NEURORADIOLOGY: SPINE IMAGING

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Case 8.1

HISTORY: A 16-year-old male presents with intermittent lower back pain. Scoliosis on physical examination

FINDINGS: Frontal radiograph (Fig. 8.1.1) shows an expanded left L2 transverse process with soft-tissue calcification adjacent to it. Subsequent axial CT image (Fig. 8.1.2) demonstrates a lytic lesion in the L2 vertebral body with a sclerotic rim, extending into the posterior elements. On T1-weighted, T2-weighted, and contrast-enhanced magnetic resonance imaging (MRI) sagittal images (Figs. 8.1.3 to 8.1.5), the heterogeneously enhancing, expansile, L2 vertebral body lesion is causing partial vertebral body collapse and spinal canal stenosis.

DIAGNOSIS: Spinal osteoblastoma

DISCUSSION: Osteoblastomas are rare, benign primary neoplasms of spine and long bones, accounting for 1% of primary bone tumors (1,2). Forty percent occur in the spine, usually the posterior...
Case 8.1 (Continued)

FIGURE 8.1.5

elements, with an additional 17% diagnosed in the sacrum. Secondary aneurysmal bone cysts are seen between 6% and 10% of osteoblastoma cases (1,3).

Spinal osteoblastomas are usually diagnosed in young adults (mean age: 20) and have a male predominance (2:1). Symptoms at presentation include back pain or neurological problems. Painful scoliosis may also result from these lesions.

Similar to osteoid osteomas, these lesions comprise osteoid and primitive woven bone, yet differ given their ability to grow ≥2 cm (average size: 3.2 cm) (1). Unlike osteoid osteomas, they may display rapid re-sorption of bony cortex, with extension into the surrounding soft tissues. Malignant osteoblastomas have also been reported—with characteristics similar to osteosarcomas (osteoblastoma-like osteosarcoma) (4,5). These malignant-type osteoblastomas demonstrate greater recurrence at prior excision sites as well as disease metastatic to the lungs (6).

Radiographic appearances of osteoblastomas vary (7). The most common pattern is a lytic lesion with multiple small calcifications and a thin sclerotic rim. These lesions may also appear completely lucent with minimal central calcifications. Pseudoma-lignant features may be present in 25% of the cases, including cortical thinning, expansion of the bone, and the presence of a soft-tissue mass (8). Given the nonspecific radiographic findings, further imaging studies are usually warranted.

CT scans allow characterization of the lesion’s nidus, matrix mineralization, and extent of involvement. With MRI, osteoblastomas have hypointense T1-weighted and hypo-to-hyper-intense T2-weighted signal, relative to bone marrow, based on the amount of ossified matrix material (7). Although limited in its visualization of subtle calcifications, MR allows better visualization of any soft-tissue edema or masses. Percutaneous biopsy will often be performed prior to definitive treatment.

Surgery (curettage or wide excision) is the primary treatment modality. Wide excision is chosen for cases with biopsy or radiographic features of the more aggressive variant. High recurrence rates (up to 24%) have been reported with curettage—significantly less with en bloc resections (1,6). As a result, a wider excision than simple curettage of the lesion may be chosen even in those without more aggressive features. Radiation therapy is controversial, especially given reports of radiation-induced sarcomas, yet utilized in fast growing or recurrent disease (9,10). Few case studies proposed a potential benefit of chemotherapy in aggressive lesions (11,12). Associated aneurysmal bone cysts are usually treated with excision and/or embolization.

Aunt Minnie’s Pearls

Although rare, spine osteoblastomas should be considered when a lucent, sclerotic, or mixed lesion is seen within the spine, the posterior elements in particular.

CT and MRI play complementary roles in its characterization.

Treatment involves curettage or wide surgical excision.
Case 8.2

HISTORY: A 37-year-old man with gradual onset of weakness of the lower extremities and a history of a recent flu-like illness

**FINDINGS:** Post-contrast, sagittal (Fig. 8.2.1), and axial (Fig. 8.2.2), T1-weighted images of the lumbar spine show contrast enhancement of the ventral roots of the cauda equina.

**DIAGNOSIS:** Guillain–Barré syndrome (GBS)

**DISCUSSION:** The diagnosis of GBS is based on clinical features, including weakness, sensory loss, pain, and hypoflexia, or areflexia in the lower extremities. Similar to poliomyelitis, the disease progresses cephalad. After days or weeks, the symptoms plateau and may regress. Some patients show brainstem involvement and may necessitate respiratory support.

