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

Associate Editor
Tammam Beydoun, D.O., Phoenix, AZ

Editorial Board
Christopher Cerniglia, D.O.    Rocky Saenz, D.O.
Dell Dunn, M.D.                Susann Schetter, D.O.
Bernard Laya, D.O.             Clayton Trimmer, D.O.
John Lichtenberger, M.D.       Frederick White, D.O.
Timothy McKnight, D.O.         Michael Zapadka, D.O.
From the Editor

In this Issue ............................................................................................................................................ 4
Alysha Vartevan, D.O.

Review Articles

Overview of Parotid Gland Masses ........................................................................................................ 5
Andrew Teh, D.O., Aswin Kumar, D.O., Claire Teh, OMS II, Lea Alhilali, M.D.

MRI Degenerative Disease of the Lumbar Spine: A Review ................................................................. 11
Mark Buller, M.D.

Differential-Based Case Reviews

Cystic Neck Mass ..................................................................................................................................... 20
Aswin Kumar, D.O., Andrew Teh, D.O., Alysha Vartevan, D.O.

Enhancing Intraventricular Atrial Mass ................................................................................................. 23
Anirudh Chaudhary, M.D., Alysha Vartevan, D.O.

JAOCR at the Viewbox

Discitis/Osteomyelitis ............................................................................................................................... 26
Nicholas Matthees, M.D., Alysha Vartevan, D.O.

Transient Global Amnesia ....................................................................................................................... 27
Mark Bailey, M.D., Alysha Vartevan, D.O.
“Nothing in life is to be feared, it is only to be understood. Now is the time to understand more, so we may fear less.”

Marie Curie

In this Issue

Alysha Vartevan, D.O.
Clinical Instructor in Neuroradiology, University of Arizona College of Medicine, Phoenix, AZ

I would like to take the time to thank Dr. O’Brien and the JAOCR for the opportunity to serve as guest editor for this issue dedicated to neuroradiology. Editing and organizing this issue gave me the chance to work with many wonderful colleagues. I want to thank each of the authors for dedicating their time and energy to writing the articles to make this issue a success.

The articles included in this neuroradiology-focused issue were selected with the general practicing radiologist in mind. Both review articles were intended to help with everyday challenges radiologists face. The first review article focuses on a topic that most radiologists dread: head and neck. The “Overview of Parotid Gland Masses” article discusses the most common benign and malignant pathologies that you will encounter. Additionally, the article discusses patterns of disease spread and treatment recommendations to add value for our referring clinicians.

The second review article, which focuses on degenerative spine imaging, is “Magnetic Resonance Imaging of Degenerative Disease of the Lumbar Spine: A Review.” It is a fantastic overview of lumbar spine anatomy and the accepted degenerative lumbar spinal nomenclature. Given the prevalence of back pain and spine imaging that is regularly performed, this article is intended to be a go-to reference for lumbar imaging.

Next, the two cases selected for the case review section are intended to help develop a differential diagnosis for common pathologies: the cystic neck mass and the intraventricular mass. Each case provides the top three (Dr. O’Brien’s favorite number) differential diagnoses, the final diagnosis, and a discussion on each pathologic entity.

Lastly, the two cases selected for the Viewbox section are intended to show the classic imaging findings to establish a case as an “Aunt Minnie.” Both the discitis/osteomyelitis and transient global amnesia Viewbox cases have helpful clinical and imaging findings to guide radiologists to the ultimate diagnosis.

I hope this dedicated neuroradiology issue will serve as a useful learning tool for the radiology trainee and a good review for the seasoned radiology veteran. I again want to thank each of the contributing authors; this issue could not have happened without their hard work and dedication. I also want to thank my former attending neuroradiologists at Barrow Neurological Institute for their guidance and input. Lastly, I want to thank my husband for always supporting my passion for radiology. Hopefully this issue will reignite all of our love for neuroradiology and introduce trainees to this wonderfully challenging subspecialty. I hope you all enjoy this neuroradiology-focused issue of the JAOCR.
Overview of Parotid Gland Masses

Andrew Teh, D.O.,¹ Aswin Kumar, D.O.,¹ Claire Teh, OMS II,² Lea Alhilali, M.D.³

¹Department of Radiology, Larkin Community Hospital, South Miami, FL
²Touro University Nevada College of Osteopathic Medicine, Henderson, NV
³Department of Neuroradiology, Barrow Neurological Institute, Phoenix, AZ

Of the major salivary glands, the parotid gland has the highest rate of tumor association, accounting for 64% to 80% of primary epithelial salivary gland tumors. Most parotid tumors are benign with malignancy only comprising approximately 15% to 32%.¹ The typical clinical presentation is a painless mass or swelling in the cheek about the mandibular rami. Facial nerve involvement generally suggests a malignant tumor, which may present with pain or paralysis. Imaging studies provide insight on the degree of glandular involvement, the nature of the tumor, and potential spread, and serve as an important baseline for therapeutic interventions. Familiarity with the normal anatomy of the parotid gland, as well as the imaging characteristics of common neoplasms involving the parotid gland, is crucial in establishing appropriate differential diagnoses and guiding clinical management.

Benign Pleomorphic Adenoma/Benign Mixed Tumor

Pleomorphic adenomas, commonly referred to as benign mixed tumors (BMTs), are the most common benign salivary gland tumors (70% to 80%). Initially presenting as a slow-growing, painless cheek mass, these neoplasms typically appear in middle-aged females 30 to 60 years old.²³ They are mixed tumors comprised of epithelial and myoepithelial cells.

On US, the appearance of BMT is a homogeneous hypoechoic, well-circumscribed mass. A nuclear medicine (NM) pertechnetate scan shows a photopenic/cold defect, differentiating them from a Warthin tumor (typically hot), but the appearance is indistinguishable from malignant parotid lesions (usually cold).¹⁴ CT will demonstrate a well-circumscribed, homogeneously enhancing ovoid mass. Larger BMTs can show some heterogeneity to their enhancement pattern and may even present with central necrosis or dystrophic calcifications.⁵ T1 MRI sequences show a homogeneous hypointense mass, with larger BMTs showing hyperintense foci in cases of intratumoral hemorrhage. T2-weighted sequences will show uniform intermediate to high signal (Figure 1); very high T2 intensity greater than cerebrospinal fluid is fairly specific for BMTs. Diffusion-weighted imaging (DWI) usually shows higher apparent diffusion coefficient (ADC) signal compared to other parotid tumors, but this is not accurate enough to preclude biopsy. Contrast studies vary, ranging from mild to moderate enhancement (Figure 2).

