JAOCR
Official Journal of the American Osteopathic College of Radiology

Aims and Scope
The Journal of the American Osteopathic College of Radiology (JAOCR) is designed to provide practical up-to-date reviews of critical topics in radiology for practicing radiologists and radiology trainees. Each quarterly issue covers a particular radiology subspecialty and is composed of high-quality review articles and case reports that highlight differential diagnoses and important teaching points.

Access to Articles
All articles published in the JAOCR are open access online. Subscriptions to the journal are not required to view or download articles. Reprints are not available.

Copyrights
Materials published in the JAOCR are protected by copyright. No part of this publication may be reproduced without written permission from the AOCR.

Guide for Authors
Submissions for the JAOCR are by invitation only. If you were invited to submit an article and have questions regarding the content or format, please contact the appropriate Guest Editor for that particular issue. Although contributions are invited, they are subject to peer review and final acceptance.

Editor-in-Chief
Daniel J. Wale, D.O., Ann Arbor, MI

Editor Emeritus
William T. O’Brien, Sr., D.O., Cincinnati, OH

Editorial Board
Abdominal/Body Radiology
Sharon A. Kreuer, D.O., Monroeville, PA

Breast Radiology
Matthew Tommack, D.O., Eugene, OR
Michelle C. Walters, D.O., Dallas, TX

Chest and Cardiac Radiology
Mark Guelfguat, D.O., Bronx, NY
Douglas Johnson, D.O., Charlotte, NC

Musculoskeletal Radiology
Christopher Cerniglia, D.O., M.Eng., Westborough, MA
Matthew Tommack, D.O., Eugene, OR
Donald von Borstel, D.O., Tulsa, OK

Neuroradiology
Alysha Vartevan, D.O., Scottsdale, AZ

Nuclear Medicine
Timothy McKnight, D.O., Farmington Hills, MI
Daniel J. Wale, D.O., Ann Arbor, MI

Pediatric Radiology
Brooke S. Lampl, D.O., Cleveland, OH
Emily Janitz, D.O., Akron, OH

Vascular and Interventional Radiology
Aaron T. Rucks, D.O., M.S., Erie, PA
TRAUMA IMAGING


From the Editor

In this Issue ............................................................................................................................................. 4

Review Articles

CT Imaging and Interventional Radiology in Solid Organ Injury .......................................................... 5
Jonathan A. Friedman, M.D., Thomas J.D. Wilczynski, D.O., Neelabh Maheshwari, M.D., Brian A. Bianco, D.O.

Imaging of Traumatic Intracranial Hemorrhage ..................................................................................... 13

Differential-Based Case Reviews

Globe Injury After Head Trauma ............................................................................................................. 21

Complex Facial Fracture ......................................................................................................................... 24

Diaphragmatic Hernia in a Patient with Chest Trauma ............................................................................. 26
Nadia Lushina, M.D., Niveditha Thangaraj, M.D., Christopher Brown, M.D., Nancy Mohsen, M.D.

JAOCR at the Viewbox

Lunate Dislocation ...................................................................................................................................... 29

Knife Injury to the Cervical Spine ........................................................................................................... 30
David Kopylov, M.D., Valeria Potigailo, M.D., Brendan McCracken, M.D.

Acute Compression Fracture with Unipedicular Approach Kyphoplasty ................................................. 31
In this Issue

Professor of Radiology, Department of Radiologic Sciences, Hahnemann University Hospital / Drexel University College of Medicine, Philadelphia, PA

I am delighted to present this trauma issue of JAOCR to our radiology community. Hahnemann University Hospital, like so many others across the United States, is an urban, level 1 trauma center. As such, we must be prepared for any trauma that enters our doorways. Unfortunately, violent and nonviolent bodily injuries involve everyday people both across the United States and worldwide on a daily basis. No question, we see the full spectrum of trauma here at Hahnemann, from simple fall injuries to motor vehicle collision/pedestrian or other transportation-related injuries, weather-related injuries or mass casualties. We certainly see our share of people injured due to knife and gunshot violence. Regardless of injury types, we must be knowledgeable and ready to triage, image and stabilize all such patients to save lives.

Needless to say, most patients who enter our facility undergo plain film and/or CT imaging for immediate assessments and treatment planning. As such, we as radiologists must be familiar with a wide variety of potential injuries to provide immediate information and guidance to our colleagues. And certainly, a subset of patients must also be treated urgently by our interventional radiology team to rapidly stabilize vascular and solid organ injuries.

In this issue, we highlight a number of traumatic conditions, both common and uncommon. We emphasize the role of interventional radiology in trauma patients, illustrating evaluations and treatments involving both solid organ and vascular injuries, all in an effort to immediately stabilize our injured populace. Our intracranial hemorrhage article illustrates the different compartments of intracranial hemorrhages and injuries that can occur. Our Case Reports and Viewbox cases illustrate additional traumatic chest, body, musculoskeletal, and spine injuries as well as treatments.

Our authors consist of medical students, house staff and faculty from our practice at Hahnemann University Hospital/Drexel University College of Medicine. We hope that these articles help to both educate our readership and aid in diagnosing and treating future injured patients, all to improve long-term patient care, survival and outcomes. I would like to offer a most sincere shout-out to both Daniel Wale, D.O., our current editor-in-chief for JAOCR and of course, William O’Brien, D.O., editor-in-chief emeritus, for their patience and guidance in helping us design and publish this issue. They are both true visionaries in our osteopathic radiology community.

Further, I would personally like to dedicate this trauma issue to the memories of needless traumatic gunshot injuries and deaths that have so deeply affected the core of our great nation. I would specifically like to mention the following shootings and massacres that have occurred in the United States since the events of 9/11: Virginia Tech, Sandy Hook Elementary, West Nickel Mines School, Orlando Pulse Nightclub and, separately, the Orlando shooting of Christina Grimmie, the Las Vegas Strip massacre, and the Marjory Stoneman Douglas High School, Emanuel AME Church, Congressional baseball, and Pittsburgh synagogue shootings. May our country continue to heal from these senseless shootings and find a meaningful compromise for gun safety.

“The Second Amendment is very important, but we have to have common-sense gun safety.”

Deborah K. Ross
According to the World Health Organization, traumatic injuries kill more than 5 million people worldwide every year, accounting for 9% of the world’s annual death toll. Approximately one-quarter of the 5 million deaths are the result of suicide (16%) and homicide (10%) with another one-quarter due to road traffic injuries. Even larger is the burden of the tens of millions of nonlethal injuries resulting in hospitalizations, emergency department visits, and outpatient encounters.

The liver, spleen, and kidneys are among the most commonly injured solid organs and are particularly vulnerable to blunt or penetrating trauma, including iatrogenic injury, leading to arterial laceration, parenchymal or peritoneal hemorrhage, subcapsular hematoma, pseudoaneurysm, or arteriovenous fistula formation. In the emergent setting, focused assessment with sonography in trauma (FAST) is preferred over diagnostic peritoneal lavage as the screening tool for detecting intra-abdominal bleeding. Exploratory laparotomy is indicated in hemodynamically unstable patients, while hemodynamically stable patients typically undergo initial diagnostic imaging.

CT with intravenous contrast is the modality of choice because of its speed, availability, diagnostic accuracy, noninvasive nature, and ability to detect additional abdominal injuries that may require surgery. Subsequent angiography may be necessary to evaluate for and potentially treat vascular injury. Typical angiographic findings in blunt abdominal trauma include contrast extravasation, subcapsular or parenchymal hematomas, and/or arterial occlusion, while penetrating trauma is usually more focal, demonstrating extravasation, pseudoaneurysms, and arteriovenous fistulas.

**Splenic Injury**

The spleen is among the most commonly injured organs in blunt abdominal trauma, accounting for up to 49% of all visceral injuries, with rates of injury in penetrating abdominal trauma less than that of other organs such as the liver and bowel. A substantial portion of penetrating splenic injury arises...
In splenic trauma, the ability to preserve functional spleen is dwarfed by the need for prompt diagnosis and management. CT with intravenous contrast is the diagnostic gold standard for the assessment of hemodynamically stable patients with suspected splenic injury.

