertebral anomalies and multilevel rib fusion. The chest radiograph demonstrates multilevel lower cervical and upper thoracic vertebral anomalies (black arrows) and multilevel rib fusion (white arrows) seen in Klippel-Feil syndrome (KFS). It also depicts right-sided elevation of the scapula and an omovertebral bone (inferior to black arrow), correlated with both KFS and Sprengel deformity.



The dermomyotome differentiates into muscle cells and the dermis of the skin, while the sclerotome forms the spine.[11] Owing to sclerotomal rostrocaudal polarity, it undergoes resegmentation. The rostral part of the sclerotome becomes the caudal half of one vertebral body while the caudal part of the same sclerotome becomes the rostral half of subjacent vertebral body.[10] Sections of adjacent sclerotomes then join to form the centrum, which stimulates the surrounding bone to develop into the vertebral body.[11,12] Concurrently, the notochord within the vertebral bodies degenerates, while the notochord remaining in the interspaces gives rise to the nuclei pulposi of the intervertebral discs.[12]

Pathoembryology of Vertebrae in Klippel-Feil Syndrome

KFS results from the improper segmentation of cervical vertebrae during embryogenesis, which leads to the anomalous formation of intervertebral discs.[11,12] As segmentation progresses caudally, vertebral ossification progresses rostrally.[1,13] The bidirectional nature of this process may be significant in the pathogenesis of KFS.

In KFS, the C1 vertebrae fails to develop its centrum and is therefore unable to direct the normal development of the surrounding bone. C1 remains attached to the portion of C2, which forms the odontoid process. This also prevents development of the intervertebral disc between C1 and C2.[1] When the C2 vertebral body and the rostral odontoid process fail to fuse, the resulting abnormality known as *os odontoideum* occurs.[14]

The notochord also influences initial spinal cord development. During weeks 3 and 4 of gestation, the neural folds fuse dorsally to form the neural tube, a spinal cord precursor. The parallel formation of vertebrae and the spinal cord provides a possible explanation for the synchronous presence of congenital spinal cord abnormalities and vertebral bone anomalies.[12]

KFS is also commonly associated with a variety of other abnormalities of anomalous embryogenesis, including the Sprengel deformity of the scapula.[15] At the end of the eighth week of gestation, the scapula descends into its thoracic position. Given that the scapula and midcervical vertebrae develop simultaneously, correlation between KFS and Sprengel deformity is not surprising.[12]

Classification

The variability of KFS has made it challenging to establish specific criteria for diagnosis and treatment. Advancements in genetics and imaging techniques have contributed to the understanding of its etiology and pathophysiology.

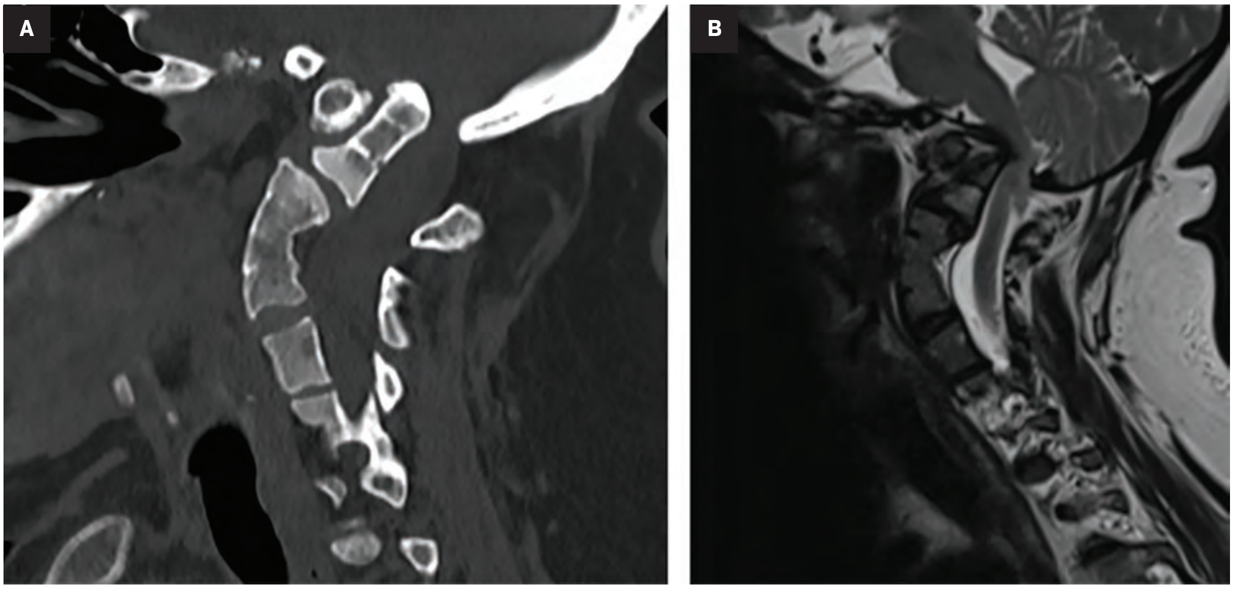
Klippel-Feil Classification System

Maurice Klippel and Andre Feil originally presented a case report featuring a 46-year-old French tailor with extreme discomfort in his neck secondary to fusion of the cervical and

Table 1. Klippel-Feil Classification System

SUBTYPES	VERTEBRAL FUSION
I	Massive fusion (3 or more levels) of cervical and upper thoracic vertebra with synostosis
II	Fusion at 1 or 2 interspaces with other cervical spine anomalies, for example, hemivertebrae, atlanto-occipital fusion
III	Cervical fusion associated with lower thoracic or lumbar fusion

Figure 2. Vertebral anomalies and basilar invagination. Sagittal CT in the bone window (A) and sagittal T2-weighted MRI (B) both demonstrate multilevel segmentation abnormalities, particularly C3-C5, characteristic of Klippel-Feil syndrome. These images also reveal platybasia and basilar invagination. (B) also demonstrates the associated compression of the medulla.



upper thoracic vertebrae.[16] Based on their observations and examination of patients with similar symptoms, they proposed the first classification model for KFS (Table 1). They divided the syndrome into 3 types based on the extent of vertebral fusion.[1,5,8,16] Type 1 is commonly associated with the classic clinical triad of KFS (Figure 2).[17]

Gunderson Modifications to the Klippel-Feil Classification System

The Klippel-Feil classification system of KFS has since been re-evaluated. One study by Gunderson et al[18] included radiographic examination of 121 family members of patients with KFS. They demonstrated definitive cervical fusion in 11 probands and 11 relatives, with an additional 7 relatives having related cervical abnormalities. They reported that C2-3 fusion is inherited in an autosomal-dominant fashion, while C5-6 is inherited autosomal recessively, and variable cervical fusion is inherited dominantly with variable expression and penetrance.

Table 2. Clark Classification of Klippel-Feil Syndrome and a Comparison with Klippel-Feil Classification System

CLASS	VERTEBRAL FUSION	INHERITANCE	ASSOCIATED ANOMALIES	OVERLAP WITH KLIPPEL-FEIL CLASSIFICATION
KF1	C1 fusion	Recessive	Short neck, cardiac, craniofacial, aural, ocular, limb	Types I-III
KF2	C2-3 fusion	Dominant	Craniofacial, aural, tolaryngeal, skeletal, limb	Types I-III
KF3	Isolated fusions	Recessive or reduced penetrance	Craniofacial, facial dysmorphology	Type II
KF4	Cervical vertebrae fusion	Possibly X-linked	Aural, ocular, cardiac, abducens palsy	-

Adapted From Clark et al.[1]

They also reported that types I and III are more severe, presenting with associated neurological and cardiac anomalies.[18]

Clark Classification System

In 1998, Clark proposed a classification system that considers genetic inheritance patterns in addition to morphologic presentation.[1] Clark examined 3 KFS families using radiography and cytogenetic banding and found distinct variations in the timing of the postnatal vertebrae fusion and gross morphology.

Based on these differences, and to address genetic heterogeneity,

Table 3. Molecular Genetics of Klippel-Feil Syndrome Development[19-32]

GENE/PATHWAY	ROLE	MUTATIONS
GDF6	Chromosome 8 involvement, joint and vertebral development, joint fusion	Missense mutations, incomplete penetrance in carpal and tarsal fusions
Notch pathway	Vertebral segmental disorders, somitogenesis, embryonic patterning	Mutations in Notch1, Dll1, Dll3, Hes7, Psen1, Lfng, defects in segment borders and rostrocaudal polarity
PAX genes	Somite segmentation, development	Mutations linked to aniridia and Waardenburg syndrome, PAX1 mutation linked with KFS
HOX complexes	Vertebral development	Hoxd3 mutations cause occipitalization of atlas, Hoxd4 mutations cause supernumerary cervical vertebrae
BAZ1B	Association with Klippel-Feil syndrome	

they proposed a new classification system (Table 2). They defined KF1 as C1 rostral fusion, which is inherited recessively. KF2 entails C2 ± 3 fusion and has dominant inheritance. KF3 usually presents as a single fusion in the ±C3 region, but also includes C5-6 fusion with recessive inheritance. Lastly, KF4 may be an X-linked vertebral fusion with a synchronous presentation of Wildervanck syndrome.[1]

Molecular Genetics of Spinal Development

KFS is characterized by a substantial variation in combinations of genetic and clinical presentation.[1,16,19] Of the genes that have been studied, several show a strong association with familial KFS, as summarized in Table 3.[19-32]

Sporadic Incidence

While recent studies have shown familial inheritance, KFS has historically been understood to exhibit a largely sporadic incidence without a family history of consanguinity, potentially pointing to a significant influence of environmental or multifactorial determinants.[33,34] Tredwell examined similarities between fetal alcohol syndrome (FAS) and KFS,[35] finding common physical abnormalities, such as fused cervical vertebrae, microcephaly, and thoracic cage abnormalities.