Although benign, up to 15% of untreated pleomorphic adenomas can undergo malignant transformation, known as carcinoma ex pleomorphic adenoma. Characteristics include rapid growth over the course of a few months and pain.¹ For both conditions, surgical excision remains the gold standard, although recurrence is common if the tumor extends past its capsule. A partial or total parotidectomy has been found to dramatically decrease recurrence rates compared to lesional excision.⁶

Warthin Tumors

Warthin tumors are the second most common benign salivary gland tumor, accounting for 10% of parotid tumors. They present with painless swelling, with 20% of lesions appearing multifocal (unilateral or bilateral). Warthin tumors are classically seen in elderly men in the 6th decade of life, with a strong association with smoking and radiation exposure.¹⁷ Warthin tumors have also been termed
Overview of Parotid Gland Masses

Lymphomatous papillary cystadenoma, corresponding with their histological characteristics: glandular structures with papillary cystic arrangement, along with a stroma filled with lymphoid tissue. \(^1\)

Contrast-enhanced CT shows a smoothly margined, ovoid mass occasionally located in the tail (posterior portion) of the superficial lobe of the parotid gland (Figure 3). Cystic components can be seen in up to 30% of lesions and may be difficult to differentiate from a cystic lymph node, branchial cleft cyst, or other cystic mass. Presence of a mural nodule may also be suggestive of a Warthin tumor. \(^{5,8,9}\) T1 MRI sequences typically show low signal in the solid and cystic components, although the presence of proteinaceous debris or hemorrhage may increase the T1 signal. Solid components show minimal enhancement (Figure 4). On US, Warthin tumors will show well-defined anechoic areas toward the tail of the superficial parotid gland reflecting cystic components. \(^{5,8,9}\)

The incidence of malignancy is <1%. Management involves either surgical excision or routine monitoring, which can be advantageous to avoid iatrogenic complications. \(^7\) Local recurrence is exceedingly rare but more likely in multifocal disease.

**Facial Nerve Schwannoma**

Facial nerve schwannomas (FNSs) are rare benign neoplasms arising from Schwann cells along cranial nerve (CN) VII, the facial nerve. In the parotid parenchyma, they may present similarly to pleomorphic adenomas as a painless and slow-growing mass. Uncommonly, they present with facial weakness or paralysis. Multiple schwannomas have an association with neurofibromatosis type 2 (NF-2).

Imaging findings of FNS on contrast-enhanced CT are a round or oval well-circumscribed enhancing intraparotid mass. Proximal lesions may cause...
enlargement of the stylomastoid foramen. MRI shows a well-defined mass that is T1 isointense and T2 slightly hyperintense to muscle with enhancement on postgadolinium images (Figure 5). Larger lesions may have a characteristic intramural cyst.

Preoperative diagnosis of FNS is extremely difficult and uncommon. Diagnosis is often made intraoperatively via electrical stimulation and tissue biopsy, followed by radiographic staging to determine neoplastic extent. Total resection is curative; however, this may be declined if the nerve cannot be salvaged.

Benign Lymphoepithelial Lesions (BLELs)

Benign lymphoepithelial lesions (BLELs) are relatively common in HIV patients and are sometimes concurrent manifestations of Sjögren syndrome. Both parotid glands are often involved and can range from purely cystic lesions to mixed cystic and solid masses. They occur more frequently in women than men (3:1), and within the 4th to 7th decades of life. Similar to other benign parotid masses, these typically present as painless swelling with enlargement of the parotid glands.

Imaging features overlap with Warthin tumors of the parotid and show bilateral cystic and solid masses within enlarged parotid glands (Figure 6). US shows the cystic components to be anechoic with variable posterior acoustic enhancement. Solid components are predominantly hypoechoic in appearance, with identified intraparotid lymph nodes showing prominent cortex and hilar architecture. CT will show bilateral solid and cystic masses involving the parotid glands. Postcontrast images show thin rim enhancement of the cystic components and heterogeneous enhancement of the solid components. MRI sequences show hypointense T1 and hyperintense T2 signal in cystic components with variable enhancement of the solid components. Waldeyer’s lymphatic ring is typically enlarged with high T2 signal and can suggest BLEL in an HIV patient.

Histology shows lymphocytic infiltration with lymphocytes and germinal center hyperplasia, resulting in atrophy of the parotid parenchyma. Malignant transformation is rare and can arise from the epithelial or lymphoid component, known as lymphoepithelial carcinomas (LEC). BLEL may be monitored, whereas LEC should be excised along with lymph node dissection or with radiation therapy.

Malignant

Mucoepidermoid Carcinoma

Mucoepidermoid carcinoma is the most common primary malignant tumor of the parotid gland. Initial presentation is a palpable parotid mass. Additional symptoms may include pain, facial nerve paralysis, or sensory deficits in the V3 distribution. These tumors typically affect adults ages 35 to 65 years but can also occur in children. Histology consists of epidermoid and mucous-secreting cells. Treatment depends on the grade of tumor, with local resection sufficient for low-grade tumors, but wide surgical excision and
radiotherapy required for high-grade lesions.\textsuperscript{16-17}

Imaging characteristics may vary based on the histologic grade of the tumor. Low-grade lesions can present as a well-circumscribed parotid mass, mimicking benign entities, while high-grade lesions can have ill-defined or infiltrative margins (Figure 7). Evaluation for malignant nodes or perineural spread along CN VII is important for accurate staging (Figure 8). Loss of the normal fat in the stylomastoid foramen, abnormal enhancement in the mastoid segment of CN VII, or osseous involvement of the mandible or skull base indicates a higher-grade malignancy and delineates the extent of disease.\textsuperscript{8}

Contrast-enhanced CT will typically show an enhancing soft-tissue mass in the parotid gland. Cystic changes can be seen due to mucous-producing cells. On MRI, the lesion will have heterogeneous T1 and T2 signal with areas of high T2 signal indicating cystic changes. Indistinct margins suggest a higher-grade tumor. DWI may show restricted diffusion and/or low ADC signal, but is non-specific, as a Warthin tumor may show similar findings.\textsuperscript{8} Enhancement is typically heterogeneous, with cystic components having little enhancement.\textsuperscript{8,11}

Recurrence rate correlates with higher histologic grade. Lower-grade tumors have been reported to have up to a 90\% 10-year survival rate. Evidence of metastatic spread or infiltrative margins portends a poorer prognosis and increased rate of recurrence. Late recurrence is possible and routine monitoring for up to 10 years is recommended.\textsuperscript{8,16-17}

**Adenoid Cystic Carcinoma**

Adenoid cystic carcinoma (ACC) is the second most common primary malignancy of the parotid gland. The lesion presents as a slow-growing parotid mass with pain reported in up to one-third of
cases. Peak incidence is between the 5th and 7th decades, and it is rarely seen before age 20. Among all head and neck tumors, ACC has the highest propensity for perineural spread.\textsuperscript{19}