Imaging features of blunt splenic injury include laceration, nonperfusion, subcapsular or parenchymal hematoma, active hemorrhage, hemoperitoneum, sentinel clot and major vascular injury. Laceration on CT appears as an irregular linear hypodensity. Subcapsular hematomas present as elliptical collections of hypodense blood between the capsule and enhanced parenchyma causing indentation or flattening of the organ contour. Ongoing bleeding appears as a punctate hyperdensity reflecting active contrast extravasation. Hemoperitoneum from splenic injury results in blood pooling in the left paracolic gutter and/or pelvis, possibly passing into the right upper quadrant. A sentinel clot is typically a higher attenuation focus of clotted blood indicating an adjacent anatomic area of injury causing hemorrhage.

The most commonly used injury classification is the American Association for the Surgery of Trauma (AAST) grading scale, which demarcates 5 grades of splenic injury (grades I-V) with a higher number indicating worse from inadvertent intraoperative injury.
severity. The 2018 version reflects advancements of newer CT scanners and the relative success of nonoperative management (NOM). Notably is the addition of vascular injury on imaging as an indicator of high-grade injury. This includes pseudoaneurysm and arteriovenous fistula.

The utility of repeat CT imaging in the acute inpatient setting is controversial. In a study by Davis et al with 524 patients, NOM failure was most likely to occur within the first 72 hours following traumatic injury. Their protocol of repeat CT with intravenous contrast at 48-72 hours after initial imaging found that 74% of splenic pseudoaneurysms were not present on initial imaging. Subsequent angioembolization lowered the overall failure rate for NOM to 6%. However, a study by Haan et al with 472 patients and a similar protocol found only 2 cases of delayed vascular injury on follow-up CT imaging, of which both were preceded by a drop in hematocrit. The average AAST injury grade in this study was 1.8, which may indicate that repeat CT imaging may not be necessary for low-grade splenic injuries.

The use of angiographic embolization varies by institution with no accepted practice guidelines or consensus on patient selection criteria. Some institutions favor aggressive endovascular management by performing embolization as the predominant therapy for grade III-V injuries and reserve surgery for patients with hemodynamic instability or peritonitis. Other institutions favor medical or surgical management as the first-line therapy and reserve endovascular techniques for active extravasation. Factors associated with NOM failure include age (> 55), high-grade injury (> grade III), active extravasation, large-volume hemoperitoneum, concomitant solid organ injury and vascular abnormalities.

Most authors support the recommendation of splenic surgery or angiembolization for grade IV and higher injuries, meaning any injury with evidence of a vascular component such as active hemorrhage or pseudoaneurysm. According to Martin et al, “literature supports practice paradigms with aggressive IR intervention in grades IV-V injuries and injuries with evidence of active arterial injury.” The Eastern Association for the Surgery of Trauma (EAST) recommends consideration of angiography for patients with AAST splenic injury grade III or higher, the presence of a contrast blush, moderate hemoperitoneum, or evidence of ongoing splenic bleeding.

Three common interventional radiology techniques used in the trauma setting include: transarterial embolization...
TAE), balloon occlusion, and stent grafts. Splenic arterial embolization (SAE) has proven successful with 3 general methods: proximal embolization, distal embolization, or a combination of both (Figure 4). Proximal embolization entails deploying coils or plugs approximately 2 cm distal to the dorsal pancreatic artery and is typically performed for diffuse splenic trauma (eg, shattered spleens and/or multiple areas of contrast extravasation) allowing collateral circulation from pancreatic, gastroduodenal, and gastric branches to maintain distal parenchymal perfusion (Figure 5). Distal embolization may be performed with Gelfoam (Pfizer Inc., New York, NY) distributed by flow or superselective embolization of a focal defect or single injured vessel with coils or particles, with the latter technique requiring increased time and technical skill (Figure 6).

Splenic artery embolization carries a high success rate. A systematic review and meta-analysis performed by Rong et al of 876 patients with 2 study sets demonstrated a primary success rate of SAE to be 90% with an overall incidence of severe complications at 20% and cases requiring further surgical intervention even fewer at 6%. Although success rates were higher for proximal embolization, no statistically significant differences between success rates and embolization location were identified, although the study suggested a reduced risk of adverse events with proximal SAE compared to distal and combination embolization. The use of coils is associated with higher success rates and a lower risk of developing life-threatening complications compared to Gelfoam.

Hepatic Injury

Similar to splenic trauma, hemodynamic status and contrast CT imaging are the cornerstones in directing management by assessing the liver parenchyma as well as evaluating for other signs of injury including hemoperitoneum, pneumoperitoneum, hepatic

FIGURE 7. Contrast-enhanced axial CT (A) illustrates a grade IV injury involving most of the right hepatic lobe (yellow arrows). No active extravasation is seen. Selective arteriography (B) shows abnormal perfusion characteristics of the right hepatic lobe corresponding to CT findings (yellow arrows). Right hepatic artery embolization was performed using Gelfoam.

FIGURE 8. Contrast-enhanced axial CT (A) illustrates a grade IV injury of the liver with multiple small ovoid areas of enhancement within an intraparenchymal hematoma consistent with traumatic pseudoaneurysm (arrows). Selective arteriography (B) shows small pseudoaneurysms (red arrow) within a branch of the segment 4 hepatic artery. Proximal superselective segment 4 hepatic artery embolization was performed using Gelfoam.

FIGURE 9. Contrast-enhanced axial CT (A) illustrates a grade IV hepatic injury with pseudoaneurysms (red arrows). Selective arteriography (B) shows hepatic artery pseudoaneurysms corresponding to CT findings (red arrows). Segment VIII and IVa hepatic artery embolization was performed using Gelfoam.
venous injury, periportal low attenuation, sentinel clot(s), additional organ injuries, and active bleeding (Figure 7, 8). Active hemorrhage is identified by a contrast blush, seen as a hyperattenuating focus on arterial or venous phase imaging and has a similar Hounsfield unit with nearby arterial vasculature. If there is uncertainty as to whether the hyperattenuating focus represents active hemorrhage, delayed images may show worsening contrast extravasation or morphologic changes.

Follow-up CT with intravenous contrast may play a role in management. Re-evaluation using CT is recommended when there is a persistent systemic inflammatory response syndrome (SIRS), increasing or persistent abdominal pain, jaundice, or a decrease in hemoglobin. Even in asymptomatic patients repeat CT may be useful. A study involving 259 patients with blunt liver trauma reimaged patients 4-5 days following traumatic injury and found that 3% of asymptomatic patients had developed a pseudoaneurysm. In a study by Yoon et al, the authors explained that CT is useful in the assessment of delayed complications of blunt liver trauma, including hemorrhage, hepatic or perihepatic abscess; post-traumatic pseudoaneurysm; hemobilia; and biliary complications such as biloma and bile peritonitis. Their study also confirmed that follow-up CT is needed for patients with high-grade liver injuries to mitigate future issues requiring intervention.

The AAST scale for liver injury demarcates 5 grades of splenic injury (grades I-V) with a higher number indicating worse severity. Hepatic trauma can be divided into 3 management classifications: NOM, TAE, and surgery. CT can accurately characterize the severity of hepatic injury and has reduced the number of patients undergoing surgery. According to EAST, “NOM of blunt hepatic injuries currently is the treatment modality of choice in hemodynamically stable patients, irrespective of the grade of injury or patient age,” but only in an environment that can support monitoring for acute decompensation and provide emergent interventional or surgical management. Patients with hemodynamic instability or peritonitis still require surgical intervention as the first-line option; however, TAE may be considered before surgery if the patient transiently responds to resuscitative efforts and imaging shows identifiable arterial bleeding.