Cerebrospinal fluid (CSF) analysis shows only elevated proteins. Current research shows that there are variants of GBS (13). The most common is an acute, inflammatory, demyelinating polyneuropathy (AIDP) for which pathologic examination shows lymphocyte and macrophage infiltration of peripheral nerves with segmental demyelination. Two other subtypes of GBS are acute motor and sensory axonal neuropathy (AMSAN) and acute motor axonal neuropathy (AMAN). These two are known as acute axonal forms, demonstrate marked wallerian degeneration, and have poor prognosis. AIDP is the most common form of GBS in North America and Europe (13). The incidence of AIDP is 1 to 2 cases per 100,000 persons per year, with males affected more often (14). Most cases of AIDP are sporadic, but a preceding infectious episode is common (15). AMAN and AMSAN are more common in the Far East, affect younger patients and occur more frequently during the summer months (14,16). AMAN is, in particular, associated with a recent infection with Campylobacter jejuni (15). The mainstay of treatment is supportive therapy, and other treatments such as plasma exchange, intravenous immunoglobulin, and corticosteroids have shown some efficacy in treating GBS (13). The prognosis is usually good, but 7% to 15% of patients have substantial neurologic deficits. The incidence of death is 5% (16). Overall, 80% of patients recover.

Unenhanced, T1-weighted images may show slightly high signal in the spinal nerve roots (17) but
Case 8.2 (Continued)

Aunt Minnie’s Pearls

Nerve root enhancement in a patient with an acute onset of rapidly progressive lower-extremity weakness along with appropriate laboratory findings suggest the diagnosis of GBS.

Enhancement of only the anterior rootlets of the proximal cauda equina is generally seen in GBS and in other polio-like variants.

are nearly always normal (18–20). Nerve root enhancement is seen in patients with GBS (17, 20–23). The syndrome has a predilection for the proximal nerve roots (16). Enhancement of the anterior nerve roots without enhancement of the posterior nerve roots suggests GBS, especially in patients without sensory changes (17). Occasionally, the anterior gray-matter horns within the conus medullaris may show enhancement.
Case 8.3

HISTORY: A 47-year-old woman with midthoracic pain, a chronic cough, weight loss, and night sweats

**FINDINGS:** A sagittal, pre-contrast, T1-weighted MR image (Fig. 8.3.1); corresponding post-contrast image (Fig. 8.3.2); sagittal, T2-weighted image (Fig. 8.3.3); and axial, post-contrast, T1-weighted image (Fig. 8.3.4) show that the disc space at the T9 to T10 level is mostly preserved. There is end-plate erosion and erosion of the anterior-inferior corner of T9 and of the anterior-superior corner of T10. An enhancing soft-tissue mass is recognized under the anterior longitudinal ligament. The axial image shows extension into the paravertebral regions and epidural space. There is cord compression.

**DIAGNOSIS:** Tuberculosis of the spine (i.e., Pott disease)

**DISCUSSION:** Approximately 3% to 5% of tuberculosis cases involve the skeleton in HIV-negative patients, and 60% of cases are skeletal in HIV-positive patients (24). In cases of skeletal tuberculosis, the spine is most commonly involved. Spinal tuberculosis occurs in 50% of patients with skeletal involvement (25). Untreated spinal tuberculosis can lead to extensive bone destruction and possible compression of the spinal cord. Compared with pyogenic
spondylitis, spinal tuberculosis more commonly leads to paraplegia. The relatively common involvement of the neural arches in tuberculosis contributes to cord compression (24). Spinal tuberculosis usually results from a hematogenous spread to the spine or from direct extension from a paraspinal abscess. Spinal tuberculosis more commonly involves the thoracic and lumbar regions (26,27). Vertebral body destruction begins anteriorly at the superior and inferior end plates, and sclerosis may be seen. Infection spreads under the anterior or posterior longitudinal ligaments.