Imaging characteristics include an enhancing parotid mass with either well-circumscribed borders or infiltrative margins depending on the histologic grade. Enhancement on CT or MR is typically homogenous with T1- and T2-weighted images showing variable low to intermediate signal intensity (Figure 9). DWI may show restricted diffusion but is nonspecific in differentiating ACC from a benign Warthin tumor.\textsuperscript{8} As with all parotid masses, but especially ACC, close attention should be paid to potential perineural spread.\textsuperscript{8,11}

ACC typically has a good short-term prognosis but poor long-term prognosis. Late recurrence can occur up to 20 years after diagnosis. Treatment is typically surgical resection with postoperative radiotherapy. Metastatic involvement of the lungs and bones is more common compared with lymph node spread.\textsuperscript{8,18}

**Lymphoma**

Lymphoma of the parotid glands is of the non-Hodgkin lymphoma (NHL) variety with three distinct forms: primary nodal, systemic, or primary parenchymal. Initial presentation is of a painless, enlarging parotid mass with cervical lymphadenopathy. Mean age of presentation is 55 years with a 1.5:1 male-to-female predominance.\textsuperscript{19}

Imaging characteristics of parotid NHL depend on the type. Nodal NHL usually presents as a well-circumscribed lesion, while the parenchymal type can have infiltrative or indistinct margins. Contrast-enhanced CT shows mild to moderate enhancement and frequent periparotid or upper cervical

---

**FIGURE 9.** Adenoid cystic carcinoma. Axial T2 image with fat suppression (A) shows a large, lobulated, infiltrative mass involving the right parotid gland that is slightly hyperintense to muscle (arrows). Coronal T1 precontrast (B) and fat-suppressed postcontrast (C) images better demonstrate the infiltrative and aggressive margins of the enhancing right parotid mass (arrows). Case courtesy of Courtney Tomblinson, M.D.

**FIGURE 10.** Lymphoma. Axial unenhanced CT image (A) shows a round, circumscribed mass isoattenuating to muscle in the left parotid gland. Fused PET/CT (B) shows avid FDG uptake. Axial T2 image (C) demonstrates homogeneous low signal with mild heterogeneous enhancement on fat-suppressed postcontrast T1 images (D).
lymphadenopathy. MRI may show an intermediate T1 signal intensity mass within a background of hypointense parotid gland. Post-gadolinium administration shows mild to moderate enhancement (Figure 10). F-18 fluorodeoxyglucose (FDG) PET/CT will show avid activity in nodal NHL.\(^{11}\)

NHL of the parotid has an increased incidence with autoimmune disorders or immunosuppression and is frequently associated with Sjögren syndrome, rheumatoid arthritis, or systemic lupus erythematosus. Treatment is typically with chemotherapy and radiation.\(^{1,8,19}\)

**Metastases**

Metastases should be a consideration for parotid lesions in patients with a known malignancy, especially head and neck malignancy, such as squamous cell carcinoma. Skin lesions involving the face and scalp, such as squamous cell carcinoma or melanoma, account for the majority of parotid metastases. Systemic metastases to the parotid gland are extremely rare, usually originating from lung or breast cancers.

Imaging findings include one or more intraparotid masses. Cervical lymphadenopathy may also be present. Lesions can be well circumscribed or have indistinct margins. Enhancement pattern is typically homogenous, although if necrosis is present, there may be central areas of decreased enhancement. MR is the best modality for determining perineural spread, and FDG PET/CT can be helpful in assessing involvement of small extra parotid nodes and other sites of metastatic disease.\(^{8,11}\)

**Summary**

Parotid masses have a variety of etiologies that range from benign to malignant. Although many lesions have some overlapping features, imaging appearance and patient demographics often aid in narrowing the list of differential considerations. Familiarity of the imaging characteristics of common parotid masses is critical in providing a comprehensive evaluation that includes determining lesion etiology, assessing staging for malignant lesions, and guiding overall management.

**References**

Low back pain is an exceedingly common problem, with a lifetime prevalence of 70% to 85%. This condition is the most common cause of disability in people ages 45 years or younger, with an estimated economic impact of over $100 billion dollars per year, predominantly due to loss of productivity. The etiology of low back pain is multifactorial and is influenced by genetics, age, sex and mechanical stresses.

Imaging plays a critical role in the diagnosis of low back pain. MRI has become a mainstay in the workup of low back pain due to its excellent soft tissue contrast, cross-sectional capability, and lack of ionizing radiation. This paper will present common MRI findings associated with low back pain, as well as grading systems and common nomenclature to assist in consistent and reproducible reporting of these findings.

MRI Imaging Techniques

An MRI of the lumbar spine generally includes a sagittal T1-weighted spin echo sequence, a sagittal T2-weighted spin echo sequence, and axial T2-weighted images. Additional sequences including axial T1-weighted sequences, sagittal fat-nulling T2-weighted sequences such as short tau inversion recovery (STIR) or modified Dixon (mDixon), and gadolinium-based contrast enhanced T1-weighted sequences may be obtained depending on the institution and the indication for the MRI examination.

Sagittal T1-weighted images are useful in the assessment of bone marrow, which is normally fatty in adults and demonstrates high T1 and T2 signal. Alignment of the vertebral bodies can also be assessed on the sagittal T1-weighted sequence. Due to the high contrast between fat and nerve roots, the T1 sagittal sequences are excellent for assessing the degree of neural foraminal stenosis.

Sagittal T2-weighted images provide excellent contrast between cerebrospinal fluid (CSF) in the thecal sac and the surrounding structures, allowing for assessment of the degree of spinal stenosis at multiple levels on a single image. These sequences are also useful for assessment of the intervertebral discs, and the presence of disc herniation. Fluid sensitive sequences such as STIR and mDixon are used for detecting areas of bone marrow edema.

Axial T2-weighted images provide a level-by-level assessment of the relationship between the thecal sac and the surrounding bony and ligamentous structures and are particularly useful for assessing spinal stenosis and narrowing of the lateral or subarticular recesses. These sequences are also used in assessing the facet joints and ligamentum flava.

In addition to the previously mentioned techniques, several other MRI sequences can be used for assessment of the lumbar spine. There is evidence to suggest that upright MRI of the lumbar spine provides a more accurate assessment of the physiology of low back pain as many patients are more symptomatic when standing. However, limited availability, high false-positive rates, and increased motion artifact have limited widespread adoption of this technique. Other techniques such as T1 and T2 relaxation mapping and new sequences like sodium MRI, magic echo and T1ρ are being developed to assess early molecular changes in the intervertebral disc.