Even in high-grade injuries, the use of NOM in hemodynamically stable patients remains successful. A meta-analysis conducted by Melloul et al utilized data from 4743 patients with grade III-V hepatic injury. NOM in hemodynamically stable patients with grade III-V hepatic injury showed a success rate of 82% to 100%, an overall 90-day mortality rate of 0% to 8%, and liver-related mortality of 0% to 4%. Similarly, TAE showed a success rate of 81% to 100%, with biliary leak cited as the most common complication (5.9%). TAE should be utilized after the identification of contrast blush on CT imaging. A retrospective series with 351 blunt hepatic trauma patients identified high-grade injury (grade III-V) and CT angiographic contrast blush as prognostic indicators for the likelihood of NOM failure. In those patients, TAE in NOM can reduce the likelihood of failure and the need for surgery (Figure 9).

TAE can also be used if clinical signs indicate continuing or worsening hemorrhage in the setting of known hepatic injury. Celiac and mesenteric arteriography localizes previously seen or clinically suspected active hemorrhage. Even without a blush on CT, angiography and TAE may be performed if a patient shows clinical signs of hemorrhage. One study identified all liver trauma patients with severe liver injuries from their institution over 10 years, regardless of hemodynamic status, and all underwent TAE with a success rate of 90%. Hepatic TAE is successful and generally well tolerated by patients with procedure-related death being
extremely rare. A systematic review by Virdis et al evaluated 3855 patients and found success rates of TAE to range between 80% to 97%. All-cause mortality following TAE is < 10%; the risk of liver-related mortality is rated at 6%; and the most significant possible risks of TAE include bile leak at 5.7%, hepatic necrosis, and abscess.

Biliary and hepatic venous injury can also be repaired by interventional radiology. Hepatic venous trauma is almost always handled surgically, but there are cases of endovascular repair in which 2 endovascular covered stents were successfully placed to bridge flow from the hepatic vein to the IVC following traumatic injury. Biliary injuries may develop into a complicating biloma, abscess, stricture, or arteriobiliary fistula, all of which are amenable to repair through percutaneous transhepatic cholangiography, stenting, embolization, and/or drain placement.

Renal Injury

The kidneys are the third most common abdominal organ to be traumatically injured. Blunt trauma is a major mechanism, but there is an increasing number of iatrogenic causes (both during interventional and intraoperative procedures) due to the rise in interventional procedures such as renal artery angioplasty, stenting, percutaneous biopsy, nephrostomy, and nephro-ureterolithotomy. Traumatic renal injuries are identified on CT imaging with intravenous contrast and graded using the AAST classification system (grade I-V, with grade V the most severe), which takes into account specific complications such as renal vascular thrombosis, segmental renal artery or vein injury, and damage to the collecting system. There has been a trend toward the NOM of renal traumatic injury that is often institution-dependent and based on the injury grade and the patient’s clinical status. Grade I-III parenchymal or vascular injuries are always initially managed conservatively with observation. NOM is also increasingly becoming the standard of care for grades IV and V parenchymal injury provided the patient is hemodynamically stable and there is no evidence of active contrast extravasation or urine leakage. A multicenter study of 206 patients with grade IV or V blunt renal injury demonstrated safe NOM of hemodynamically stable patients, with a nonoperative failure rate of 7.8%. Furthermore, NOM decreases ICU stay, lowers transfusion requirements, and yields fewer complications. Current guidelines from the American Urological Society recommend observation in hemodynamically stable patients and intervention in hemodynamically unstable patients.

Surgical treatment is always indicated for hemodynamically unstable patients, in grade V vascular injury (avulsion of the renal artery, vein, or collecting system), and in expanding retroperitoneal hematomas discovered during exploratory laparotomy for other abdominal injury. Ureteral stenting is the treatment of choice in lacerations involving the collecting system and ureteropelvic junction laceration.

Several retrospective studies have shown early follow-up CT imaging does not detect or prevent any urologic...
complications. For example, a study at the University of Tennessee of 207 patients who sustained grade I-III renal injury found that follow-up CT with intravenous contrast in renal cortical and excretory phases did not detect or prevent any urologic complications. Another study at Cork University Hospital of 102 patients with grade I-V renal injury demonstrated all complications of renal trauma were symptomatic. The European Association of Urology Guidelines refer to repeat imaging for renal injuries and recommend repeat CT imaging in grades I-IV only if the patient demonstrates clinical deterioration such as fever, flank pain, and decreasing hemoglobin.

Interventional radiology is slowly taking a larger role in the treatment of blunt renal trauma with selective renal artery embolization (Figure 13); however, there is little data reported in the literature. A study of 20 patients who underwent renal artery embolization for blunt trauma and gross hematuria demonstrated successful cessation of bleeding in all patients. A study of 52 patients with grade III or IV renal laceration showed peri-renal hematoma size and contrast extravasation to be predictors for renal artery embolization. Renal pseudoaneurysm is another possible vascular injury from blunt renal trauma. Again, data are limited to case series, including a study of 5 patients treated successfully with embolization.

Conclusion
Identification and management of abdominal organ injury is rapidly evolving. Injuries once identified and treated operatively are now diagnosed by CT and predominantly treated nonoperatively and/or via interventional techniques in hemodynamically stable patients. These more conservative and minimally invasive techniques are driven by the goals of increased patient safety and reduced morbidity and mortality. Given these trends, there will likely be an increasing role for interventional radiology in patient management and treatment as part of a multidisciplinary clinical team.
REFERENCES


Imaging of Traumatic Intracranial Hemorrhage

Christian Koegel, M.D., Raluca McCallum, M.D., Mark Greenhill, B.S., Diana López García, M.D., Ajay Kohli, M.D., Mea Mallon, M.D., * Robert Koenigsberg, D.O., Khuram S. Kazmi, M.D.

Departments of Radiology, Hahnemann University Hospital and St. Christopher Children’s Hospital, * Drexel University, Philadelphia, PA

Intracranial hemorrhage (ICH) is a common entity encountered in clinical emergency medicine. Imaging is the cornerstone in the diagnosis of traumatic ICH. In a large study of patients with a head injury and a decreased Glasgow Coma Scale (GCS), 46% of patients demonstrated intracranial hemorrhage. Of these, 30% were subdural hematomas (SDH), 22% were epidural hematomas (EDH), 22% were intraparenchymal hematomas (IPH), and 14% were subarachnoid hemorrhages (SAH). Current literature reports up to 72% incidence of diffuse axonal injury (DAI) in moderate to severe head injury. Timely and accurate diagnoses of ICH is key to successful patient management given the emergent nature of ICH.

CT is the initial modality of choice due to its accuracy, short study time, low cost, and robustness against artifacts. MR is also used given its increased sensitivity in detecting DAI, as well as subacute and chronic hemorrhage. Although lumbar punctures were previously more common in the diagnostic workup of SAH, there has been a paradigm shift away from performing this procedure, as nonenhanced CT (NECT) has been shown to be highly accurate in detecting SAH when performed within 6 hours of symptom onset.

This article will review the key vascular anatomy associated with ICH followed by a strategy for imaging evaluation and reporting. This is followed by a detailed review of the different types of traumatic ICH with an emphasis on imaging findings.

Meningeal Vascular Anatomy

The three meningeal layers (dura, arachnoid and pia mater) are perfused by the anterior, middle and posterior meningeal arteries. The anterior and posterior meningeal arteries originate from the anterior ethmoidal and ascending pharyngeal arteries, respectively, and perfuse dura of the anterior and posterior cranial fossa. The middle meningeal artery, a maxillary artery branch, enters the skull at the foramen spinosum. Trauma to the parietotemporal skull can lacerate these vessels resulting in hemorrhage.

Imaging Evaluation and Reporting

Given the critical nature of ICH, the interpreting physician must take necessary steps to prevent overlooking ICH on imaging. Although axial imaging is the standard for evaluating head CT, using additional reconstructions should be considered in routine clinical practice. ICH detection rate increases when axial and coronal images are viewed. Among missed acute intracranial hemorrhages, SDH accounts for 39%, followed by SAH with 33%. A customized checklist approach (Table 1) may be beneficial to the radiologist, particularly to ensure classic areas for subtle findings are evaluated. CT windows should also be considered when interpreting studies as subtle findings may be obscured due to windowing. In addition to standard CT head windows, the authors’ institution has found a width...
of 99 Hounsfield units, level of 93 HU images can make hemorrhage optically more conspicuous (Figure 2).