While 20% of patients with KFS had 1 cervical level involved and 50% had 4 or more involved levels, patients with FAS had rates of 53% and 5%, respectively. They also noted that alcohol was unlikely to be a contributing factor in KFS but were unable to determine a specific alternative teratogen.

Conclusion

In Part 1 our review, we have detailed the embryological development and genetic basis of KFS. This foundational understanding is essential for enhancing diagnostic accuracy and developing targeted therapeutic interventions for KFS. Part 2 of this review will focus on the clinical manifestations and diagnostic strategies for KFS.

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Key Steps Toward Climate-Resilient Imaging

Maura Brown, MD, MHA

Introduction

Climate change refers to long-term shifts in temperatures and weather patterns, primarily driven by human activity, notably the burning of fossil fuels (coal, oil, and gas).[1] [1]The "triple planetary crisis" of climate change, biodiversity loss, and air pollution is a health crisis with significant direct and indirect adverse impacts on human health and on the resiliency of the health care system.[2] As discussed in the previus issue of *Applied Radiology*, planetary health is the concept that human well-being over the long term depends on clean air, clean water, healthy soil, and the well-being of all living and nonliving systems.[3]

The health care sector is responsible for approximately 5% of total global greenhouse gas (GHG emissions and 8.5% of GHG emissions in the US. As a substantial contributor to the climate crisis and environmental degradation, the health care sector faces increasing pressure to minimize waste and emissions associated with health care delivery while being simultaneously stressed by the impacts of extreme weather events. Medical imaging is estimated to be responsible for up to 1% of global GHG emissions and 20% of total health care emissions.[4] The need for change is urgent. As global health care systems set net-zero targets, radiology is well positioned to lead low-carbon, high-quality, transformative health care initiatives.

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Planetary Health Care in Radiology

While the previous Global Health column introduced radiology's roles in planetary health, here we take a closer look at how the following 3 sustainability principles by MacNeill et al can help position radiology on a promising course toward low-carbon, high-quality care[5] (Figure 1):

1. Reduce the demand for resource-intensive hospital care.
2. Provide the right care to the right patient at the right time.
3. Reduce emissions and the environmental impact of medically necessary health care.

Reduce Demand

The first principle emphasizes that reducing the demand for resource-intensive hospital care is achievable through a strong focus on primary and preventive care.[6] Many health care systems operate in a reactive manner, providing care in response to illness. This is both costly and resource-intensive as hospital care accounts for 36% of all health care emissions.[7]

While hospital care is essential, prioritizing health promotion, addressing social determinants of health, and expanding patient-centered primary care can enhance population health. This approach reduces the burden and severity of disease, while reducing the environmental footprint of the health care sector.

Access to robust screening programs plays a crucial role in early disease detection. For instance, screening mammography is an important tool for reducing the incidence of late-stage breast cancer. Treatment costs, intensity, and duration of care are significantly higher for late-stage breast cancer diagnoses.[8]

Match Supply to Demand

The second principle is to align supply to demand by ensuring the right care is provided at the right time, avoiding over- or underutilization of health

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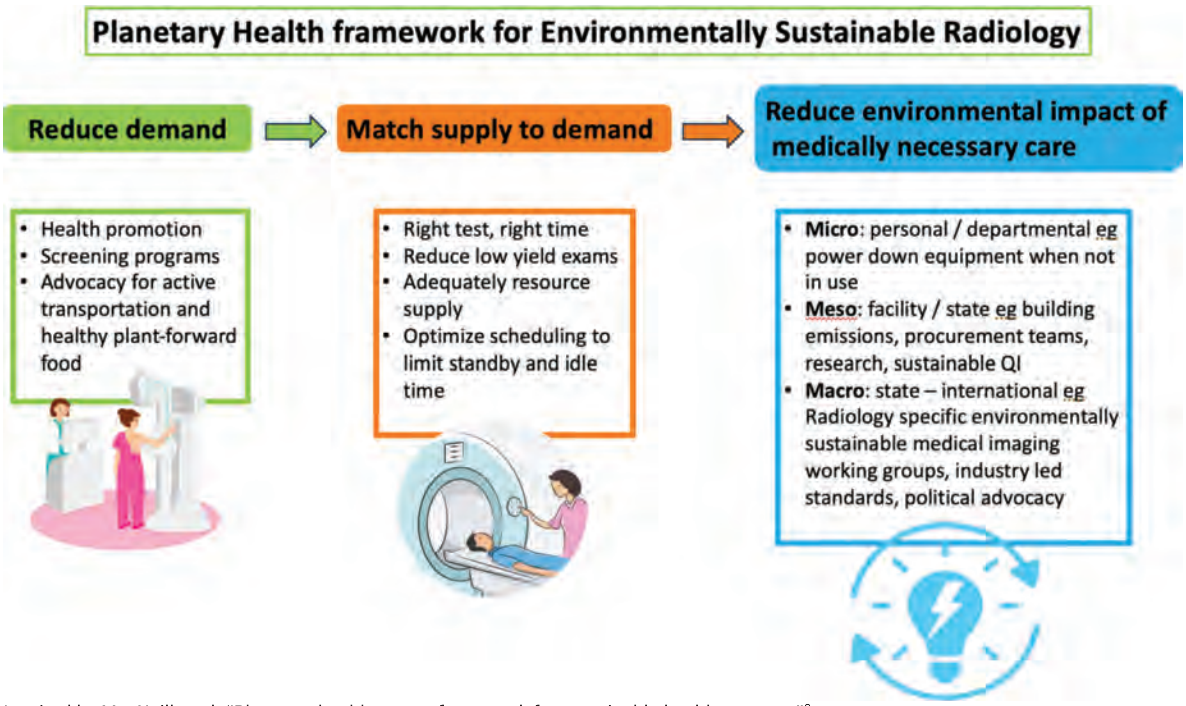
Figure 1. Planetary health care framework for environmentally sustainable radiology.



Inspired by MacNeill et al, "Planetary health care: a framework for sustainable health systems."[6]

care resources.[6] In radiology, this involves reducing low-value imaging exams and enhancing the efficiency of available resources. Avoiding unnecessary investigations and treatment requires broad stakeholder engagement including collaboration with referring clinicians and patient partners. Clinical decision support tools such as Choosing Wisely and those provided by the American College of Radiology offer valuable guidance and patient education resources.[9-11] It is essential to avoid delays in medically necessary imaging tests as this may result in later presentation with more advanced disease and ultimately require more resource-intensive, in-hospital care.

Inefficient use of resources in diagnostic imaging may occur when medical imaging equipment remains idle due to scheduling gaps. Instead, optimizing staff and patient scheduling to reduce scanner idle time between patients can reduce scanner-on time for the same workload per scanner. The transformation to low-carbon, climate-resilient medical imaging requires patient-centered resource allocation prioritizing health and well-being, thereby improving access and health equity.[6]

Reduce the Environmental Impact of Medically Necessary Care

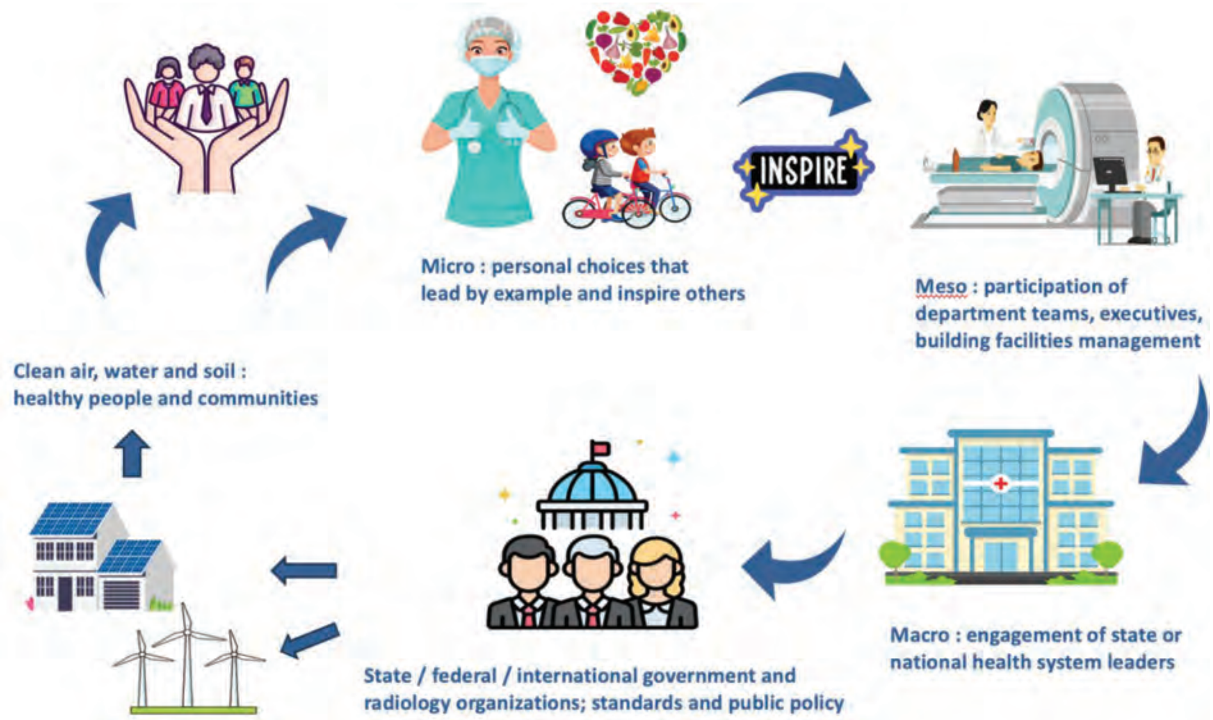
The third principle is to reduce the environmental impact of medically necessary care.