Normal Anatomy

Vertebral Bodies

Lumbar vertebrae are composed of a vertebral body anteriorly, which gives rise to bilateral pedicles from its superior aspect. These extend posteriorly and connect to the transverse processes, which project laterally, and the lamina, which project posteromedially. The
lamina come together in the midline and connect to the posteriorly projected spinous process. Interposed between each pedicle and lamina are the superior and inferior articular processes, joined by the pars interarticularis.

The vertebral bodies consist of an outer layer of cortical bone, which is low signal intensity on T1- and T2-weighted imaging and surrounds the inner trabecular bone. Trabecular bone is normally high signal on T1- and T2-weighted sequences in adults due to its fatty marrow.

The posterior wall of the vertebral body and inner margins of the pedicles and lamina form a bony ring around the thecal sac. The neural foramina are bordered superiority and inferiorly by the pedicles of adjacent vertebral bodies, anteriorly by the posterolateral margin of the suprajacent vertebral body and intervertebral disc, and posteriorly by the superior articular process of the subjacent vertebral body. Neural foramina allow the passage of lumbar nerve roots from the thecal sac to the peripheral tissues. Nerve roots within the neural foramina are low signal on T1- and T2-weighted imaging and are normally surrounded by a rim of perineural fat.

### Intervertebral Discs

Interposed between the vertebral bodies are the intervertebral discs (Figure 1). These discs form the anterior articulation of the vertebral column and have two components: the outer annulus fibrosis (AF) and the inner nucleus pulposis (NP). The AF is a dense fibrocartilaginous structure comprised of 15 to 20 layers of obliquely oriented fibers that run from the inferior endplate of the suprajacent vertebral body to the superior endplate of the subjacent vertebral body. These fibers are primarily comprised of type 1 collagen. This portion of the intervertebral disc normally demonstrates low T1 and low T2 signal. The NP is composed of a loose type 2 collagen matrix and is 70% to 90% water and proteoglycans. The NP demonstrates high T2 and low T1 signal, due to its high water content. A low T2 signal band can be seen centrally within the NP in patients over age 30 and represents a fibrous band or cleft. These discs contact the vertebral body endplates, which are made up of hyaline cartilage.

### Table 1. Pfirrmann Classification for Disc Degeneration*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Disc Structure</th>
<th>NP/AP Distinction</th>
<th>NP Signal</th>
<th>Disc Height</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Homogeneous</td>
<td>Sharp</td>
<td>Bright hyperintense</td>
<td>Preserved</td>
</tr>
<tr>
<td>2</td>
<td>Inhomogeneous</td>
<td>Sharp, cleft may be present</td>
<td>Hyperintense</td>
<td>Preserved</td>
</tr>
<tr>
<td>3</td>
<td>Inhomogeneous</td>
<td>Unclear</td>
<td>Isointense</td>
<td>Preserved or slightly decreased</td>
</tr>
<tr>
<td>4</td>
<td>Inhomogeneous</td>
<td>No distinction</td>
<td>Hypointense</td>
<td>Mild to moderate decrease</td>
</tr>
<tr>
<td>5</td>
<td>Inhomogeneous</td>
<td>No distinction</td>
<td>Black signal</td>
<td>Collapsed</td>
</tr>
</tbody>
</table>

*Modified from reference 19
cartilage on the vertebral body side and fibrocartilage along the disc.\textsuperscript{11}

**Facet Joints**

The posterior articulation of the vertebral bodies is formed by the facet (zygoapophyseal) joints. These are obliquely oriented synovial joints comprised anterolaterally of the superior articular process of the subjacent vertebral body and posteromedially by the inferior articular process of the suprajacent vertebral body. Facet joints have articular surfaces composed of hyaline cartilage within a fibrous joint capsule lined with synovium.\textsuperscript{11} The joint spaces of the facet joints normally measure 2 to 4 mm and demonstrate isointense to high T2 signal.\textsuperscript{14}

**Ligamentum Flava**

Multiple ligamentous structures contribute to the stability of the spinal column. These include the anterior longitudinal ligament, the posterior longitudinal ligament, the interspinous ligament, and the supraspinous ligament. Of particular interest when considering degenerative disease of the lumbar spine are the ligamentum flavum, paired ligaments that extend between the lamina of adjacent vertebral bodies. These ligaments are normally thin and low signal on T1- and T2-weighted sequences.

**Degenerative Disease**

**Intervertebral discs**

Normal intervertebral discs transition through three phases: growth, maturation, and degeneration.\textsuperscript{15} The growth phase is characterized by synthesis of aggrecan and procollagens and increased type 2 collagen and takes place between ages 0 and 15. The maturation phase occurs with a reduction in the synthesis and volume of type 2 collagen in the NP from approximately 15 to 40 years of age. The final stage is degeneration, characterized by increased fibrosis with decreasing type 2 collagen and increasing type 1 collagen, which takes place after age 40.

This disc degeneration, as well as annular fissures and apophyseal osteophyte formation, in the absence of disc height loss, have been termed spondylosis deforms and are considered normal processes associated with aging.\textsuperscript{16-18} On MRI, apophyseal osteophytes are characterized by low T1 and T2 outgrowths along the anterior and lateral margins of the endplates. Disc degeneration manifests as loss of T2 signal in the NP. Annular fissures are small areas of T2 hyperintensity in the posterior AF. More extreme changes, including severe disc fissuring, disc height loss and endplate erosion, have been termed intervertebral osteochondrosis, which is a pathologic process.\textsuperscript{16-18}

Pfirrmann et al proposed a grading system for intervertebral disc degeneration based on disc structure, distinction between the NP and AF, NP signal intensity and disc height (Table 1).\textsuperscript{19} This grading system demonstrated good interobserver reliability. A review of the literature in 2005 by Kettler et al found that the Pfirrmann grading system was the only MRI-based system for disc degeneration with a kappa value of greater than 0.6.\textsuperscript{20} This system has since been modified by Griffith et al to increase the discriminatory power in the elderly population, with three additional severity levels and a quantitative measurement of disc height reduction.\textsuperscript{21}

Annular fissures are regions of high T2 signal intensity seen in the posterior AF of degenerated discs (Figure 2). Nearly all degenerated discs have annular fissures, although these may not be visible on MRI.\textsuperscript{22} The role of annular
fissures in pain generation is uncertain, with multiple studies noting that annular fissures are often seen in asymptomatic individuals.\(^{23-26}\) Additionally, evidence suggests that the presence of annular fissures does not increase progression of degenerative disc disease when compared to discs without fissures.\(^{27}\) For these reasons, the Combined Task Force (CTF) of the North American Spine Society, American Society of Spine Radiology and American Society of Neuroradiology recommend the term “annular fissure” instead of “annular tear” to avoid implying that these regions of signal intensity are a type of acute disc injury.\(^{28}\)