Additionally, it is important to have a general understanding of imaging findings and information that referring physicians need to guide management. In particular, findings that help determine a need for surgical decompression/evacuation should be reported, although the decision for surgery is based on clinical information in addition to imaging. When encountering ICH on CT or MR several items should be routinely reported including midline shift (particularly if > 5 mm), hematoma thickness/volume, mass effect, effacement of ventricles and/or cisterns, evidence of herniation, evidence of hydrocephalus, and coexistent injuries such as skull fractures and intracranial foreign bodies.\(^\text{11}\)

### Imaging Features of Intracranial Hemorrhage

#### Diffuse Axonal Injury

Unequal rotational and acceleration-deceleration forces cause the brain to pivot around the brainstem, causing stretching and shearing of axons and resulting in the clinical diagnosis of DAI. Shear forces peak at the grey-white matter interface, due to its peripheral location and differences in tissue density; hence, 67% of DAI lesions are in this region.\(^\text{12}\) In more severe cases, additional sites of injury occur in the brainstem. Within hours, axonal deformation damages its cytoskeleton, resulting in arrest of axoplasmic flow on a microscopic level, followed by axonal swelling and subsequently axon rupture.\(^\text{13}\) Mechanisms of injury vary, but include high-impact falls and motor vehicle accidents.

Foci of axonal edema with or without hemorrhage are imaging findings of DAI and these foci may vary in size

---

**Table 1: Example Checklist for a Systematic Search Pattern for Subtle Intracranial Hemorrhage**

**Axial images**
- Check the posterior tip of occipital horns and interpeduncular cistern
- Check for asymmetric increased gyrus to inner table distance
- Check for asymmetrically effaced Sylvian fissure

**Coronal images**
- Evaluate for falcine/tentorial hemorrhage

**Axial and coronal images**
- Check for extra-axial fluid collection of the anterior and middle cranial fossa floor
- Check for vertex bleed
- Check for skull fracture and, if present, evaluate for contralateral injury

---

**FIGURE 1.** Two patients with subtle intracranial hemorrhage. (A) Axial unenhanced CT demonstrates high-density blood (arrow) in the posterior horn of the left lateral ventricle following a fall. (B) Axial unenhanced CT in a second patient with small amount of high-density blood in the interpeduncular fossa (arrow).

**FIGURE 2.** Subdural hematoma detection aided by windowing. (A) Axial unenhanced CT demonstrates a small subdural hematoma (yellow arrow) along posterior falx and superior sagittal sinus. Asymmetric effacement of left Sylvian fissure and extra-axial fluid collection (blue arrow) lateral to anterior left temporal lobe appears similar in brightness to overlying skull. Its biconvex shape mimics an epidural hematoma. (B) Axial unenhanced CT with a more optimally windowed (width of 99 HU, level of 93 HU) improves conspicuity and reveals subdural blood, indicated by wavy crescent-shaped contour of the collection (red arrow).
Despite DAI being present in up to 72% of patients with moderate to severe head injuries, 50% to 80% of initial CT and MRI studies are negative. MRI is more sensitive than CT in detecting lesions (Figure 4). Gradient echo (GRE) and susceptibility-weighted imaging (SWI) are the most sensitive sequence, revealing the highest number of lesions at the highest number of locations compared to other MR sequences. The classic triad seen in DAI is diffuse damage to axons, a focal lesion in the corpus callosum, and a focal lesion in the dorsolateral quadrant of the rostral brainstem adjacent to the superior cerebellar peduncles. DAI lesions detected within 4 weeks after injury improve functional status and long-term outcome due to changes in patient management. Therefore, MRI is recommended within 4 weeks of traumatic brain injury (TBI).

Intraparenchymal Hemorrhage
Traumatic acute IPH, commonly referred to as a cerebral contusion, usually occurs after a significant direct head injury. It commonly involves brain parenchyma adjacent to bony protuberance/dural fold. CT displays a patchy, irregular, hyperdense area of acute hemorrhage on an edematous background (Figure 5). Hemorrhages may be multiple and bilateral. Cerebral edema may be relatively mild acutely; however, progression of cerebral edema is common and can result in midline shift and herniation syndromes. IPH can be complicated by continued enlargement and/or re-hemorrhage. Likewise, new lesions can be detected on follow-up imaging. MRI is useful to identify traumatic intraparenchymal damage without blood products, termed nonhemorrhagic cerebral contusion (Figure 6). Sequelae after resolution are common and include gliosis, encephalomalacia and associated volume loss.

Subdural Hematoma
Acute SDH is characterized by an extra-axial crescent-shaped hemorrhage in a potential space between the dura and arachnoid. A direct blow to the head is not necessary to incur an SDH. Instead, an SDH may be related to acceleration-deceleration forces secondary to the impact from a fall or shaking of an infant. Regardless of the mechanism, shear forces on the bridging veins or cortical arteries can lead to injury and rupture. Elderly are predisposed to develop SDH due to cortical atrophy. Additionally, anticoagulants and antiplatelet agents (Figure 7) raise concern for increased risk of hemorrhage. Contrary to epidural hematomas, classic SDH are limited by dural duplications, but can cross suture lines and, therefore, can extend over an entire hemisphere. The vast majority of small, atypically shaped, extra-axial
hemorrhages are due to a low-pressure venous injury causing an SDH with continued CT follow-up suggested if clinical doubt remains as to whether an extra-axial blood collection reflects an EDH or SDH.

It is valuable for the radiologist to be generally aware of the appearance of blood products on CT based on the acuity. Although imaging may not occur this early, in the first few hours (hyperacute phase), SDH might be isodense to the underlying cortex, as it is still liquid (40-50 HU), and may not be detectable. Hemorrhage becomes increasingly dense as it coagulates and condenses during the acute phase (0-3 days) due to clot retraction by a decreased fluid component and relative increase in iron content in the red blood cells. Those clots measure between 80-100 HU and show the typical hyperattenuation. The attenuation decreases in the subacute phase as the blood products degrade and eventually becomes similar to CSF density in the chronic phase (Figure 8).21

SDH requires heightened vigilance and follow-up imaging to ensure early detection of complications, such as continued bleeding, rebleeding (8% of cases), midline shift, or herniation.22 Mixed attenuation blood in which isodense areas are intermixed by hyperdense areas suggest active (re)bleeding, which is termed the “swirl sign” on NECT. Cerebral spinal fluid

FIGURE 5. Delayed intraparenchymal hemorrhage. (A) Initial unenhanced axial CT demonstrates a nondisplaced longitudinal right temporal bone fracture (arrow). (B) Initial unenhanced axial CT demonstrates an equivocal left posterior frontal/temporal hemorrhage (arrow) raising the possibility of a coup-contrecoup mechanism. (C) Follow-up axial unenhanced CT one day later, demonstrates marked worsening with more typical appearing intraparenchymal hemorrhage (yellow arrow) and new intraventricular hemorrhage (white arrow) with associated midline shift.

FIGURE 6. Nonhemorrhagic cerebral contusions in a pedestrian struck by a car. (A, B) FLAIR (A) and GRE (B) MR axial images demonstrate bilateral frontal lobe FLAIR hyperintensities (arrows) without associated susceptibility artifact on GRE (B) most suggestive of a nonhemorrhagic cerebral injury.

FIGURE 7. Right subdural hematoma with mass effect in an elderly patient on anticoagulant who fell. (A) Axial unenhanced CT demonstrates an acute subdural hematoma (yellow arrow) with midline shift (blue arrow). (B) Improved midline shift after decompression craniotomy. A residual layer of acute residual blood products is present (blue arrow) as is an adjacent low-density fluid collection (yellow arrow) in a surgical bed reflecting diluted blood products.