Opportunities for low-carbon, high-quality diagnostic imaging are present at the micro (radiologist), meso (department, facility, or regional), and macro (state, federal, or international) levels (Figure 2).[12]

Micro-level actions include personal choices to reduce one's carbon footprint, such as cycling to work or making plant-forward food choices, as well as leading departmental initiatives to power off medical imaging equipment during non-operational hours.[13]

Meso-level actions at the facility or regional level may include participation on procurement teams to ensure sustainability criteria are included in requests for proposals and advocating for procurement teams to support the principles of the circular economy. Radiologists can support purchase of refurbished medical imaging equipment, which significantly reduces production-phase (raw materials to delivery) emissions.[14] The technical guide to Good Refurbishment Practice provides frameworks for safe use of refurbished medical imaging equipment. Additional opportunities may include advocacy for hybrid or virtual meetings and conferences, or organizational work-from-home policies. Research and sustainable quality improvement are essential. As new artificial intelligence (AI) tools evolve that shorten protocols, saving time and money, and improving patient experience, it will be essential to measure their benefits against the increased energy use and

Figure 2. Opportunities to reduce emissions and waste in medical imaging at the micro (personal), meso (department/facility), and macro (state, federal, or international) levels.



data storage requirements associated with AI.[15]

Macro-level actions may include engagement with state, national, and international organizations to endorse low-carbon, sustainable health care, or joining environmentally sustainable medical imaging working groups in university, state, or national organizations. This may include participating in grassroots efforts such as Radiologists for a Sustainable Future or the task force on climate change and sustainability by the American College of Radiology.[16]

Available national and international resources include the industry-led European Coordination Committee of the Radiological, Electromedical and Health care IT Industry, and governmental agencies such as the US Environmental Protection Agency, which have developed criteria for the environmental sustainability and energy efficiency of medical imaging equipment.[17,18]

Conclusion

Climate change is a continuing threat to local, regional, and international human health and well-being. As an important new tool, climate-resilient imaging can advance patient-centered care and improve access to imaging overall. Fortunately, radiologists are well positioned within the health care system to engage others and to lead the transition to net zero health care.

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Alzheimer Disease Imaging in the Age of Disease-Modifying Therapies

Kerri Reeves

Kerri Reeves is a contributing editor based in Ambler, PA.

Emerging anti-amyloid, disease-modifying therapies (DMTs) promise to revolutionize the management of patients with Alzheimer disease (AD), particularly the estimated 1.5 million who are in the earliest stages of the disease.[1]

Neurologists will need a helping hand from their medical imaging colleagues, who will face dramatically higher demand for their services to maximize the potential of the new therapies.

"This year has brought tremendous advances in the dementia landscape, including expanded opportunities for anti-amyloid targeting therapy to decrease the rate of cognitive decline," says Suzie Bash, MD, a neuroradiologist and medical director at RadNet, and editorial advisory board member. "The earlier AD is diagnosed, the greater the treatment benefit, so early beta-amyloid confirmation is critical."

Alzheimer Disease Treatment: A Quick Synopsis

Historically, AD treatments aimed to manage symptoms by primarily targeting the imbalance of neurotransmitters in the brain such as acetylcholine and glutamate. In most cases, however, they only modestly improve or stabilize symptoms for a limited amount of time.

But new DMTs such as lecanemab (Leqembi) and donanemab-azbt (Kisunla) are emerging as major game changers. These drugs consist of monoclonal antibodies that focus on reducing the underlying pathology of AD, specifically the beta-amyloid plaques, to help slow progression of the disease.

Clinical trials have shown that lecanemab can slow the rate of cognitive decline by about 27% in patients with early AD (mild cognitive impairment secondary to AD or mild AD); in the Clarity-AD sub-study analysis, lecanemab performance was even more impressive, with 76% reduction in cognitive decline and 60% clinical improvement at 18 months in the low-tau group (representing patients at the earliest stage of the disease).[2] Donanemab-azbt slowed cognitive and functional decline by about 35% in patients with early AD. Both lecanemab and donanemab achieved significant plaque clearance.

Although they are not a cure for AD, these treatments reflect a shift from managing symptoms to modifying disease trajectory, offering hope for improving patients' quality of life over a longer period.

The Critical Role of Imaging

Promising as they are, before DMTs can be implemented, the presence of beta-amyloid plaque must be confirmed either through amyloid PET (Figure 1) or, less commonly, through invasive cerebrospinal fluid analysis. Baseline brain MRI scans are also needed to document preexisting microhemorrhages or other findings that may preclude treatment with these drugs.

As a result, the demand for imaging is growing, says Lawrence Tanenbaum, MD, editorial advisory

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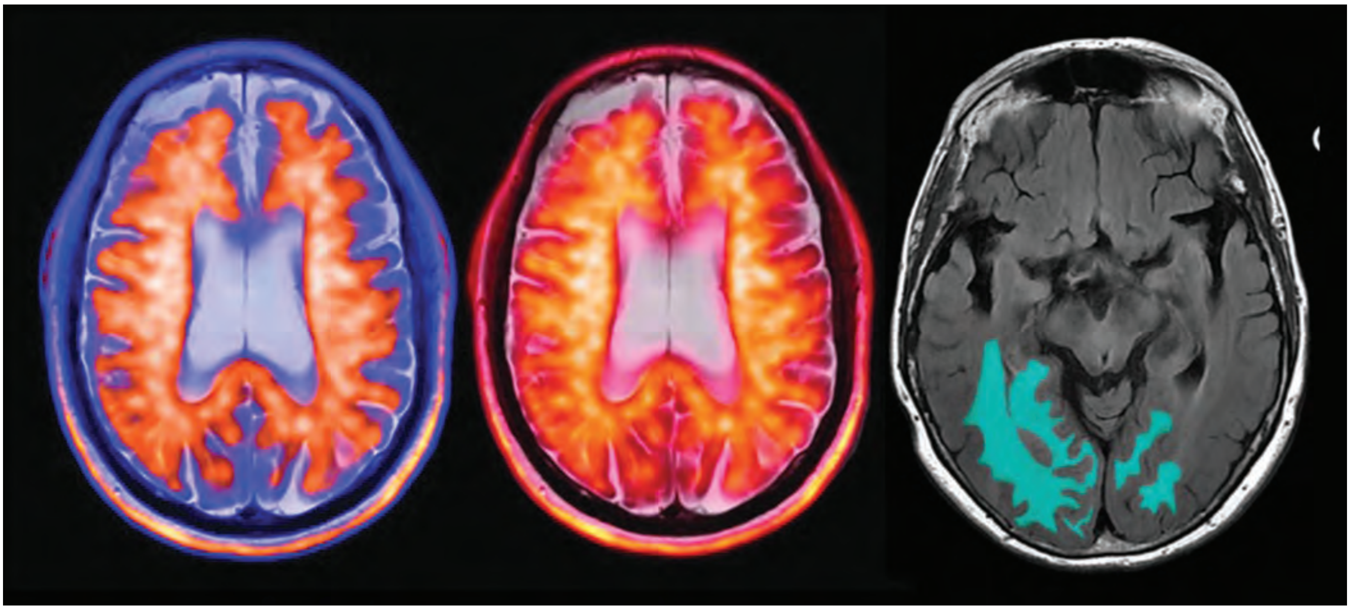
Figure 1. The positive amyloid PET image with color PET-MR fusion on a T2-weighted MR sequence is of a patient with early Alzheimer disease (AD) (left and middle images). A quantitative MRI artificial intelligence-assist tool with automated amyloid-related imaging abnormality segmentation on a FLAIR MR sequence (right image) shows a patient with early AD who was on disease-modifying therapy (image courtesy of Dr Suzie Bash).



board member and former chief technology officer at RadNet.

"There is great impact on the imaging enterprise as a result of this patient population coming in for clinical assessment and consideration for DMT," Dr Tanenbaum says. "Amyloid PET volumes across the U.S. are skyrocketing, going from the occasional case to many cases per day, per facility, because of its key role. Brain MRI is also increasing as it is used for pre-treatment screening baseline analysis and ARIA [amyloid-related imaging abnormality] surveillance."

Debbie Gibbons, senior director of sales, PETNET Solutions, agrees, noting that because of the new treatment options, the Centers for Medicare & Medicaid Services last October began approving reimbursement for amyloid PET—a development certain to accelerate demand for imaging.

"Now that there is a pathway to treat with multiple options and it's covered [via reimbursement], more people can get the answer as to whether they have AD versus a different dementia," says Gibbons.