Another common disc-related finding in degenerative disease of the lumbar spine is disc herniation. There are multiple systems for classification of disc herniations; however, the two most studied systems are those proposed by Jensen et al.\(^{29}\) and Fardon et al.\(^{28}\) The Jensen classification system splits disc herniations into three categories: disc bulges, protrusions and extrusions. Bulges are defined as symmetric extensions of disc material beyond the interspace. Protrusions are focal or asymmetric extensions of disc material beyond the interspace with the base of the herniation being wider than the apex. Finally, extrusions are defined as more extreme extensions of disc material beyond the interspace with the dimension of the extruded component either wider than the base or not connected to the base. The findings of the CTF published by Fardon et al do not consider disc bulges a form of herniation as bulging can be a normal variant or the result of adjacent bony remodeling or ligamentous laxity.\(^{28}\)

The CTF defines protrusions and extrusions as involving less than 25% of the circumference of the disc, in addition to the features described by Jensen et al (Figure 3). Additional terminology proposed by the CTF includes “sequestration” as a subset of extrusion where the extruded material is not continuous with the parent disc (Figure 4). A recent systematic review of the literature found that the recommendations by the CTF demonstrated superior interrater reliability compared to Jensen et al.\(^{30}\)

The CTF recommends that this classification be coupled with the localization system proposed by Wiltsie et al.\(^{31}\) which divides the spinal canal into central, subarticular, foraminal and extraforaminal zones (Figure 5). An alternate method for classification of disc herniations differentiates subligamentous herniations from extra-ligamentous herniations. This classification scheme, proposed by Oh et al,\(^{32}\) describes five criteria that can be used to determine extra-ligamentous herniations: spinal canal compromise of more than half its dimension, internal signal difference in the herniated disc, ill-defined margin of the herniation, disruption of low-signal intensity line covering the herniation, and the presence of an internal dark line in the herniated disc. The authors note that this type of classification is potentially more clinically useful as minimally invasive methods are more successful with subligamentous disc herniations than extra-ligamentous herniations.

Another clinically oriented classification scheme was developed at Michigan State University by Mysliwiec et al.\(^{33}\) This grading system proposes to separate disc herniations into those that are “substantial” and require surgical intervention, and those that are not and are more likely to have poor surgical outcomes. The authors propose using a line drawn between the anterior margins of the facet joints in the axial plane
as a reference for the extent of disc herniation. Those herniations that do not extend more than 50% of the distance between the interspace and intrafacet line are classified grade 1 and should be managed conservatively. Those extending beyond grade 1 but not beyond the intrafacet line (grade 2), and those extending beyond the intrafacet line (grade 3) were treated surgically, with good outcome rates that compared favorably to existing literature. While this technique is less subjective than other schemes, it is somewhat limited in that it cannot be used in patients with abnormal facet joints and ligamentum flavum hypertrophy.

**Vertebral Body Endplates**

Degenerative endplate changes have been classified by Modic et al.\(^{34,35}\) into three categories: Type 1 changes are edematous changes related to subchondral end plate fractures, formation of vascularized fibrous tissue and an acute reparative response. On MRI, these changes are characterized by increased T2 and decreased T1 signal in the bone marrow adjacent to the endplate. Type 2 changes are related to fatty replacement of normal marrow and are more chronic and stable. These changes will demonstrate increased T1 and T2 signal, with loss of signal on fat suppression sequences. Type 3 changes relate to chronic endplate sclerosis and development of dense woven bone. This dense bone is low signal intensity on both T1- and T2-weighted sequences. Transitions through these stages are not uniformly progressive, and multiple studies have shown resolution of Type 1 changes or progression from Type 2 change to Type 1 change.\(^{35-37}\) Type 1

---

**FIGURE 4.** Disc sequestration. Axial T2-weighted image (A) demonstrates herniated disc material in the left lateral recess (asterisk), posteriorly displacing and compressing the traversing nerve root (arrow). Para-midline sagittal T2-weighted image (B) of the same disc sequestration (asterisk), which demonstrates higher T2 signal than the parent disc. Note the lack of continuity with the parent disc (arrowhead). Pre-contrast T1-weighted (C) and postcontrast T1-weighted (D) axial images demonstrate the T1 hypointense sequestration (asterisk) with thin peripheral enhancement, again displacing and compressing the traversing nerve root (arrow).

**FIGURE 5.** Anatomic scheme proposed by Wiltse et al.\(^{31}\) Solid line = midline, dashed line = medial margin of the articular facet, dotted line = medial margin of the pedicle, dot-dash line = lateral margin of the pedicle. These landmarks are used to separate the left-central (LC), subarticular (SA), foraminal (F), and extra-foraminal (EF) zones.
and Type 3 changes are more associated with low back pain and instability, while Type 2 change is more frequently seen in degenerative disc disease and is less associated with back pain. The Modic classification system has been shown to have good interrater reliability.\(^3^8\) This system grades defects in the endplate, with severity based on the area of the endplate involved. The authors demonstrated good correlation between increasing stage of endplate destruction and increasing degeneration of the associated disc.

**Facet Joints**

Degenerative changes can occur in the facet joints independent of the presence of degenerative disc disease.\(^4^1\) Findings of degenerative disease in the facet joints include joint space narrowing, subchondral erosions and cystic change, osteophyte formation, and synovial cyst formation (Figure 6). Weishaupt et al utilized these features to develop a grading system for facet disease on MRI.\(^1^3\) (Table 2). This system was found to have moderate to good agreement with CT grading of facet disease, and excellent agreement when allowing for differences of only one grade. This was the only system for MR facet joint degeneration grading recommended by Kettler et al following their literature review.\(^2^0\)

Superimposed on independent degeneration of the facet joints, loss of disc height can produce a cascade of events causing increased degeneration of the facet joints and surrounding structures. Loss of height causes abnormal contact of the superior tip of the superior articular process of the subjacent vertebra with the undersurface of the pedicle of the suprajacent vertebra. Additional abnormal contact forms between the inferior tip of the inferior articular process of the suprajacent vertebra with the posterior surface of the pars interarticularis of the subjacent vertebra. These contact points cause additional degenerative remodelling, osteophyte formation, and neocyst/synovial cyst formation secondary to altered mechanical forces. These changes can lead to thinning and fractures of the pars interarticularis, neoarthroses of the superior articular facet/pedicle, and narrowing of the neural foramina. These changes are summarized and ac-
Ligamentum Flava