FIGURE 8. Delayed intraparenchymal hemorrhage. (A) Initial unenhanced axial CT demonstrates a nondisplaced longitudinal right temporal bone fracture (arrow). (B) Initial unenhanced axial CT demonstrates an equivocal left posterior frontal/temporal hemorrhage (arrow) raising the possibility of a coup-contrecoup mechanism. (C) Follow-up axial unenhanced CT one day later, demonstrates marked worsening with more typical appearing intraparenchymal hemorrhage (yellow arrow) and new intraventricular hemorrhage (white arrow) with associated midline shift.
Imaging of Traumatic Intracranial Hemorrhage

in the subdural space (subdural hygromas) is thought to be related to subdural hematomas. An arachnoid membrane tear with a “flap-valve” mechanism results in CSF leakage into the subdural space.23,24

Epidural Hematoma

Hemorrhage into a potential space between the inner table of the skull and the dura mater occurs in 1% of minor head injury and 10% following head injury in patients who present in a comatose state.25,26 A provided history of head injury days-to-months prior to imaging should not eradicate the possibility of an EDH, as 20% to 50% of patients experience a symptom-free lucid interval.27 Of patients with an EDH, 91% suffer from an associated skull fracture.28 Typically supratentorially located, EDH frequently involves injury to the middle meningeal artery or anterior ethmoidal artery. Rapid hematoma expansion is common due to the relatively high pressure of arterial blood.29 Classic CT findings are a high-density convex or biconvex shaped extra-axial fluid collection that does not cross the sutures (Figure 9).

Venous epidural hemorrhage accounts for 5% to 10% of all EDH and occurs at specific locations including the vertex (superior sagittal sinus injury), anterior middle cranial fossa (sphenoparietal sinus injury), and

FIGURE 8. Evolution of subdural hematoma. (A-E) Multiple time-sequential unenhanced CTs of the head in an elderly patient on an anticoagulant who fell. (A) Day of the head injury. Bilateral parietal-occipital loculated acute subdural hematomas (yellow arrows) with a mixed-density subacute center (red arrow). (B) Day 2 with interval rebleeding with increasing density of the subdural hematomas (yellow arrows). (C) Day 3 with continued high-density blood products (yellow arrows). (D) Day 14 with decreasing density reflects degradation of blood products (yellow arrows). (E) Day 39 with chronic subdural hematomas demonstrating density similar to CSF (CSF = 3HU).

FIGURE 9. Classic appearance of epidural hematoma. (A) Axial and (B) coronal unenhanced CT of acute right temporal epidural hematoma (yellow arrows) confined by the squamous suture.

FIGURE 10. Venous epidural hemorrhage. (A) Lateral skull radiograph of a 2-month-old after fall showing acute skull fracture at the vertex (arrow). (B) Coronal T2 MR image showing biconvex high-intensity midline collection, layering symmetrically between vertex skull fracture and superior sagittal sinus, consistent with a venous EDH (arrow). In contrast to arterial EDH, venous EDH may cross the midline, as seen with this case, as the bleeding origin is centered underneath the suture.
posterior occipital region (transverse/sigmoid sinus injury). The latter can rapidly cause tonsillar herniation. However, the majority of venous EDH are unlikely to expand because venous pressure is insufficient to further strip off the dura from the skull.30 Similar to arterial EDH, an associated fracture/diastasis is common. Venous EDH typically displaces the sinus away from the fracture and, in contrast to arterial EDH, can occasionally cross suture lines (Figure 10). One should suspect a venous EDH if a skull fracture runs through the expected location of a dural sinus.

Traumatic Subarachnoid Hemorrhage

Traumatic SAH (tSAH) results from disruption of pial vessels with bleeding into the subarachnoid space. It is often coexistent with other pathologies in the setting of trauma. Vessel injury more commonly arises in the cerebral convexities/sulci following a high-impact mechanical force to the head. Of patients with severe TBI, 40% develop tSAH.34 Be cautious with SAH in the setting of acute trauma as differential diagnosis includes tSAH as well as nontraumatic SAH (eg, ruptured aneurysm) that may have preceded (and perhaps led to) the reported trauma (Figure 11). Differentiating traumatic from nontraumatic SAH by imaging is not always possible. However, SAH identified around the cerebral convexities with relative sparing of the basilar cisterns around the circle of Willis would favor trauma.35 Similarly, SAH associated with other signs of trauma on CT such as a contrecoup pattern associated with a parenchymal contusion may also favor a traumatic etiology. When doubt exists, the CT or traditional angiography may be performed for further evaluation to exclude an aneurysm.

Imaging characteristics that portend a worse prognosis include other evidence of brain trauma including brain contusions, especially if enlarging.36 Complications of tSAH include hydrocephalus and cerebral vasospasm.35,37 Table 2 summarizes differentiating key features of EDH, SDH and SAH.

Conclusion

Traumatic intracranial hemorrhage is an urgent finding requiring prompt and accurate evaluation by the interpreting radiologist with excellent communication and documentation of key findings that may affect patient management. Given the trauma and further complicating patient management, additional critical coexistent injuries may also be present and require urgent treatment. Thus, at many medical centers a multidisciplinary neurotrauma team
guides management and determines the need for surgical decompression/evacuation (Figure 12).

Furthermore, the radiologist must be vigilant about new and/or worsening edema, midline shift, signs of hydrocephalus, worsening hemorrhage (re-bleeding), or other complicating factors on follow-up studies that may alter patient management. Medical and surgical management in these patients is complex and multifactorial requiring timely and accurate imaging information, thus solidifying the radiologists’ key role in the management of traumatic ICH.

REFERENCES
Globe Injury After Head Trauma


Hahnemann University Hospital, Philadelphia, PA

Case Presentation
A 45-year-old woman with end-stage renal disease, hypertension, and diabetes presented with acute-onset, right-eye blurriness with floaters following head trauma. An unenhanced CT head/orbits was obtained (Figure 1) followed by MR (Figure 2).

FIGURE 1. Axial (A) and sagittal (B) CT images through the right orbit demonstrate a biconcave V-shaped hyperdensity in the posterior aspect of the right globe (arrows).

FIGURE 2. Axial T2-weighted (A) and gradient echo (B) MR images of the orbits. Folded membranes (white arrows) are seen in the vitreous humor that extend to the ora serrata anteriorly. T2-weighted hypointense subretinal fluid is demonstrated with associated susceptibility artifact representing blood products (red arrows).
Key Imaging Finding
- V-shaped hyperdensity along the retinal surface of the globe

Differential Diagnosis
- Retinal detachment
- Choroidal detachment
- Vitreous hemorrhage
- Lens dislocation
- Globe rupture
- Intraorbital foreign body

Discussion
Orbital trauma with the potential of impairment and/or loss of vision is a critical clinical matter with an important role for radiologists. The interpreting physician must be aware of the imaging appearances of common injuries to the orbit and globe. CT is likely to be the initial imaging modality in the setting of trauma. MR imaging may also be encountered for further evaluation, but it is important to exclude an orbital metallic foreign body before MR acquisition.1-12

Although nontraumatic causes of vision impairment exist, the history of trauma provides a more focused differential diagnosis in this case. The initial diagnostic considerations included retinal detachment, choroidal detachment, vitreous hemorrhage as well as other traumatic causes such as a lens dislocation, globe rupture, and a foreign body.