Imaging Protocols for DMT

Amyloid PET imaging can help determine whether a patient has AD. Brain MRI is required prior to starting treatment, as well as for amyloid-related imaging abnormality (ARIA) surveillance during treatment.

"The non-ionizing nature of MRI and its versatility makes it ideal for longitudinal tracking of brain changes," says Suchandrima Banerjee, senior global director of Neuro MR for GE HealthCare, "as well as identifying preexisting hemorrhage and managing surveillance."

With lecanemab, which requires twice-a-month infusions, MRI scans are performed at baseline and prior to the 5th, 7th, and 14th doses. Donanemab, meanwhile, is administered every 4 weeks and requires MRI scans just before the second, third, fourth, and seventh doses. If ARIA signs or symptoms are present, treating physicians often request additional MRI scans.

As pathophysiological changes that result from mobilization of amyloid in the brain, ARIA causes inflammation in the blood vessel walls. MRI can identify associated edema and/or microhemorrhages or superficial siderosis to help physicians decide whether to pause or discontinue therapy, explains Dr Tanenbaum.

"ARIA complications are generally mild and self-limiting, but serious events have occurred," he says.

Throughput Considerations

As more patients undergo treatment with DMTs, PET and MRI volumes are rising, bringing challenges to imaging enterprises, such as timely distribution of amyloid PET tracers, nationwide shortages of PET technologists, and reimbursement delays, according to Dr Bash.

"Growth has been tremendous for all amyloid imaging agents that have been filed to Medicare," Gibbons observes, while noting that many imaging providers do not have the bandwidth to accommodate the new patients.

"We're seeing bottlenecks on the imaging side," she says, adding that some imaging practices are using mobile units to scan patients 1 or 2 days a week, while some neurology practices want to run their own PET scanner or mobile set-up.

Similar impacts face MRI centers, says Dr Tanenbaum. Patients with AD require at least 4 or 5 scans annually, and potentially as many as 10 in the event of higher-risk genetics or treatment-related complications.

"Imaging enterprises are already gearing up for this significant boost in MR volume," he says.

Technology Support

Dr Tanenbaum and other experts envision greater demand for additional MRI scanners and other tools, including artificial intelligence (AI), to optimize throughput across imaging enterprises.

"Our AI reconstruction technology can help reduce scan times, in some cases, approximately a 70% reduction for brain imaging," says Saurabh Sharma, business development manager, MRI, for Siemens Healthineers. Sharma adds that biometric technology will address biovariability in patients and automation will reduce technologist burden to make imaging centers more efficient.

"We have prioritized AI-driven innovations that not only reduce scan times, but also make scans more consistent and less susceptible to [variation], allowing radiologists to have greater confidence," says Banerjee. GE's deep-learning-based landmark identification technique can ensure a patient's images are acquired with the same angulation at each exam.

Quantitative assessment of brain volume changes with AI-driven segmentation can be helpful in early diagnosis and may be used to augment interpretation over time, Dr Tanenbaum says.

A recent study shows that AI-based assistive software may enhance the diagnostic accuracy of monitoring ARIA for patients receiving amyloid-beta-directed antibody therapies.[3] Quantitative AI-powered tools can improve the quality and consistency of ARIA surveillance through automated quantification and radiographic grading. One AI-assist tool recently received US Food and Drug Administration 510(k) clearance,[4] and an additional tool is approaching the market and aspiring for approval under the CADx designation.[3] In a recent poll by the American Society of Neuroradiology, the majority of respondents indicated that they were interested in utilizing an AI solution designed to enhance ARIA surveillance.[5]

"Brain MRI and quantitative MRI [QMRI] volumes have dramatically increased," says Dr Bash. "Dedicated vendor-neutral QMRI CPT III codes are starting to get reimbursed by Medicare administrative contractors, paving the way for continued increased utilization benefits."

A Collaborative Future

Radiologists must be educated on the unique requirements of AD imaging, particularly the need to identify the MRI findings of ARIA, which can be "subtle and unfamiliar," says Dr Tanenbaum, who notes that the findings of ARIA are frequently missed.[3]

"There's the continued need for all neuroradiologists to receive ARIA training, since accurate and timely interpretation of ARIA surveillance MRIs directly impacts therapeutic decisions," Dr Bash agrees. "The neuroradiology community has stepped up by initiating dedicated ARIA training opportunities, and original equipment manufacturers have worked collaboratively to make dedicated dementia protocols available."

Radiologists will focus on establishing appropriate scanning protocols and optimal workflows, from scheduling through exam execution, as well as providing clear reports to treating physicians. Communication among all, particularly with ordering physicians, is also key to effective diagnosis and treatment, Dr Tanenbaum says.

Dr Bash notes that the medical community is responding well to the challenges and opportunities of DMTs in "remarkably collaborative and creative ways by employing measures to streamline communication, optimize workflow, overcome capacity barriers, train staff, and provide necessary services and resources to allow imaging and treatment access for all eligible patients."

"As patient volumes continue to increase, providers will further adapt their practices to accommodate, because patient-centric care always remains the highest priority," Dr Bash concludes.

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Transforming Radiology: The Role of AI, Automation, and Advanced Cloud Computing

Alejandro N. Bugnone, MD

In the ever-evolving field of radiology, artificial intelligence (AI), machine learning, automation, and advanced cloud computing technologies are paving the way for significant improvements. By addressing various challenges, these technologies are helping to enhance patient care, bolster operational efficiency, and ultimately improve outcomes. Here's a look at the common problems that outpatient imaging centers, radiology practices, and teleradiology groups face, and the innovative solutions that AI, automation, and cloud computing can help provide.

Tackling Inefficiencies and Revenue Loss in Outpatient Imaging Centers

Patient engagement, often inconsistent and unreliable, can be significantly enhanced by AI-driven multichannel communication and automation systems.[1] These systems can help patients receive timely reminders, educational materials, and follow-ups through various channels such as email, a short message service (SMS), and patient portals.

Automation and AI can also help alleviate staff burnout and inefficiencies in indexing, data entry, and order creation in outpatient imaging centers and radiology practices.[2] Technologies such as optical character recognition for document recognition and data entry can streamline processes and reduce manual workload, allowing staff to focus on more critical tasks while likewise decreasing burnout. Additionally, scheduling, a perennial challenge, can be optimized with AI-powered algorithms and self-scheduling options via patient portals. These systems predict optimal times, reduce wait times, and enable patients to schedule appointments at their convenience, with the aim of creating a more efficient and patient-friendly scheduling process. For instance, automated patient confirmation through SMS helps prevent appointment cancellations that disrupt workflow and affect revenue. Patients receive reminders and can confirm or reschedule their appointments, reducing no-shows and ensuring a smoother operational flow.

Staffing challenges, such as verifying insurance and obtaining prior authorizations, can be efficiently managed by automated systems that handle these processes without manual intervention, helping to ensure accuracy and freeing up staff to focus on patient care. Decreased revenue from inefficiencies in collecting patient payments can be mitigated with automated patient estimation tools. These tools send payment links to patients ahead of their appointments or at check-in, facilitating upfront collection and improving revenue flow. Furthermore, paperwork, often time-consuming and error-prone, can be significantly reduced with smart electronic forms available at the patient self-scheduling portals or sent to patients via SMS or links. These options help eliminate the need for physical paperwork, streamlining the administrative process and reducing cost and errors.

Regarding report delivery, sending these documents to referring physicians through multichannel delivery systems fosters timely and efficient delivery via the ordering physician portal, fax, SMS, or HL7 interfaces. Patient access to reports and images, often difficult, can be simplified through patient portals. These portals provide access to health information, which can help patients manage their health effectively. In addition, sharing

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images and reports with other health care providers is facilitated via web links to help provide seamless sharing, improving collaboration and care continuity.

Enhancing Radiology Departments and Teleradiology Practices

All-in-one, cloud-based, zero-footprint picture archiving and communication systems (PACS) that efficiently run on a web browser with embedded AI-powered dictation, help allow for immediate access to radiology workflows, image analysis, and dictation, bypassing the hassles of installation, integration, and compatibility issues.

Automated systems are also designed to provide accurate and timely assignment of studies to radiologists and workflow management based on expertise and availability. Workload balancing rules can further help provide prompt and accurate case handling and improved delivery of the radiology reports.

Radiologist burnout and dictation inefficiencies are significant issues as well. To address these concerns, fully integrated AI dictation systems can help streamline the reporting process with features such as automatic pre-population of measurements or other findings extracted from images, modality-generated speech recognition, or technologist notes. In turn, these systems help expedite dictation through AI interpretation of natural language, automatically generating structured reports, report impressions, comparison with previous reports, and detection of critical findings.

Finally, detection, tracking, and delivering critical findings efficiently are essential for patient safety. AI systems can automatically detect and categorize critical findings, providing robust tracking to help provide prompt and accurate delivery of critical information.[3]

The Transformative Role of AI in Radiology: Automating Key Workflow Processes

AI has the potential to revolutionize radiology by automating critical workflow components, allowing radiologists to focus more on patient care and diagnostic accuracy. AI systems now have the capability to automatically populate essential patient data, such as medical history, demographics, and prior studies, streamlining the process for comparisons and faster diagnosis.

In addition, AI can autofill measurements directly from technologists' worksheets or structured reports, reducing manual input and the potential for errors. Embedded quality checks help ensure the accuracy of data entry, while clinical reference insertions — whether via linked medical websites or contextual footnotes — enhance the report's clarity by offering real-time guidance based on the dictation.