Ligamentum flavum thickening is a common finding in degenerative disease of the lumbar spine, manifested by increased thickness of low T1 and T2 signal along the posterolateral spinal canal (Figure 7). Debate remains about the etiology of the thickening of the ligamentum flavum seen in degenerative spine disease. Some authors suggest that this is due to true hypertrophy of the ligament secondary to increased fibrotic change in response to adjacent inflammatory markers. Others suggest that the thickening observed is not true hypertrophy, but rather buckling of a redundant ligament secondary to loss of disc height.42,43

Structural sequelae

Spinal Stenosis

The previous section discussed the common degenerative findings affecting the ring of structures surrounding the thecal sac—at the endplates and discs anteriorly, the facet joints posterolaterally, and the ligamentum flavum posteriorly. There is conflicting evidence regarding the clinical significance of these findings in isolation and uncertainty regarding which degenerative findings are associated with low back pain.5,45-47 Indeed, a literature review by Brinjikji et al47 found that the percentage of asymptomatic 80-year-old patients with disc degeneration, disc bulging and facet hypertrophy was 96%, 84%, and 83%, respectively. This uncertainty makes clinical correlation of back pain with imaging findings extremely difficult. However, these degenerative findings in combination often cause narrowing of the spinal canal and neural foramina with resultant compression of lumbar nerve roots. This compression results in radicular symptoms such as leg pain and weakness, which can be correlated with imaging findings of nerve compression.48,49

As with disc herniation, there are multiple grading systems for nerve root compression in the spinal canal. Pfirrmann et al proposed a system to grade the effect of disc herniation on the lumbar nerve roots using three grades: contact of the nerve root, displacement of the nerve root (Figure 3A), and compression of the nerve root (Figure 4). This scale was found to have good interrater reliability and good correlation with surgical grading.50

An alternate grading system was subsequently published by van Rijn et al, which used a 5-point scale that was subsequently dichotomized to either “no root compression” (for initial categories “definitely no root compression,” “possibly no root compression” and “indeterminate”) and “root compression” (for initial categories “possibly root compression” and “definitely root compression”).51

A recent review of grading systems for lumbar disc herniations noted that while the van Rijn system was the most reliable grading system to date, the Pfirrmann system has been clinically correlated and demonstrates very good reliability at higher grades, allowing for accurate capture of symptomatic and clinically relevant lesions.52

Neural Foraminal Stenosis

Classification systems for neural foraminal narrowing are based on the degree of effacement of perineural fat within the foramen on T1-weighted sagittal images (Figure 8). Two such systems were proposed by Wildermuth et al53 and Lee et al.54 Both systems use four grades that represent normal
foramina and mild, moderate, and severe foraminal narrowing (Table 3).

The clinical correlation of the Wildermuth and Lee systems was compared by Park et al.\(^5^5\) who concluded that while both systems had similarly excellent interrater reliability, the Wildermuth grading scheme more precisely reflected clinical symptoms, particularly in patients over 50 years of age.

Conclusion

Degenerative disease of the lumbar spine is a common condition that radiologists will encounter frequently. MRI is a mainstay in the assessment of low back pain and degenerative disease of the lumbar spine. This paper has reviewed the common findings affecting the vertebral bodies, intervertebral discs, facet joints, and ligamentum flavum, as well as the combined effects of these changes on the spinal canal and neural foramina. Multiple grading systems were presented, with supporting evidence, to help increase the accuracy and consistency when reporting these findings.

References


Cystic Neck Mass

Aswin Kumar, D.O.,¹ Andrew Teh, D.O.,¹ Alysha Vartevan, D.O.²

¹Department of Radiology, Larkin Community Hospital, South Miami, FL
²Clinical Instructor in Neuroradiology, University of Arizona College of Medicine, Phoenix, AZ

Case Presentation

A 34-year-old man presented with worsening neck pain and an enlarging neck mass. Physical examination demonstrated a palpable left neck mass that was tender to palpation with overlying skin erythema; a contrast-enhanced neck CT was performed (Figure 1).

FIGURE 1. Axial (A) and reformatted sagittal (B) and coronal (C) contrast-enhanced CT images through the neck reveal a well-circumscribed, nonenhancing, low-attenuation cystic mass posterior to the left submandibular gland, lateral to the carotid sheath, and anteromedial to the sternocleidomastoid muscle.
Key Imaging Finding
Cystic neck mass

Differential Diagnosis
Cystic nodal metastasis
Abscess
Branchial cleft cyst

Discussion
Cystic neck masses are commonly encountered in imaging practices and can result from a variety of entities, both benign and malignant. The most important factors in diagnosing cystic neck masses are a thorough history and physical, as the clinical presentation and patient age often determine the differential diagnosis. Diagnostic imaging provides anatomic characterization and assessment of potential complications, and aids in management and treatment planning.

Interrogation of a neck mass may be initially performed with ultrasound, followed by contrast-enhanced CT. Neck masses can be characterized by anatomic location, attenuation, vascularity, and whether they are cystic or solid. These characteristics, in combination with demographic factors, can yield a fairly accurate differential diagnosis. Cystic neck masses can be congenital or acquired.1,2

Common congenital cystic neck masses include thyroglossal duct cysts, branchial cleft anomalies, and lymphatic malformations. Acquired cyst-like lesions include abscesses, cystic nodal metastasis, and neurogenic tumors.2 For the purpose of this article, the discussion will be limited to cystic nodal metastasis, abscesses, and branchial cleft cysts.

Cystic Nodal Metastasis
Up to 80% of cystic neck masses that present in adults over age 40 are determined to be cystic nodal metastasis.3 Necrotic lymph node metastases frequently result from primary head and neck squamous cell carcinoma or well-differentiated papillary thyroid carcinoma.3 CT demonstrates a thick enhancing wall with eccentric solid components, and possible calcifications in the setting of papillary thyroid metastases. Necrotic lymph nodes may be solitary, multiple, or conglomerate. Occasionally, the necrotic component of the lymph node is the dominant feature with imaging characteristics similar to a second branchial cleft cyst, particularly if posterior to the submandibular gland, lateral to the carotid sheath, and medial to the sternocleidomastoid muscle near the angle of the mandible. On US, the solid component of a necrotic lymph node may demonstrate intralesional color Doppler signal. Although ultrasound cannot reliably distinguish between malignant and benign cystic lesions, it may provide sonographic features that can help determine which lesions to biopsy if the clinical suspicion for metastasis is high, for example, in a patient with known papillary thyroid cancer.4

Abscess
Abscesses can occur anywhere in the neck, but most commonly occur in the parapharyngeal, parotid, submandibular, or retropharyngeal spaces.5 Abscesses in the neck do not have a gender preilection, but parapharyngeal abscesses occur most commonly in the pediatric population and in adult diabetic patients.5 On US, abscesses appear as hypoechoic or anechoic masses with thick walls, variable compressibility, and peripheral vascularity, and may demonstrate internal septations. On CT, abscesses appear as hypoattenuating masses with thick rim-enhancing walls. They may be unilocular or multilocular and may contain foci of air.2 Surrounding inflammatory changes are often seen.