Differential Diagnosis
Retinal Detachment
The retinal layer is the innermost layer of the globe. Anteriorly, the retina is adherent to the ora serrata, which is a transitional zone between the anterior margin of the retina and the ciliary body. The ora serrata cannot be directly visualized on CT/MR; however, its position can be inferred at the 2 and 10 o’clock positions of the globe just posterior to the ciliary bodies in the axial plane. Posteriorly, the retina is attached to the optic disc with the other portions of the retina less adherently attached to the underlying choroid layer.2

Retinal detachment occurs when the retina separates from the choroid layer. This can result from a variety of etiologies including inflammatory, neoplastic, postoperative, or traumatic.2-3 Regardless of the cause, retinal detachment results in vitreous humor and/or blood collecting within the subretinal space. This fluid collection takes on a characteristic triangular V-shaped appearance, with the base at the ora serrata and the apex at the optic disc.1-3

Symptoms of retinal detachment include flashes of light, floaters, and peripheral field vision loss. Although radiographic imaging is not necessary to make the diagnosis, ultrasound (US), MR, and CT can be helpful to further evaluate and/or confirm clinical findings detected on the ophthalmoscopic examination.1 On US imaging, a folded free-floating membrane (representing detached retina) can be seen in the vitreous humor.1-2 On CT/MRI, a folded membrane can be visualized in the subretinal space fluid, which is usually detected as a vitreous hyperdensity on CT imaging. A variety of treatment procedures exist.3

Choroid Detachment
The choroid is the middle layer of the globe, situated between the scleral and retinal layers. Choroidal detachment occurs when choroidal vessels rupture and cause fluid to accumulate, subsequently leading to detachment of the choroid from the underlying sclera.2

Symptoms of choroid detachment are similar to those of retinal detachment. On CT/MRI, choroidal detachment appears as a lentiform fluid collection that is of mixed intensity/signal based on the components of the collection (simple vs hemorrhagic fluid). It is important to note that choroidal detachment is not limited by the ora serrata anteriorly (unlike retinal detachment).1-2 Additionally, unlike retinal detachment, choroidal detachment diverges as it approaches the optic disc and may not involve the posterior aspect of the globe.4 Treatment may be conservative/medical or surgical.

Vitreous Hemorrhage
Vitreous hemorrhage occurs as a result of retinal vessel perforation that causes hemorrhage in the vitreous humor.4 This is most commonly seen in the setting of diabetic proliferative retinopathy, retinal detachment/tear, orbital trauma, or orbital malignancy.5 Patients generally present with sudden painless loss of vision with associated “floaters” or shadows in vision. On CT, small dependent areas of hyperattenuation may be seen in the posterior segment of the globe representing regions of acute hemorrhage.4 Treatment of vitreous hemorrhage is usually conservative as nearly half of patients will show spontaneous resolution.5 In persistent cases, surgical treatment with posterior vitrectomy may be necessary.5

Lens Dislocation
The intraocular lens is held in place by the zonular fibers, which also attach to the ciliary body. Lens dislocation is most commonly due to trauma that disrupts the zonular fibers. Although anterior or posterior subluxation of the lens can occur, posterior dislocation is more common with the lens identified in the dependent portion of the vitreous humor.2,4 Symptoms are similar to those of retinal detachment, including blurry vision or loss of vision. Pain is usually absent, unless there are other injuries in addition to the dislocation of the lens.

Anterior dislocations are less common, but carry greater risk of complications, such as cornea or iris injuries, as well as acute glaucoma.6 Lens dislocation can also be nontraumatic, associated with systemic connective tissue disorders, such as Marfan syndrome, Ehlers-Danlos syndrome, and homo-cystinuria.2

On CT, the lens appears as a hyperdense lentiform structure in an abnormal position and/or orientation within the globe. Treatment varies and may be conservative, with corrective lenses and close follow-up, or surgical.2

Globe Rupture
Blunt or penetrating trauma may result in injury to the sclera and subsequent globe rupture. As the sclera is relatively thinner posteriorly to the extraocular muscle insertions, this is
often the location of the defect in blunt trauma. This is a clinically significant injury and is a common cause of vision loss. Imaging evaluation involves CT, with MRI recommended for further evaluation in cases with high clinical suspicion but no abnormality identified by CT. US is generally contraindicated when globe rupture is suspected.

Imaging findings of globe rupture include abnormalities in globe contour, decreased globe volume, and discontinuity of the sclera. Additional findings may include intraorbital air or foreign bodies. Less obvious injuries, however, may only exhibit subtle scleral discontinuity. Increased anterior chamber depth can also suggest globe injury. Treatment of open globe injuries includes antibiotics and typically urgent or emergent surgery. Presence of an afferent pupillary defect and poor visual acuity are poor prognostic indicators and indicate greater risk of the need for enucleation.

**Intraorbital Foreign Body**

Intraorbital foreign bodies typically result from penetrating trauma, and are often seen in conjunction with other orbital injuries, including in up to 40% of open globe injuries. Intraocular foreign bodies are usually found in the posterior compartment, and can lead to inflammation, infection, and vision loss if not identified and treated promptly. For-eign bodies can be classified as either organic or inorganic, with inorganic substances such as metal or glass most common. Wood has a variable appearance that appears more low attenuating acutely and may be mistaken for air.

CT is the imaging modality of choice, and the attenuation of the foreign body depends on its composition, with metal being highly hyperattenuating and glass being more variable but also generally hyperattenuating. MRI can also be used for imaging of nonmetallic foreign bodies, but only after the presence of a metallic foreign body is excluded. Treatment for an acute foreign body usually involves antibiotics and surgical removal.

**Diagnosis**

**Retinal detachment**

**Summary**

Although a broad differential was initially considered, the diagnosis was quickly narrowed based upon the imaging findings. A lens dislocation was discounted as a diagnosis as the hyper-density in the posterior globe did not have the morphological appearance of an ocular lens. A globe rupture was not suspected given that the globe contour and globe volume appeared grossly symmetric compared to the contralateral globe. No intraocular foreign body was identified on CT imaging.

Thus, the main diagnoses to distinguish between for this case were retinal detachment, choroidal detachment, and vitreous hemorrhage. The V-shaped abnormality extending from the ora serrata converging toward the optic disk is most consistent with a retinal detachment. Follow-up ophthalmoscopy confirmed the diagnosis of retinal detachment.

Careful evaluation of the orbit and its contents is important to overall patient outcome. Familiarization with radiographic findings of various ocular trauma etiologies is crucial in making the correct diagnosis and guiding appropriate ophthalmologic therapy.

**References**

Complex Facial Fracture


Hahnemann University Hospital, Philadelphia, PA

Case Presentation
A 33-year-old man presented to the emergency department after being hit by a car while crossing the street. Physical examination was significant for left orbital contusion without conjunctival hemorrhage, left eyebrow laceration, and a bloody nose. An unenhanced CT of the facial bones was obtained (Figures 1-4).

Key Imaging Findings
- Multiple facial bone fractures

Differential Diagnosis
- Transfacial fracture (Le Fort)
- Zygomaticomaxillary complex (ZMC) fracture

Naso-orbital-ethmoidal (NOE) fracture
Orbital fracture

Discussion
Facial bone fractures are routinely encountered in emergency radiology as the face is commonly involved in trauma. Specifically, traumatic injuries from motor vehicle accidents and assaults are common causes of midfacial fractures in addition to gunshot wounds and falls.\(^1\)\(^2\)

The midface bones support the facial soft tissues and function. These osseous
structures are, therefore, often compared to architectural buttresses with the stability of the bony buttresses coming from their attachment to the skull base or cranium. Search patterns along common fracture planes should include analysis of the buttresses and associated soft tissues to accurately diagnose and aid in treatment planning.

CT is the initial modality of choice for evaluating facial trauma. Many classic facial bone fractures and fracture patterns have been described, which largely comprise the differential diagnoses initially considered in this case. The radiologist must be ever observant because these fractures may not occur in isolation.

Differential Diagnosis

Transfacial fracture (Le Fort)
Le Fort fracture patterns are relatively common, occurring in approximately 25% of midface fractures. There are 3 Le Fort patterns, each with a unique fracture not seen in the other patterns:

- **Le Fort I.** Fracture of the anterolateral margin of the nasal fossa resulting in separation of the maxillary arch from the skull.
- **Le Fort II.** Fracture of the inferior orbital rim resulting in separation of the maxillary bone from the skull.
- **Le Fort III.** Fracture of the zygomatic arch resulting in separation of the face from the skull.