AI goes further by generating comprehensive impressions, recommendations, and comparisons, intelligently focusing on new findings while cross-referencing previous reports. It can identify critical results, flagging them for immediate attention to allow for timely responses to life-threatening conditions.

One of AI's most powerful applications lies in its ability to monitor quality control at multiple levels. For example, it can alert users to laterality errors or when findings for the wrong sex are documented (such as when a male-specific disease is mentioned for a female patient). Similarly, it monitors anatomy dictation, providing immediate alerts when an incorrect body part is referenced.

These innovations are not only designed to boost efficiency, but they also help produce a higher standard of accuracy, leading to better patient outcomes and more streamlined radiological workflows.

Conclusion

AI, automation, and advanced cloud computing technologies are tools that can transform radiology by addressing key challenges and enhancing the patient journey, from order creation to report delivery, while aiming to improve the experience of patients, staff, and radiologists.

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Germinal Matrix Hemorrhage

Avery A. Williams, MS; Richard B. Towbin, MD; Carrie M. Schaefer, MD; Alexander J. Towbin, MD

Case Summary

A neonate was born prematurely at 33+0 weeks' gestation. In utero diagnoses inlcuded interrupted aortic arch and suspected tracheoesophageal fistula. A second neonate was born prematurely at 25+0 weeks' gestation. The infant had a history of spontaneous intestinal perforation.

Imaging Findings

Neonate 1 developed bilateral grade 2 germinal matrix hemorrhage (GMH, Figure 1). Neonate 2 developed periventricular hemorrhagic infarctions in the periventricular white matter with associated mass effect and secondary ventricular enlargement (Figure 2).

Diagnosis

Germinal matrix hemorrhage in 2 premature infants (grade 1 GMH in neonate 1 and grade 4 in neonate 2).[1]

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Discussion

GMH is a common complication of preterm infants younger than 37 weeks, especially neonates less than 32 weeks of gestation, and can lead to significant brain injury. Complications of GMH include IVH, periventricular hemorrhagic infarction (PVHI), posthemorrhagic hydrocephalus, cerebellar hemorrhagic injury, and periventricular leukomalacia.[1] The outcome depends on the severity of the GMH and any associated brain injury.

The germinal matrix is composed of immature vasculature in the periventricular subependymal region of the developing brain[2] and is most prominent around the head of the caudate nucleus. This is the site of mitosis for neuroblasts and glioblasts before their migration out into the brain.[3] The germinal matrix reaches maximum volume at 24 weeks' gestation and slowly decreases until it is fully involuted around 36 weeks' gestation.[4,5] Owing to the immaturity of the vasculature and the limited connective tissue support, the germinal matrix is at risk of rupture if the infant is born before complete involution.[4]

Two common grading scales classify severity of GMH: Papile and Volpe. The Papile grading scale was originally published in the 1970s using CT scans and was later modified to include head US findings.[6] In this grading scale, 4 tiers classify the hemorrhages from minor (grades 1 and 2) to severe (grades 3 and 4). Grade 1 is defined as hemorrhage contained within the germinal matrix; in grade 2, the hemorrhage extends intraventricularly without ventricular dilatation; in grade 3, ventricular dilatation is noted secondary to the ventricular hemorrhage; and in grade 4, intraparenchymal hemorrhage can be seen.[2,6]

The Volpe grading scale also uses tiers of increasing severity of GMH. Volpe grading criteria initially also included grades 1-4; however, grade 4 has been separated into its own distinct category described as PVHI.[5] Again, as with Papile, Volpe categories can be grouped as minor hemorrhages (grades 1 and 2) to severe (grade 3 and PVHI). For Volpe, grade 1 is defined as a GMH with little to no IVH seen; in grade 2, the hemorrhage extends into the ventricle, filling 10% to 50% of ventricular volume at the parasagittal section; in grade 3, the IVH fills more than 50% of the ventricular volume, creating ventricular dilatation; and lastly, previously grade 4, is PVHI.[5,6]

Up to 50% of neonates with GMH present with no signs.[5-8] Typically, lower-grade hemorrhages in asymptomatic patients are identified using cranial US, which supports the need for routine screenings in high-risk neonates. Approximately 80% to 90% of GMH-IVHs occur in the first 72 hours of life, while

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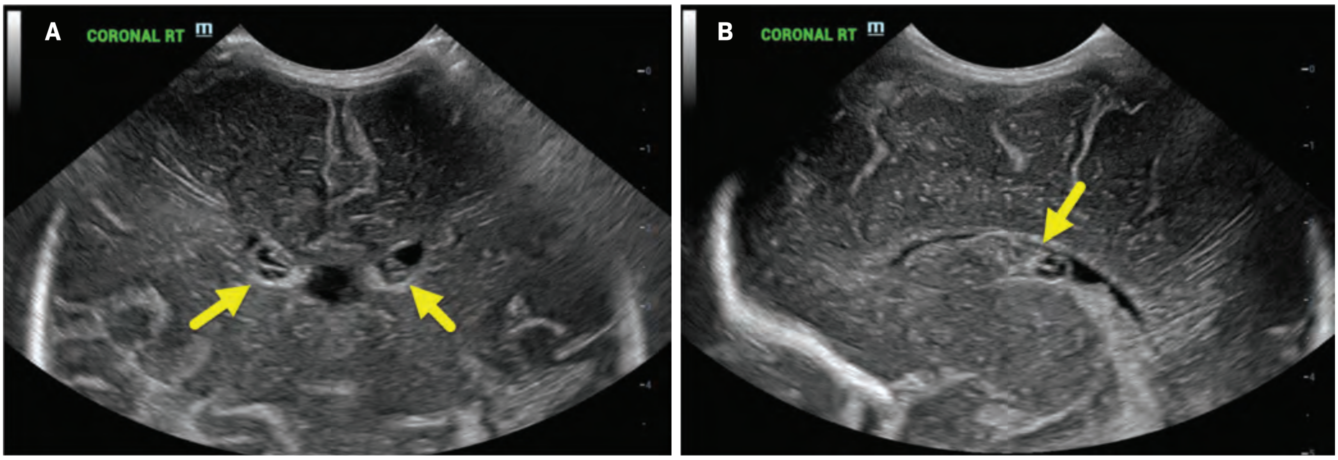
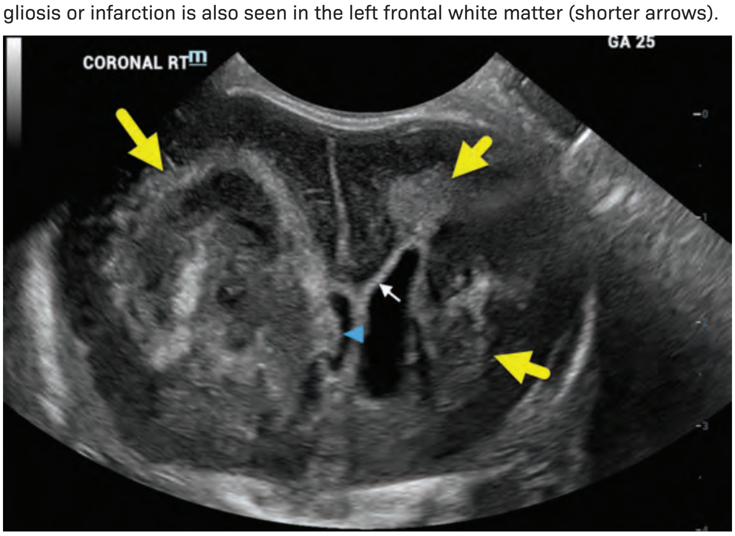
Figure 1. Head US on neonate 1 (born at 33 weeks' gestation) was obtained at 6 days of life. The coronal image (A) shows bilateral, grade 1 germinal matrix hemorrhages that are primarily hypoechoic (arrows). In a parasagittal plane (B), the one of the hypoechoic hemorrhages is seen in the right caudothalamic groove (arrow).



up to 50% of cases occur in the first 24 hours.[9] Neonates with severe GMH or PVHI may present with changes in their level of consciousness, bulging fontanelle, decreased movement, abnormal muscle tone, apnea, bradycardia, acidosis, and anemia.[2,7] The more severe cases may progress to respiratory failure, seizures, fixed pupils, decerebrate posturing, coma, and death. Those who survive higher-grade GMH are more likely to develop long-term complications such as cerebral palsy, cognitive delay, sensory deficits, and epilepsy.[4,6]

The risk of GMH increases as the gestational age at birth decreases.[1,7] The incidence of severe lesions (grade 3 and PVHI) is 10% to 25% in survivors born at 24 weeks. In contrast, severe injuries are only seen in fewer than 5% of those born after 28 weeks' gestation and are rarely observed beyond 32 weeks' gestation.[7] Additionally, the overall incidence of GMH is 20% to 25% of very-low-birth-weight infants.[7,8]

Figure 2. Head US on neonate 2 (born at 25 weeks' gestation) was obtained at 1 day of life. The coronal view demonstrates massive lateral ventricular enlargement with a large, heterogeneously hyperechoic area involving the right frontal white matter and local mass effect (larger arrow). Part of the blood clot is protruding into the right lateral ventricle (arrowhead). Increased frontal white matter hyperechogenicity representing gliosis or infarction is also seen in the left frontal white matter (shorter arrows).