More than two vertebrae are involved in >50% of patients. Bony erosion anteriorly leads to wedging of the vertebral body, and when multiple segments are involved, kyphosis occurs. Radiographs show ind distinctness of the end plates, narrowing of the disc space, and loss of vertebral body height. Erosions and sclerosis of the vertebral body may be seen. Involvement of an entire vertebral body may occur and lead to complete collapse, resulting in a vertebra plana. Large paraspinal abscesses can be seen in the psoas muscles. Occasionally, an abscess may contain a bone sequestrum. Although the focus of infection is usually the anterior portion of the vertebral bodies (27), posterior spinal tuberculosis occurs with an incidence of about 2% to 10% (28). MRI is the preferred modality for evaluating spinal tuberculosis. T1-weighted images show low signal within the vertebral body, and T2-weighted images show high signal owing to bone marrow edema. A thick rim of enhancement around paraspinal and intraosseous abscesses is typical of tuberculosis (26,29). Cold abscesses are usually disproportionately large when compared with the degree of bone involvement. Abscesses tend to be more prominent in children. When tuberculosis involves only a disc, differentiation from a pyogenic infection is not possible. Brucellosis has imaging features similar to those of tuberculosis but more often involves the lumbosacral junction.

Treatment of spinal tuberculosis is predicated on eradication of the infection and correction of any spinal deformity, particularly kyphosis. Spinal tuberculosis can be successfully treated with chemotherapy alone (30). In patients where surgery is indicated, in particular those with kyphotic deformity, anterior or posterior stabilization instrumentation can be placed. Involvement of no more than two vertebral bodies can be stabilized using anterior instrumentation, the first reported method for treating tuberculous involvement of the spine. Multisegment involvement is likely best treated with posterior instrumentation (31).

### Aunt Minnie’s Pearls

**Large paraspinal abscesses with a thick rim of enhancement accompanied by little bone destruction, particularly in children, suggest tuberculosis.**

**Isolated destruction of the posterior elements particularly in the cervical region accompanied by adenopathy or abscesses is typical of tuberculosis.**

**Fluid collections under the anterior or posterior (or both) longitudinal ligaments with involvement of only the anterior aspect of one or more vertebral bodies are suggestive of tuberculosis.**
Case 8.4

HISTORY: A 34-year-old man with AIDS and back pain (Figs. 8.4.1 to 8.4.3). Another patient is illustrated in Figures 8.4.4 and 8.4.5.

FINDINGS: A sagittal, T1-weighted MR image (Fig. 8.4.1); post-contrast, T1-weighted image (Fig. 8.4.2); and a T2-weighted image (Fig. 8.4.3) show a narrowed L2–L3 disc space with erosion of the end plates. The bone marrow of L2 and L3 has abnormal signal intensity. There is a small, rim-enhancing area of low-signal intensity posterior to the abnormal disc, resulting in narrowing of the spinal canal. On the T2-weighted images, the abnormal disc is bright. Lateral radiograph of the
Case 8.4 (Continued)

the imaging method of choice and allows early identification of the infection and delineation of paraspinal or epidural abscesses in more advanced cases (35). On MRI, the affected disc is always bright on T2-weighted images; degenerated discs are dark on T2-weighted images. The adjacent end plates enhance. Involvement of other vertebrae begins generally at the level of the canal for the basivertebral vein and is clearly seen on sagittal T2 images. Epidural abscesses usually are found at the level of the infected disc and are seen as masses that enhance peripherally and compress the thecal sac. Occasionally, at the level of an epidural abscess, the spinal cord shows high-intensity T2 signal, probably caused by venous congestion and edema. Diffusion-weighted imaging demonstrates restricted diffusion in pyogenic abscesses of the spine as it does in the brain.

Radionuclide imaging in spinal infections are mostly performed with technetium-99m-labeled di-phosphonate bone scans or gallium-67 citrate. Both of these modalities are highly sensitive for infection in the spine but lack specificity. Gallium-67 scans are preferred over indium-111–labeled white blood cells, which have better diagnostic performance outside the spine because the white blood cells have

thoracic spine (Fig. 8.4.4) demonstrates marked end plate destruction centered at a midthoracic disc level. Posterior planar image from a gallium-67 scan (Fig. 8.4.5) demonstrates a corresponding focus of intense radiotracer uptake.

**DIAGNOSIS:** Discitis with epidural abscess (first patient) and discitis alone (second patient)

**DISCUSSION:** Discitis is an inflammatory process of the intervertebral disc spaces, usually of the lumbar spine. Variable symptoms include fever, abdominal pain, limp, refusal to walk or sit up, and pain in the back, hip, or knee. In adults, infection usually begins at the vertebral body end plates and spreads to the adjacent disc. The opposite occurs in children, whose intervertebral discs are vascularized, with infection spreading to vertebral bodies from an infected disc. An immunocompromised state, advanced age, diabetes mellitus, systemic infection, and genitourinary infection or surgical manipulation predispose to osteomyelitis and discitis (32). *Staphylococcus aureus* is the most common bacterial cause of spinal osteomyelitis, but other common pathogens include *Streptococcus*, *Enterobacter*, *Escherichia coli*, tuberculosis, *Klebsiella*, and *Salmonella* (32–34). Radiographs are negative early, but bone scan results are generally positive. MRI is probably
difficulty reaching the site of infection. Improved specificity can be obtained when gallium-67 scans are interpreted with a three-phase bone scan. Infections are considered to be present when the activity on the gallium-67 scan is greater than that of the bone scan (36). Although MRI has better performance characteristics in the initial diagnostic workup, radionuclide imaging is more specific in the postsurgical and posttreatment setting (37).