Absence of a pterygoid plate fracture rules out a Le Fort fracture as a pterygoid plate fracture is common to all Le Fort fracture patterns. However, the converse is not always true. In one study, up to one-third of pterygoid plate fractures were seen in the absence of a Le Fort fracture pattern. Pterygoid plate fracture not associated with a Le Fort pattern can be associated with other fracture patterns including ZMC fractures, displaced mandibular fractures, temporal bone fractures, and fractures of the sphenotemporal buttress.

Presence of a single Le Fort fracture pattern does not exclude other facial fractures including ZMC and NOE fractures.

Zygomaticomaxillary Complex (ZMC) Fracture

The ZMC is structurally important in maintaining facial width and profile, and fractures have potential to be severely disfiguring. The zygoma articulates with 4 other facial bones. Radiographically, ZMC fractures can affect up to 5 structures including the lateral orbital wall, orbital floor, anterior maxillary wall, lateral maxillary wall, or the zygomatic arch. Displaced ZMC fractures can increase orbital volume due to disruption to the lateral orbital wall.

Naso-orbito-ethmoidal (NOE) Fracture

Isolated nasal bone fractures may occur in low-velocity trauma with more complex NOE fractures involving the nasal bone likely in high-velocity trauma. NOE fractures occur along 5 fracture planes: the lateral nose and piriform aperture, the nasomaxillary buttress, the inferior orbital rim and floor, the medial orbital wall, and the frontomaxillary suture. For a fracture to be classified as NOE, 4 out of 5 planes must be involved. These fractures can be simple or comminuted.

NOE fractures are clinically significant due to involvement of the medial canthal tendon. The Markowitz-Mason classification is used to describe the degree of tendinous injury.

Type I. Fracture of a single large fragment.

Type II. Comminuted fracture with a preserved medial canthal tendon insertion.

Type III. Comminuted fracture involving the medial canthal tendon insertion.

Additional focus should be placed on the nasofrontal ducts, as fracture of the bone around the duct can result in mucocele formation if not properly treated.

Orbital Fracture

Orbital fractures can occur in isolation or as part of a more complex fracture pattern. Both Le Fort and ZMC fractures can involve the lateral orbital wall or orbital floor. NOE fractures can involve the medial orbital wall. Orbital fractures can be associated with surgical emergencies if there is entrapment of the ocular muscles or optic nerve. Fracture of the orbital roof can be associated with dural entrapment.

Orbital fractures have the potential for serious ocular injury including ruptured globe. Orbital fractures can be seen in up to 30% of facial fractures.

Diagnosis

Left Le Fort II, Le Fort III, and ZMC fractures. Bilateral orbital fractures.

Summary

Trauma resulting in multiple facial bone fractures are common. The interpreting radiologist must be familiar with the gamut of fracture patterns to guide management. This case is an excellent example of multiple fractures and fracture patterns that was accurately diagnosed using a systemic approach. This case also demonstrated a number of potential diagnostic pitfalls including: (1) Multiple Le Fort patterns can be seen on one side, (2) Le Fort fractures can be unilateral and, if bilateral, do not need to demonstrate the same pattern bilaterally, and (3) Le Fort fractures can be seen with additional fracture patterns.

References

3. Rhea JT, Novelline RA. How to simplify the CT approach. This case also demonstrated a number of potential diagnostic pitfalls including:
Diaphragmatic Hernia in a Patient with Chest Trauma

Nadia Lushina, M.D., Niveditha Thangaraj, M.D., Christopher Brown, M.D., Nancy Mohsen, M.D.

Hahnemann University Hospital, Philadelphia, PA

Case Presentation
A 27-year-old man was brought to the emergency department following a major motor vehicle collision as an unrestrained driver. On physical examination, he was in severe respiratory distress and had reduced breath sounds, palpable left rib fractures, and crepitus over the left chest. An initial chest radiograph was performed (Figure 1) followed by CT (Figures 2, 3).

**Figure 1.** Frontal chest radiograph demonstrates multiple displaced posterior left-sided rib fractures (arrows) with nonspecific opacification of the left hemi-thorax that is more focal at the base, just superior to the left diaphragm.

**Figure 2.** Coronal contrast-enhanced CT image of the abdomen demonstrates the stomach (St), spleen (Sp) and portions of the tail of the pancreas (P) herniating into the left hemi-thorax with intact portions of the left diaphragm noted laterally and appearing slightly thickened (arrow). The herniated contents slightly narrow at the defect, resulting in a so-called “collar sign.”

**Figure 3.** Axial contrast-enhanced CT at the thoraco-abdominal junction demonstrates the stomach (St) and spleen (Sp) in the left hemi-thorax abutting the posterior ribs resulting in a so-called “dependent viscera sign.” Other pathology is also present but suboptimally viewed, including a left rib fracture, subcutaneous emphysema, pneumomediastinum, and a left pneumothorax.
Key Imaging Findings
A left diaphragmatic hernia in the setting of major blunt chest trauma

Differential Diagnosis
Traumatic diaphragmatic rupture
Bochdalek diaphragmatic hernia
Morgagni diaphragmatic hernia
Hiatal hernia

Discussion
Diaphragmatic hernias are common and encountered routinely in chest imaging. However, traumatic rupture is less common compared to the ubiquitous hiatal hernia. An initial chest radiograph demonstrated nonspecific opacities in the left base. These abnormalities were further characterized on subsequent CT that revealed the opacities on radiography to represent a combination of the herniated abdominal contents in the inferior left hemi-thorax as well as pleural fluid (blood) and airspace disease (contusion and atelectasis).

The differential for this case includes traumatic and nontraumatic causes of diaphragmatic hernias and includes the eponymous Bochdalek and Morgagni hernias, a hiatal hernia, and traumatic diaphragmatic rupture. In addition to the location and CT appearance of the hernia, the history and presence of other significant traumatic pathology are key considerations when evaluating the diagnostic possibilities.

Differential Diagnosis
Traumatic Diaphragmatic Rupture
Rupture of the diaphragm is not common, but is identified in a small percent of patients with blunt trauma, with motor vehicles accidents being the most common cause. Penetrating injuries are more common causes of diaphragmatic injury. Traumatic diaphragmatic rupture from blunt trauma occurs more frequently in the posterolateral aspect of the hemi-diaphragms at sites thought to be relatively weaker structurally. It is more commonly diagnosed on the left side with a variety of reasons hypothesized for the disparity in laterality including the liver providing a degree of protection to the right hemi-diaphragm.

Traumatic diaphragmatic rupture is rarely an isolated CT finding, with other traumatic findings in the thorax such as a rib fracture(s), effusions, and pneumothoraces also present in the vast majority of cases. Diaphragmatic rupture in the setting of blunt trauma often results in larger diaphragmatic defects (often > 10 cm) compared to penetrating trauma, leading to a larger defect for upward herniation of abdominal organs due to negative intrathoracic pressure.

Diaphragmatic rupture may occasionally be suggested on chest radiography. Radiographic findings include diaphragmatic elevation, intrathoracic stomach bubble or other intrathoracic bowel, and abnormal location of an oro- or nasogastric tube tip. CT is more likely to delineate a traumatic diaphragmatic rupture due to the greater anatomic detail compared to chest radiographs. With modern scanners, the diaphragm and the defect will likely be directly visualized with coronal and sagittal reconstructions aiding visualization. In addition to directly identifying the diaphragmatic defect, other CT signs of diaphragmatic rupture include thickening of the remaining diaphragm due to blood and/or muscle retraction, the “dependent viscera sign” (herniated organs layering against the posterior ribs), and the “collar sign” (narrowing of herniated organs and/or fat at the diaphragmatic defect).

Diaphragmatic rupture has the potential to be overlooked at initial evaluation due to small size and/or obscuration from adjacent pleural and parenchymal changes. Additionally, more immediately life-threatening traumatic injuries may capture the attention of the radiologist and the clinicians. Diaphragmatic defects typically require surgical intervention. The herniated viscera are potentially at risk for complications if left untreated including obstruction, strangulation, and ischemia.