Head US is the initially recommended screening tool owing to its low cost, portability and bedside use, and lack of ionizing radiation. CT and MRI may also be used; however, CT is uncommonly used. When identifying white matter lesions secondary to GMH, MRI is the gold standard.[9] To monitor the appearance or progression of GMH, serial head USs are recommended.[7] Although protocols may differ by institution, for neonates born less than 28 weeks' gestation or 1000 g, it is recommended that scans be performed on days 1, 3, 7, 14, 21, 28, and then every other week until the infant reaches term-equivalent age. If the neonate is born at more than 28 weeks' gestation, serial cranial USs should be performed on days 1, 3, 7, 14, 28, week 6, and at term-equivalent age.[7]

The main protective factor in reducing GMH is preventing preterm delivery. When not possible, prenatal glucocorticoids have proven effective, as has generally minimizing trauma and stimulation of the newborn. Decreasing this stimulation includes minimizing post-delivery transport when possible, minimizing suctioning and handling, and optimizing ventilation.[4] Maintaining stable cerebral blood flow is crucial, which may include supporting steady blood pressures with intravenous fluids and blood products, as needed. Administration of indomethacin to promote closure of the patent ductus arteriosus may be used in the setting of grade 3 or 4 GMH; however, this may lead to oliguria, decreased kidney perfusion, gastrointestinal bleeding, and necrotizing enterocolitis.[6] Additionally, in cases of posthemorrhagic hydrocephalus, intervention may be required, although the efficacy of these interventions is not fully evident.[6]

Conclusion

GMH is a common complication of prematurity that can have profound consequences and significant long-term effects. Up to 50% of neonates with GMH can initially present with no signs, which is why serial head US screening in preterm infants is necessary. Head US is the primary modality for diagnosis and grading, and can help predict outcomes for the infant to plan potential interventions.

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Multinodular and Vacuolating Neuronal Tumor

Parag Vijaysingh Patil, MD, DNB

Case Summary

An adult presented with a 1-year history of intermittent nonfocal headaches. Physical examination was unremarkable. Routine laboratory investigations were normal.

Imaging Findings

MRI of the brain (Figure 1) revealed a cluster of multiple nodules in the gray-white matter junction and subcortical white matter in the right superior frontal gyrus. The nodules were hyperintense to gray matter on T2-weighted and FLAIR images, and iso-to-hypointense on T1-weighted images. No restriction was seen on diffusion-weighted images. No blooming was noted on susceptibility-weighted images. The nodules were coalescent, especially toward the gray-white matter junction. Neither mass effect nor contrast enhancement was noted. Based on MRI findings, a diagnosis of multinodular and vacuolating neuronal tumor (MVNT) was presumed, and the patient was offered symptomatic treatment. Follow-up MRI at 18 months later revealed a stable lesion.

Diagnosis

Multinodular and vacuolating neuronal tumor.

Differential diagnosis includes dysembryoplastic neuroepithelial tumor (DNET) and enlarged perivascular spaces.

Discussion

Multinodular and vacuolating neuronal tumor was first described by Huse et al[1] in 2013 as a non-neurocytic, seizure-associated tumor in 10 patients. The entity was subsequently included as a new cytoarchitectural pattern in the 2016 update of the World Health Organization (WHO) classification of tumors of the central nervous system.[2] This pattern was thought to be malformative and related to ganglion cell tumors.

In the 2021 classification,[3] this entity is included in WHO grade 1 glioneuronal and neuronal tumors. Genetic alterations in the mitogen-activated protein kinase pathway are found in these tumors.

Multinodular and vacuolating neuronal tumors are usually found in adults, with a mean age at presentation of 38-44 years.[4-7] Most cases present with headache (usually nonfocal) or seizure, while some can be asymptomatic.[4-7] The parietal lobe is most often involved, followed by the frontal lobe.

Histopathologically, these lesions show neuroepithelial cells arranged as nodules with conspicuous vacuolation. These dysplastic cells are in the deep portion of the cortex, gray-white matter junction, and subcortical white matter, and are oriented perpendicular to the cortical surface.[4]

Most lesions exhibit typical MRI features. They appear iso-to-hypointense on T1-weighted images and as clusters of well-circumscribed T2-weighted and FLAIR hyperintense nodules in subcortical white matter with or without involvement of overlying cortex.

The nodules demonstrate a "bubbly" appearance on T2-weighted images.[4,5] Abnormal white matter signal can surround these bubble-like lesions,[4,5] and mass effect is typically absent. The lesions demonstrate neither restricted diffusion nor paramagnetic susceptibility effect. Nunes et al[4] noted one case demonstrating subtle focal contrast enhancement; otherwise, contrast enhancement has not been reported in MVNTs. The imaging features are in keeping with its nonaggressive nature.[6]

Typical MRI patterns help differentiate MVNTs from DNET, which are cortical lesions, often wedge-shaped with FLAIR hyperintense components and a multicystic appearance. On CT, the lesions may either not be seen or appear as hypodense.[5]

Biopsy is not required in MVNTs as characteristic MRI features are

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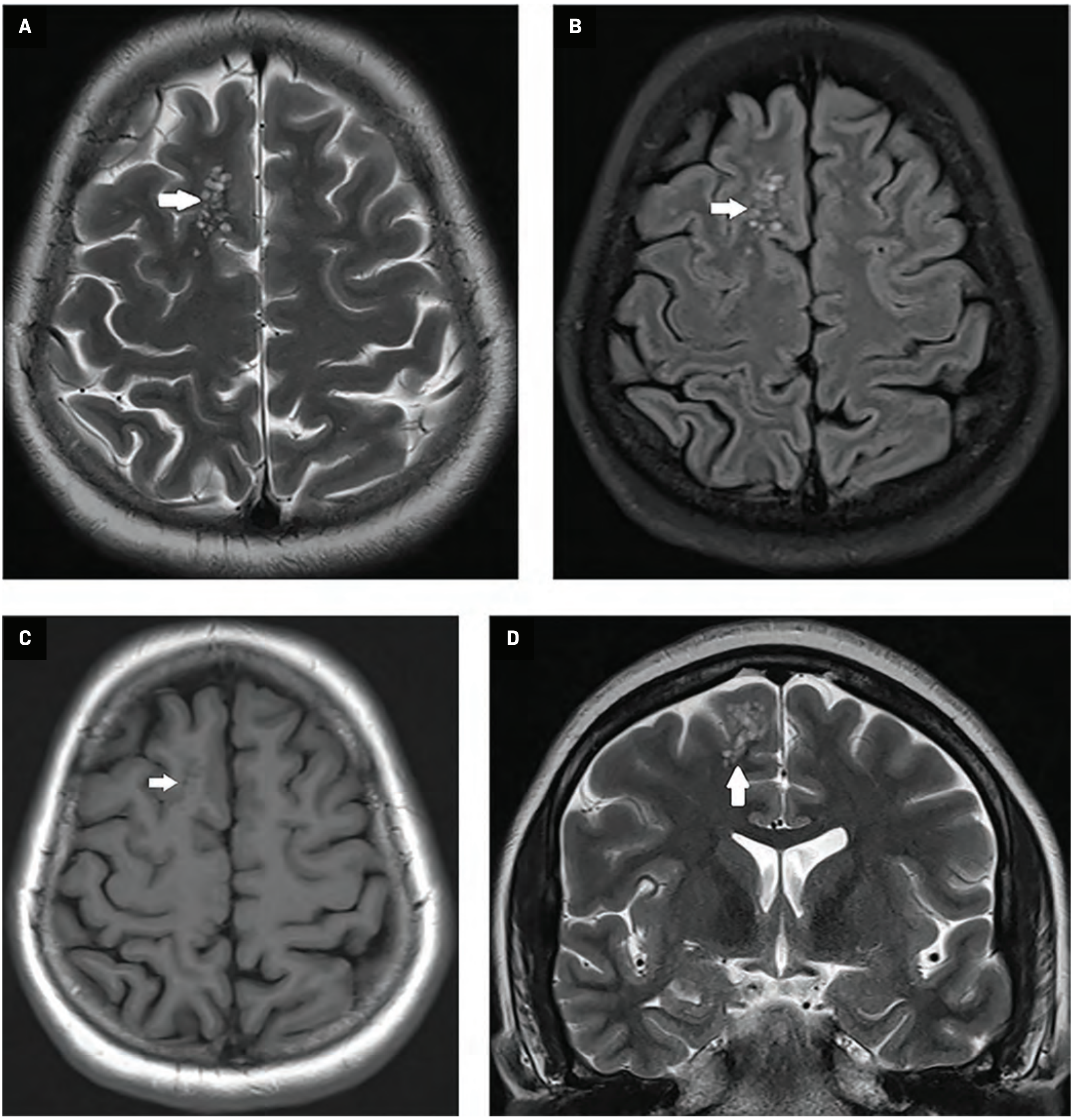
Figure 1. Axial T2-weighted (A) and FLAIR (B) MR images demonstrate a cluster of hyperintense nodules (bubbles) in the subcortical white matter in the right frontal lobe (white arrows). The nodules appear iso-to-hypointense (white arrow) on T1-weighted image (C). Conglomeration of nodules toward the gray-white matter junction (white arrow) is seen on coronal T2-weighted image (D). Note the absence of mass effect.



sufficient for diagnosis. Most lesions are managed conservatively as they are indolent. Surgical resection may be required where seizures or symptoms are attributable to the lesion.[4] Surgical resection is generally curative.[1]

Conclusion

Multinodular and vacuolating neuronal tumors can usually be diagnosed via characteristic MRI features. Familiarity with these lesions may prevent unnecessary biopsy.