Treatment of neck abscesses ranges from antibiotic therapy to emergent surgical drainage in the event of airway compromise.5

Branchial Cleft Cyst
Branchial cleft anomalies represent a variety of congenital defects that arise from aberrations in the embryonic development of the branchial apparatus, which gives rise to the ear and mesodermal structures of the head and neck.1 They do not demonstrate a gender predilection and most often present in individuals between 10 and 40 years of age.2 Branchial cleft anomalies can present as cysts, sinuses, fistulas, or a combination thereof, with cysts being the most common.5

Four branchial cleft anomalies have been described in the literature, with the second branchial cleft cyst being the most common.1-2,6 Subclassification of second branchial cleft cysts was originally described in 1929 by Bailey with four subcategories based on location.7 The type II second branchial cleft cyst is the most common, located posterior to the submandibular gland, lateral to the carotid sheath, and medial to the sternocleidomastoid muscle.2,7

On CT, branchial cleft cysts appear as nonenhancing, sharply margined, hypoattenuating masses with thin walls. Wall thickness may increase in the event of secondary infection. In the less common Bailey type III second branchial cleft cyst, a thin rim of tissue may point medially between the internal and external carotid arteries, which is referred to as a “beak sign.”1-2 On US, branchial cleft cysts commonly appear as well-circumscribed, compressible, anechoic masses with posterior acoustic enhancement and thin walls. Peripheral vascularity with mural thickening may be appreciated with superimposed infection.
Diagnosis

Second branchial cleft cyst – Bailey type II

Summary

Cystic masses of the neck are commonly encountered in imaging practices and may be benign or malignant in etiology. Patient demographics and clinical presentation in conjunction with imaging findings play a key role in developing differential diagnoses, assessing for complications, and guiding treatment and management of these lesions. In our 34-year-old patient, the imaging characteristics supported the diagnosis of a Bailey type II second branchial cleft cyst, which was confirmed upon surgical excision.

REFERENCES

Enhancing Intraventricular Atrial Mass

Anirudh Chaudhary, M.D.,¹ Alysha Vartevan, D.O.²

¹Department of Radiology, St. Joseph Hospital and Medical Center, Phoenix, AZ
²Clinical Instructor in Neuroradiology, University of Arizona College of Medicine, Phoenix, AZ

Case Presentation

A 46-year-old woman presented with a right-sided headache for 6 months, continuous during the last month. A review of systems was positive for nausea, intermittent numbness and weakness, and confusion during severe headaches. Physical examination showed no focal deficits and the patient was referred for an MRI of the brain (Figure 1).

FIGURE 1. Axial contrast-enhanced T1 MR image through lateral ventricles (A) demonstrates a large, lobulated, enhancing mass centered in the atria of the right lateral ventricle with leftward midline shift. Axial T2 fluid-attenuated inversion recovery (FLAIR) image (B) shows surrounding parenchymal edema. Axial contrast-enhanced T1 image more inferiorly (C) demonstrates entrapment and dilatation of the right lateral ventricle temporal horn with effacement of visualized basilar cisterns.
Key imaging finding

Enhancing intraventricular atrial mass

Differential Diagnosis

Intraventricular meningioma
Ependymoma
Choroid plexus tumors

Discussion

An intraventricular mass may present as an incidental finding or with signs of increased intracranial pressure, such as headache and nausea/vomiting. Intraventricular masses pose a challenge to the radiologist since the differential diagnosis is broad and many of the lesions share similar imaging characteristics. Knowledge of the patient’s age, gender, and any underlying conditions, as well as the location of the lesion, is crucial in suggesting reasonable differential considerations. Although the differential diagnosis for an enhancing intraventricular mass is quite extensive, meningiomas, ependymomas, and choroid plexus neoplasms are some of the more common lesions centered in the atria of the lateral ventricles.

Intraventricular Meningioma

Intraventricular meningiomas are relatively uncommon, accounting for 0.5% to 3.7% of all intracranial meningiomas. When located within the ventricles, the most common location is the atria or trigone of the lateral ventricle (as in the illustrated case), followed by the third, and rarely, fourth ventricles. Meningiomas are believed to arise from the arachnoid cap cells trapped in the choroid plexus. They are more common in middle-aged females with a 2:1 female-to-male ratio. Although meningiomas are uncommon in the pediatric population, up to 17% of intracranial pediatric meningiomas are intraventricular in location.

Intraventricular meningiomas are typically indolent low-grade tumors (WHO grade 1) and usually reach a large size before patients become symptomatic. However, anaplastic and atypical lesions have been reported. Metastases are uncommon, regardless of tumor grade.

On CT, meningiomas are iso- to hypodense compared to gray matter, avidly enhance, and present as sharply demarcated lobular masses. Calcification is seen more frequently with intraventricular meningiomas compared with extraventricular dural-based lesions, occurring in approximately 50% of cases. On MRI, meningiomas present as iso- to hypointense on T1 and iso- to hyperintense on T2-weighted images. They tend to be highly vascular with avid enhancement. Focal or diffuse (less common) enlargement of the ventricles may be seen depending on the size of the lesion. Periventricular edema may be seen with larger lesions, possibly due to transependymal edema or secretion of vascular endothelial growth factors, which induce edema. Reduced diffusion may be seen owing to the highly cellular nature of the meningiomas. MR spectroscopy often demonstrates elevated choline (CH); reduced N-acetylaspartate (NAA) and creatine; and variable lipid, lactate, and alanine.

Ependymoma

Ependymomas arise from ependymal cells lining the ventricular walls or presumed embryonic rests of ependymal tissue (for extraventricular lesions). They can be separated into two categories based on supratentorial or infratentorial location. Infratentorial lesions are more common, accounting for two-thirds of the cases, and occur more frequently in children. The most common location is the floor of the fourth ventricle. Ependymomas are known as “plastic” tumors because they often conform to the shape of the ventricle and “squeeze” through the foramen of Magendie (extension into cisterna magna) and Luschka (extension into cerebellopontine angles). Supratentorial ependymomas are more common in young adults with most being extraventricular in location. Supratentorial intraventricular ependymomas occur most frequently in the third, followed by lateral, ventricles.