Bochdalek Diaphragmatic Hernia
Bochdalek hernia is a congenital diaphragmatic defect located posteriorly between the diaphragmatic pars costalis and pars costalis. It is the most common congenital diaphragmatic hernia in adults and is usually discovered incidentally on cross-sectional imaging. Bochdalek hernias usually contain fat, but abdominal viscera such as the kidney may also herniate through the defect. Controversially, they are more common on the left. They may be suggested by radiography, but can be occult. A common CT appearance is discontinuity of the posterior diaphragm with intrathoracic peritoneal fat herniation.

Morgagni Diaphragmatic Hernia
Morgagni hernia is a rare congenital diaphragmatic defect located anteriorly in the cardiophrenic space between the diaphragmatic pars costalis and pars sternalis. They are much more common on the right. Morgagni hernias may contain peritoneal fat, as well as abdominal solid organs and bowel. On plain films or CT, a Morgagni hernia will appear as a discontinuity of the diaphragm at the right cardiophrenic angle with intrathoracic peritoneal fat or solid organ herniation.

Hiatal Hernia
Hiatal hernias result from chronic widening at the esophageal hiatus due to weakening of the phrenoesophageal membrane. Prevalence increases with age. Hiatal hernias are generally categorized as sliding and paraesophageal, although mixed-type hiatal hernias also exist. When the defect is severe, other abdominal viscera and/or fat can enter the thorax from a hiatal hernia. Hiatal hernias are typically evaluated on barium fluoroscopy examinations and can also be detected on radiographs. CT is not typically done for the detection of hiatal hernia, but they are often encountered incidentally on CT.
Diagnosis

Left traumatic diaphragmatic rupture with intrathoracic herniation of the stomach, spleen, and tail of the pancreas in the setting of major blunt chest trauma

Summary

This case of a left-sided diaphragmatic hernia had an initial wide differential considered. The key to narrowing the differential to the correct diagnosis of a traumatic diaphragmatic rupture is the location of the hernia, the CT evidence and history of trauma.

A Morgagni hernia would be found anteriorly and is very rarely found on the left side. A Bochdalek hernia would be located more posteriorly and typically contains fat. Although abdominal organs can enter a Bochdalek hernia, it would be extremely unusual to encounter a Bochdalek hernia containing the stomach, pancreatic tail, and spleen. Although a very large hiatal hernia can contain other abdominal organs, the defect should be able to be localized to the esophageal hiatus. The defect on this case was posterolateral in the left hemi-thorax, separate from the esophageal hiatus. Furthermore, such a large hiatal hernia would be highly unusual in a 27-year-old.

After applying these observations to the diagnostic considerations, a traumatic diaphragmatic rupture is the most appropriate diagnosis. This is an excellent example of a less common cause of a diaphragmatic hernia. As the treatment is surgical, it is important for the radiologist to be aware of this entity and to be able to differentiate it from other congenital and acquired diaphragmatic hernias that are much more frequently encountered.

References

Lunate Dislocation

A 45-year-old man presented to the emergency department with acute left wrist pain after a fall on an outstretched hand. Physical examination showed significant soft-tissue swelling along the volar aspect of the wrist with diminished 2-point discrimination in the median nerve distribution. Frontal radiograph (Figure A) shows a triangular-shaped lunate (asterisk) with widening of the radiolunate space (red arrow) and abnormal overlap of the lunate and capitate (green arrow). Additionally, an ulnar styloid avulsion fracture was noted (blue arrow).

The lateral view (Figure B) revealed volar displacement and rotation of the lunate bone (asterisk). The remainder of the carpal bones were in normal anatomic position in relation to the radius.

Lunate dislocation is an uncommon traumatic wrist injury that requires urgent management and repair. The lunate becomes displaced volarly with preserved alignment of the remaining carpal bones in relation to the radius. These injuries typically occur with significant wrist dorsiflexion from high-impact trauma. A lunate dislocation almost always occurs due to concurrent perilunate ligament injuries, most significantly the dorsal radiolunate ligament.1,2

On a frontal radiographic view, a lunate dislocation will manifest as an increased radiolunate distance with partial overlap between the lunate and capitate. The lunate will appear triangular. On the lateral view, the lunate is volarly displaced, taking on the appearance of a “spilled teacup” with preserved alignment of the capitate with the radius. Additionally, the lunate will not properly align with the capitate or radius.1 A midcarpal dislocation demonstrates less lunate volar tilt as compared to a complete lunate dislocation and demonstrates dorsal subluxation of the capitate.2 With a perilunate dislocation, the lunate remains in normal alignment with the radius but the capitate becomes dorsally dislocated.1,2

Treatment of a lunate dislocation involves immediate lunate reduction and surgical repair of the injured perilunate ligaments. Delay in treatment may lead to early osteoarthritic changes and wrist instability.1

References
Knife Injury to the Cervical Spine

A 55-year-old man suffered a knife wound to the right neck. There was resultant quadriplegia with some sensory sparing in the lower extremities. Axial CT (Figure A) and axial gradient echo MR (Figure B) of the cervical spine demonstrated a transverse fracture of the right C3 lamina (arrow) with a small focus of adjacent soft-tissue emphysema identified on CT. Sagittal STIR MR image of the cervical spine (Figure C) revealed abnormal cord signal with a small focus of high-intensity signal abnormality in the anterior spinal cord near the midline consistent with focal injury/edema (arrow).

Traumatic injury to the spinal cord can be broadly categorized as complete and incomplete. Complete spinal cord injury refers to the total disruption of the spinal cord with expected loss of sensory and motor function inferior to the injured level.\(^1\)\(^,\)\(^3\) Incomplete spinal cord injuries have a wide variety of clinical syndromes depending on the extent and location of spinal cord disruption. Due to the known locations of ascending and descending tracts within the cord, the location of the injury to the spinal cord can be used to predict the neurological deficits. Many classic incomplete cord syndromes have been described with associated neurological deficits including central, dorsal, ventral, and Brown-Sequard syndrome.\(^2\)\(^,\)\(^3\)

This patient had neurological deficits most consistent with a ventral cord syndrome, which typically results in complete motor loss as well as loss of crude touch, temperature, and pain below the injury level. In addition to trauma, disc herniation, multiple sclerosis, and cord infarct/ischemia due to compromise of the anterior spinal artery can all result in ventral cord syndrome.\(^2\)

References

Acute Compression Fracture with Unipedicular Approach Kyphoplasty

An 88-year-old man presented with acute onset back pain following a fall. MRI of the lumbar spine was obtained with T1-weighted sagittal (Figure A) and STIR sagittal (Figure B) sequences. There was signal and height loss on the T1 sequence in a lumbar vertebral body (arrow in A) with corresponding increased signal on the STIR sequence (arrow in B), consistent with marrow edema and an acute lumbar compression fracture. The patient had persistent pain despite appropriate medical management. Therefore, a unipedicular kyphoplasty was performed using radiopaque bone cement (Figures C, D).

Vertebral compression fractures are a common injury in the aging population with a prevalence approaching one-third of patients ages 80 years and older.1,2 Compression fractures may be described by the part(s) of the vertebral body involved including wedge-shaped, biconcave, or crush-type deformities.1 These fractures may be detected on a variety of imaging modalities including radiographs, CT, and MR. However, MR is the preferred imaging modality as MR can evaluate for bone edema to help determine the age of the fracture.1

Patients with compression fractures who fail to achieve adequate pain control with conservative management may be considered for augmentation procedures such as kyphoplasty and vertebroplasty.2 These are procedures in which bone cement is injected into the fractured vertebral body. Kyphoplasty is distinct from vertebroplasty as kyphoplasty procedures utilize a balloon catheter to attempt to restore vertebral body height and to create a potential space for the injected bone cement.2 Given this extra step, it is more common to see restored vertebral body height with kyphoplasty compared to vertebroplasty.3

References