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RADIOLOGICAL CASE

Mucocutaneous Leishmaniasis

Jose David Brito-Araujo, MD; Felipe Aluja-Jaramillo, MD, MHPE

Case Summary

A middle-aged adult presented with a 10-year history of HIV infection and 3-year history of upper respiratory tract leishmaniasis.

Nasolaryngoscopy demonstrated nasal septal perforation and thick yellow discharge along the larynx, base of the tongue, epiglottis, and pyriform sinuses, preventing evaluation of the glottis and subglottis.

Subsequent tongue biopsy demonstrated leishmania amastigotes. The patient improved with liposomal amphotericin treatment.

Imaging Findings

A barium swallow study demonstrated a lesion originating in the larynx and indenting the anterior hypopharyngeal contrast column, with pharyngeal contrast accumulation and delayed emptying, associated with aspiration of contrast into the airway and irregular oropharyngeal mucosa (Figure 1).

Contrast-enhanced neck CT demonstrated nonspecific thickening of the oropharyngeal, laryngeal, and tracheal mucosa. Left level V and right paratracheal lymphadenopathy were present (Figure 2).

Diagnosis

Mucocutaneous leishmaniasis.

The differential diagnosis is tracheal tuberculosis.

Discussion

Mucocutaneous leishmaniasis is a parasitic infectious disease manifest by destruction of the oronasopharyngeal mucosa and cartilage, occasionally involving the larynx.[1] It is characterized by destructive lesions of the nasal septum, lips, and palate and as a result of the strong immunopathological response by the host to the parasite.[1] The disease is potentially life-threatening and may be highly disfiguring; laryngeal involvement leads to aspiration pneumonia.[1] Orofacial disease may include nasal obstruction, dysphagia, mucosal bleeding, and/or hoarseness.[2] The disease is thought to be more common in immunocompromised individuals.[1]

Leishmania protozoans belong to the family *Trypanosomatidae* and can be present in 2 forms: flagellated, or promastigote, found primarily in the digestive tract of the insect vector; and aflagellated, or amastigote, found in the tissues of vertebrate hosts.[3]

Observation of amastigotes in a specimen establishes the diagnosis, but combination tissue sampling approaches are recommended.[1]

It is estimated that approximately 0.7 to 1 million cases of cutaneous leishmaniasis occur worldwide each year.[1] In the Americas, multiple Leishmania species are circulating in the same geographical areas, multiple reservoir hosts, and multiple vectors. The pathogenic species in the Americas belongs to *Leishmania mexicana* or *Leishmania braziliensis*. About 1% to 10% of patients infected with a strain of the subgenus Viannia develop mucocutaneous leishmaniasis.[1]

Most cutaneous leishmaniasis lesions heal spontaneously in 2 to 18 months.[1] Systemic treatment is usually reserved for immunosuppressed patients, extensive lesions, refractory disease, and mucocutaneous manifestations.[1]

Manifestations of the disease on CT are variable and nonspecific. In a study by Camargo et al, the most common findings were paranasal sinus thickening, nasal perforation, nasal conchal changes, maxillary sinus retention cysts, and collapse of the nasal pyramid.[4]

In another case report from Italy, a senior immunocompetent patient presented with a diffuse annular increase in tracheal wall thickness and mild homogeneous enhancement.[5] In a multicenter case series of 7 patients with oral leishmaniasis, the most common site affected was the tongue and the most common clinical presentation was an exophytic lesion.[6]

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Figure 1. Barium swallow (A) study shows a lesion originating in the larynx and indenting the anterior margin of the hypopharynx (arrow). Pharyngeal contrast (B) retention with delayed evacuation and aspiration of contrast into the airway (arrow), Note the irregular oropharyngeal mucosa.

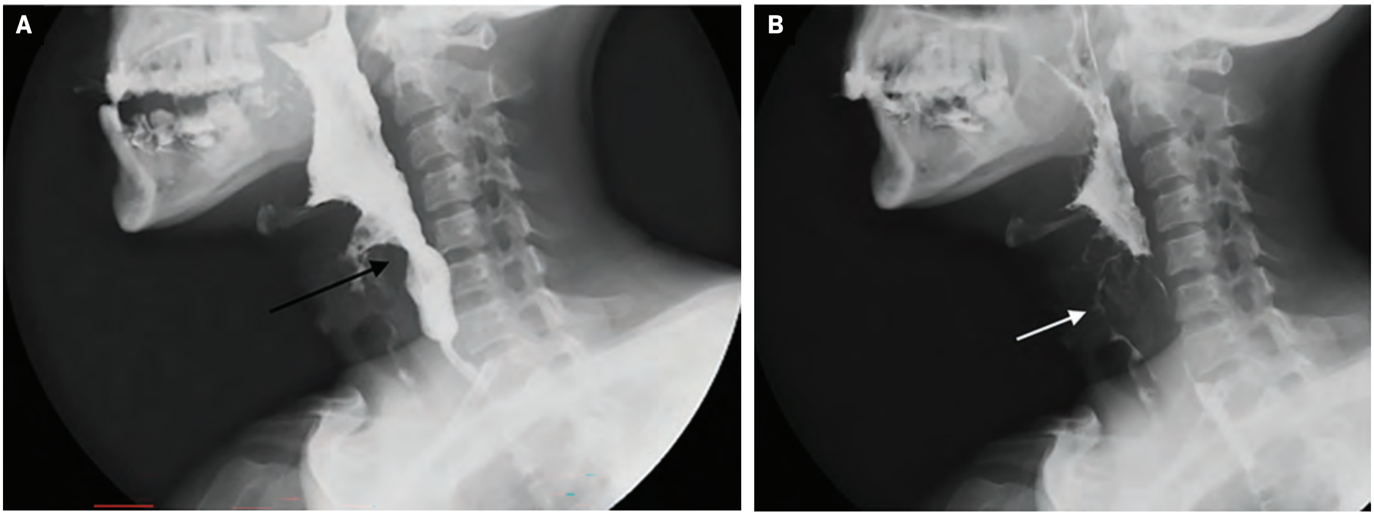
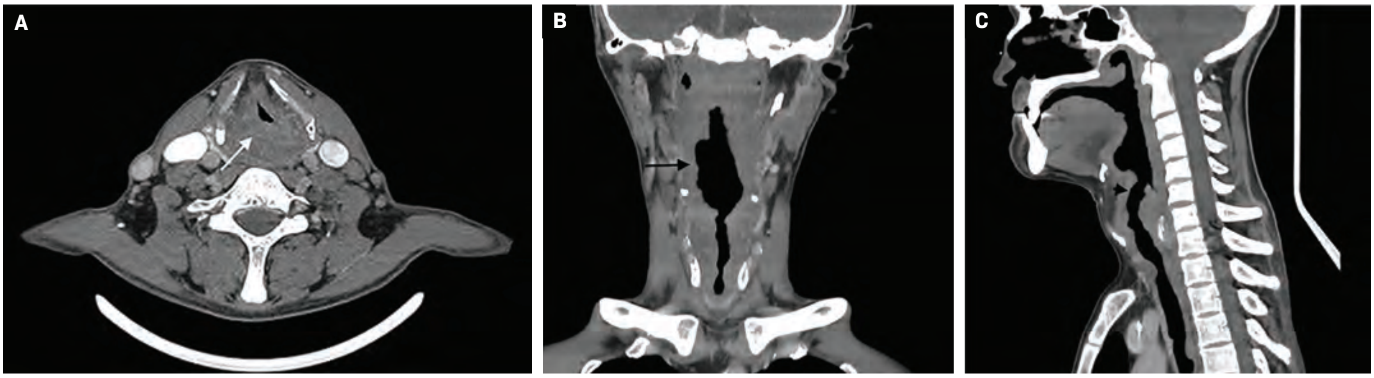


Figure 2. Contrast-enhanced CT of the neck in the axial plane (A) shows soft-tissue mass at the hypopharyngeal level (white arrow). The mass extends asymmetrically, to the proximal aspect of the larynx, causing a decrease in its lumen of more than 60%. Coronal (B) and sagittal (C) plane reformations show nodularity of the mucosa of the oropharynx, larynx, and trachea (black arrow).



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Acute Adrenal Hemorrhage

Jihoon Lim, BS; Sharon Lee, DO; Alberto Mansilla, MD

Case Summary

An elderly patient with a history of atherosclerosis, atrial fibrillation, hyperlipidemia, and pacemaker placement presented to the emergency department with right-sided pain below the breast. This pain began 1 hour earlier while the patient was operating construction equipment. The patient also complained of chest pain, nausea, and vomiting. The patient's blood pressure was 67/52 and heart rate was 67 bpm, with otherwise normal vital signs. Physical examination was significant for a diaphoretic patient with severe right upper quadrant tenderness. CT abdomen and pelvis with intravenous contrast was performed, followed by abdominal aortography.

Imaging Findings

Abdominal and pelvis CT (Figure 1) demonstrated a hemorrhagic soft-tissue mass in the right upper quadrant of the abdomen arising from the adrenal gland. A rapidly perfused pseudoaneurysm within the mass was also evident. A subsequent abdominal aortogram (Figure 2) revealed contrast extravasation from the middle adrenal artery correlating to the area on the CT. Subsequent coil embolization of the right middle adrenal artery proximal to the pseudoaneurysm was performed.