Presenting symptoms depend on lesion location. The most common clinical symptoms include seizures, focal motor, and/or sensory impairment for supratentorial ependymomas and signs of increased intracranial pressure for infratentorial ependymomas. Infratentorial ependymomas tend to occur in infants and young children and have worse prognosis compared to supratentorial ependymomas.

On CT, ependymomas present as iso- to hypodense masses. Cystic areas and calcifications are common. On MRI, ependymomas are usually iso- to hypointense on T1 and iso- to hyperintense on T2-weighted images. There is variable signal within the cystic components due to proteinaceous content and/or hemorrhage. Variable enhancement is noted. “Blooming” may be present on susceptibility-weighted imaging secondary to calcium or blood products. Diffusion restriction is not present due to relative low cellularity. MR spectroscopy typically reveals an elevated NAA:Ch ratio.

Choroid Plexus Neoplasms

Choroid plexus neoplasms arise from choroid plexus epithelium and are one of the most common pediatric brain neoplasms in the first year of life. The most common location is the lateral ventricle (most often in the atrium), followed by the fourth and third ventricles. Approximately 5% of cases may occur at more than one location. The fourth ventricle location has a higher predilection for males (3:2 male-to-female ratio) and is more common in adults (although may occur at any age), while the
lateral ventricle location is much more common in the pediatric population.\textsuperscript{1,3} Choroid plexus papillomas (CPPs) outnumber choroid plexus carcinomas (CPCs) by 5:1.\textsuperscript{1} CPPs are WHO grade I, atypical CPPs are WHO grade II, and CPCs are WHO grade III.\textsuperscript{2} CPPs are slow growing with rare malignant degeneration. The most common symptoms include macrocrania with bulging fontanelles in infants, as well as signs of increased intracranial pressure (headache, nausea, and vomiting).

Imaging alone often cannot distinguish between CPPs and CPCs. On CT, they appear as iso- to hyperdense lobulated masses ("cauliflower" like). Calcifications are common, occurring in approximately 25\% of cases.\textsuperscript{2,3} Hydrocephalus is often present due to overproduction of cerebrospinal fluid (CSF) and/or decreased reabsorption of CSF by arachnoid granulations.\textsuperscript{1} CT angiography may demonstrate enlargement of the choroidal artery.\textsuperscript{1,3} A vascular pedicle is commonly seen. On MRI, lesions are iso- to hypointense on T1 and iso- to hyperintense on T2-weighted images. T2 fluid-attenuated inversion recovery (FLAIR) imaging often demonstrates periventricular edema. "Blooming" may be present on susceptibility-weighted imaging if calcium or hemorrhage are present. On MR spectroscopy, there is mildly elevated choline. Elevated myoinositol in CPPs may help in differentiating it from CPCs.\textsuperscript{3} The tumors are vascular and avidly enhance. Heterogeneous enhancement and invasion into the parenchyma suggests CPC over CPP.\textsuperscript{3} It is important to image the spine when CPCs are suspected to evaluate for CSF seeding.

\textbf{Diagnosis}

\textbf{Intraventricular meningioma}

\textbf{Summary}

Although the differential diagnosis for intraventricular masses is extensive, lesion location, patient age, and gender are key in narrowing the list of diagnostic considerations. When located in the atria of the lateral ventricle, meningiomas, ependymomas, and choroid plexus tumors are the most common etiologies. CT and MRI play complementary roles in characterizing these lesions, as well as in evaluating for intracranial complications.

\textbf{References}

Discitis/Osteomyelitis

A 45-year-old man presented to the emergency department with a 1-week history of low back pain and recent methamphetamine use. MRI showed abnormal fluid signal intensity within the L1-2 intervertebral disc space with endplate erosions (A), abnormal marrow edema (A) and enhancement (B) within the adjacent vertebrae, and abnormal signal and enhancement within paraspinal soft tissues with rim-enhancing psoas abscesses (arrow, C).

Spondylodiscitis is an infection of the intervertebral disc and adjacent vertebral bodies. The most common pathogens are the pyogenic organisms Staphylococcus aureus and Enterobacter species. Typically, infection spreads hematogenously but may also result from direct inoculation associated with surgery or percutaneous procedures. The most common symptom is back pain; however, symptoms are often nonspecific and variable. Fever is present in > 20% of patients. Neurological symptoms are rare and are usually the result of mass effect or nerve root inflammation.

MRI is the preferred imaging modality for diagnosis and is the most sensitive in detecting early disease. Findings include fluid signal within the disc space, irregular erosive endplate changes, and vertebral body marrow edema. The intervertebral disc is usually decreased in height in pyogenic infections and spared in nonpyogenic infections such as Mycobacterium or fungi. Paraspinal abscesses result from contiguous spread of infection into the adjacent soft tissues and appear as rim-enhancing fluid collections.

REFERENCES
Transient Global Amnesia

A 54-year-old woman without significant past medical history presented to the emergency department accompanied by her friend, who had become concerned when the patient repeatedly asked questions that had already been answered. The patient complained of a mild headache and difficulty remembering events of the previous 5 to 6 hours. There were no focal deficits on neurological examination. The patient’s symptoms resolved 10 hours after onset. MRI was performed the following day, which revealed a punctate focus of restricted diffusion in the left hippocampus (arrows, A [diffusion-weighted imaging (DWI)] and B [apparent diffusion coefficient (ADC)]) without corresponding fluid-attenuated inversion recovery (FLAIR) abnormality (C). Given the clinical presentation and imaging findings, the patient was diagnosed with transient global amnesia (TGA).

Transient global amnesia is a self-limiting process that typically lasts a few hours. Clinically, patients present with sudden onset of amnesia, which is predominantly antegrade with a lesser degree of retrograde amnesia. This may be accompanied by more generalized symptoms, such as headache or nausea, but not focal neurological deficits. Symptoms resolve within 24 hours and occur in the absence of seizure activity or head trauma.¹ Some have proposed a vascular mechanism, but the etiology of TGA remains unclear.²

The imaging findings associated with TGA consist of solitary or multiple punctate foci of restricted diffusion within the hippocampus, unilateral or bilateral. These findings are transient and may be seen after the patient’s symptoms have resolved, depending on the timing of imaging.³ Although TGA is a clinical diagnosis, imaging may help in its confirmation.

REFERENCES

Mark Bailey, M.D.,¹ Alysha Vartevan, D.O.²
¹Department of Radiology, St. Joseph’s Hospital and Medical Center, Phoenix, AZ
²Clinical Instructor in Neuroradiology, University of Arizona College of Medicine, Phoenix, AZ