Diagnosis

Acute adrenal hemorrhage with pseudoaneurysm

Discussion

Adrenal hemorrhage is an uncommon condition characterized by bleeding into the suprarenal glands. It is estimated to be present in approximately 0.14% to 1.8% of postmortem examinations.[1] The actual incidence may be higher as the condition's vague symptoms, comorbid conditions, and variable laboratory findings can make diagnosis challenging. Adrenal hemorrhage is more common in men, likely owing to increased frequency of underlying causes. It can affect neonates and middle-aged individuals.[2] While hemorrhage severity depends on the etiology, the condition overall carries an approximate 15% mortality rate. In patients with Waterhouse-Friderichsen syndrome (often caused by meningococcal sepsis), the mortality rate is 55% to 60%.[3]

The etiology of adrenal hemorrhage is diverse and can be classified as primary (idiopathic) or secondary to abdominal trauma, infection, septicemia, anticoagulant use, pregnancy, acute stress, neonatal stress, surgery, neoplasm, or autoimmune disease.

Imaging remains the most important diagnostic tool; CT is the modality of choice. Acute hemorrhage typically appears as a nonenhancing mass with low or mixed attenuation observed in one or both adrenal glands. Normal adrenal contrast enhancement may still be present, especially in a peripheral distribution. Sometimes, a train-track appearance is noted, where peripheral enhancement is preserved with central low attenuation.[4] Other features that may be seen include periadrenal fat stranding and active extravasation with bleeding into the retroperitoneal area.

MRI can display high T1 signal intensity or rapidly changing signal and is the most accurate modality for differentiating acute from chronic hematomas, as well as demonstrating the presence of an underlying tumor. Bleeding may continue until the adrenal gland expands beyond its original shape, forming a round or oval hematoma from a few centimeters to more than 10 cm.[4,5] In infants, ultrasound is the modality of choice due to their small body size and relatively large adrenal size.[2] Blood loss may be associated with abnormal lab parameters such as leukocytosis and anemia. Hyponatremia, hyperkalemia, and hypoglycemia may also be detected if there is adrenal insufficiency.[5]

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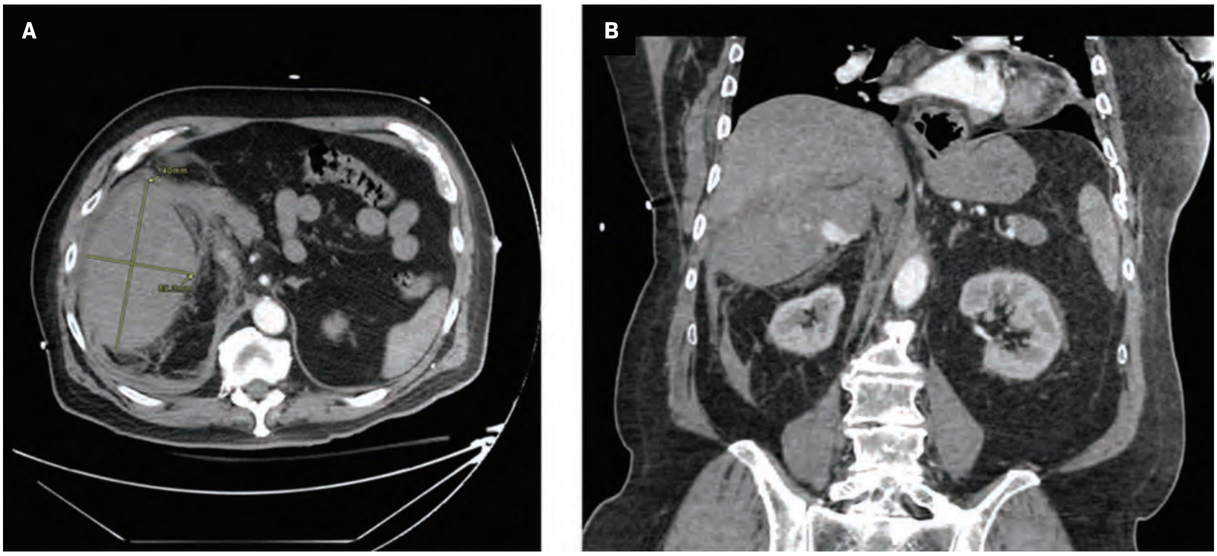
Figure 1. Axial CT (A) through the upper abdomen shows a 14 × 9-cm hemorrhagic soft-tissue mass in the right upper quadrant of the abdomen. Coronal view (B) shows avid contrast in the pseudoaneurysm within the hemorrhage.



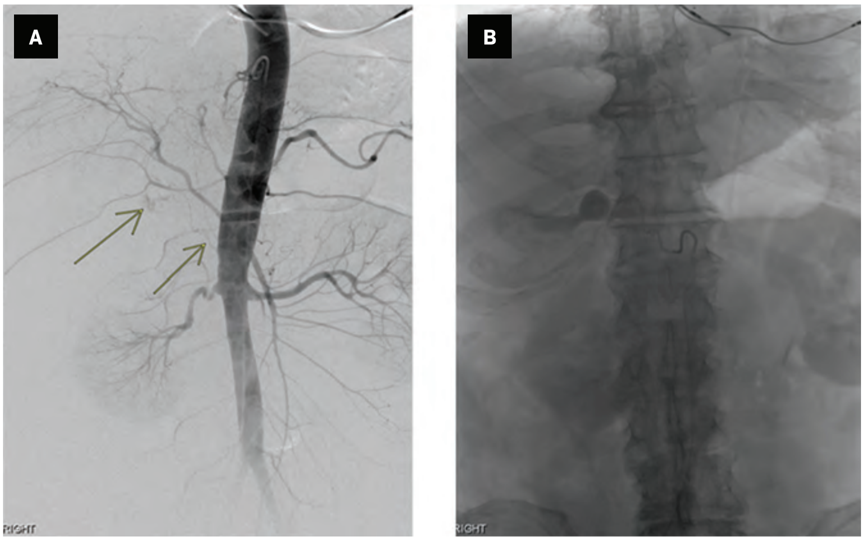


Figure 2. Anteroposterior view (A) from an abdominal aortogram depicts the middle adrenal artery originating from the aorta with active extravasation (arrows), correlating to the area of pseudoaneurysm on CT. Selective right middle adrenal arteriography (B) of the pseudoaneurysm shows the middle adrenal artery with contrast extravasation from the pseudoaneurysm.

Treatment depends on the extent of the hemorrhage and the degree of hormonal insufficiency. Patients with hemodynamic instability refractory to transfusion or rapidly expanding hemorrhagic masses are candidates for intervention.[6] Angiography and embolization of the bleeding lesion, as in this case. In the acute setting, excluding an adrenal tumor can be challenging and delayed imaging is usually required.[7]

Conclusion

Adrenal hemorrhage is a rare condition that can lead to acute adrenal insufficiency and may be fatal. The condition can present in a myriad of clinical contexts and with vague clinical features. Therefore, a high index of clinical suspicion and early diagnostic workup are required for prompt management and improved prognosis.

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"Use, do not abuse ... neither abstinence nor excess ever renders man happy."

—Voltaire

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Psst, Hey, Buddy, Want a Job?

C. Douglas Phillips, MD

I have been pretty busy of late; we've discussed this previously. Worklists are growing exponentially. We are **all** busy. I talked to an old friend the other day who told me they were fully staffed and caught up with their reading. After recovering from my momentary lapse of consciousness (simple cardioversion and IV fluids), I asked how that was possible. Did they learn to clone staff? Someone willed a few billion dollars to their department? Nope; the answer was a typical yin-yang. A radiology department across town had collapsed, most of the rads liked being in that city, and the jobs at my friend's place were easy to grab (reasonable salary, simple cross-town drive, learn a new PACS, get a new parking place and ID badge), so they had suddenly filled every position. It might be fleeting, but for now, life for them is good.

Radiology staffing is at an odd (but somehow desirable) place. We are in demand. We can pretty much pick our job and our destination. Remember when you were approaching the end of your training and wondering what to do with yourself? I've spoken to first-year residents lately who already know what they are going to do when they finish. Already committed. Looking at houses. This has altered recruiting dynamics, for sure. The best time to recruit new staff it seems now is right out of kindergarten. I think those youngsters are impressionable and you might be able to convince them that pictures on a computer screen and a cool chair are at least as neat as the fire truck. I may be on to something here; I am not clear about the legal issues with this, but a kid who has interest in images and plays well with others might be a great early grab. I do understand the lag time here is considerable, but hey, guaranteed staffing?

This, too, shall pass. I have no reason to suspect that we are always going to be a hot commodity. AI, turf wars, and "radiology providers" are out there, lurking. It doesn't seem like constancy is anything we should ever count on. I do distinctly remember a while back when jobs were few and far between; residents were suddenly very interested in fellowships; fellows were interested in second fellowships. Abstinence or excess. Voltaire was a smart dude.

Wait. I just thought of a constant. The amount you make for reading that study? It is ALWAYS going to be less next year. And less again the year after. "Volume, volume, volume," as David Letterman always said. More monitors, faster PACS, shorter reports, longer worklists.

Keep doing that good work. Talk to those kindergarten kids. Mahalo.