Identification of the causative organism is essential to the proper treatment of discitis osteomyelitis. In the absence of positive blood cultures, percutaneous biopsy of the affected disc and end plates is needed to establish a diagnosis. Unfortunately, approximately only 50% of biopsy specimens yield a positive result. Some have advocated repeat biopsy in these situations (38). Treatment for epidural abscess is surgical evacuation.

**Aunt Minnie’s Pearls**

MRI allows early identification of infection and delineation of paraspinal or epidural abscesses. The affected disc always has high signal intensity on fluid-sensitive images.

In the postsurgical and posttreatment settings, radionuclide imaging is more specific for infection than MRI.

Percutaneous biopsy is essential to establish the identity of the infecting organism(s) in the absence of positive blood cultures.

In children, discitis is an inflammatory process centered on a lumbar intervertebral disc. Children between the ages of 6 months to 4 years are more frequently affected.
Case 8.5

**HISTORY:** A 10-year-old girl after a motor vehicle crash, during which she was wearing only a lap seat belt (Figs. 8.5.1 and 8.5.2). Figures 8.5.3 to 8.5.5 are from a different patient with the same mechanism of injury.
Case 8.5 (Continued)

**FINDINGS:** Sagittal, T1-weighted (Fig. 8.5.1), and T2-weighted (Fig. 8.5.2) images show a fracture dislocation through the T12–L1 level. There is wedge compression deformity of L1 and displacement of bone fragments into the canal, resulting in compression of the spinal cord. The T2-weighted image shows edema of the involved vertebrae and of the cord. Notice the posterior extension (Fig. 8.5.2, arrow) of the fracture. A frontal radiograph of the lumbar spine (Fig. 8.5.3) in a different patient demonstrates a transverse fracture through the vertebral body and pedicles of L2. Coronal CT reformat (Fig. 8.5.4) in this patient demonstrates the same findings. The sagittal CT reformat (Fig. 8.5.5) shows the extension of the fracture line through the posterior elements, including the pedicles.

**DIAGNOSIS:** Chance-type fracture

**DISCUSSION:** The Chance fracture, or transverse spinal fracture, is created by forceful hyperflexion of the body against a rigid object some distance anterior to the spine acting as an axis of rotation. Hyperflexion creates a distraction force, resulting in a transverse fracture through the posterior elements and the vertebral body. Chance-type fractures generally occur in the setting of a head-on motor vehicle collision in which the occupant is wearing only a lap-type seat belt or as a consequence of horseback riding accidents. Chance fractures are part of the seat belt syndrome comprising spine injuries in combination with intra-abdominal injuries. In one series of Chance fractures, 44% of patients had substantial intra-abdominal injuries (39). In children presenting with the seat belt sign, 78% had intestinal injuries (40). If an abdominal wall contusion is present in the setting of a Chance fracture, one multicenter study (41) found intra-abdominal injury in 85% of patients. Conversely, they also found that the absence of an abdominal contusion was associated with a low chance of intra-abdominal injury (14%). Chance fractures usually involve the thoracolumbar junction, but these fractures can occur anywhere in the thoracic and lumbar spine (39,42). Chance fractures are unstable, but neurological injury occurs infrequently. In a series of 19 patients, 27.7% had neurologic findings (43).
A spectrum of injuries owing to the flexion-distraction mechanism seen in the Chance fracture occurs, ranging from predominantly bony injury to only soft-tissue injury (disc and ligaments). Injuries with an injury pattern different from the classic Chance fracture are known as Chance-type or Chance equivalent fractures (45).

In children, Chance fracture equivalents involving the inferior or superior end plate also occur. The end plates act like physes and may be preferentially injured. In one series, two-thirds of cases involved the end plates, whereas only one-third demonstrated the classic fracture pattern (46).

**Aunt Minnie’s Pearl**

In any trauma patient with abdominal bruising from a seat belt, search carefully for lumbar spine fractures and intra-abdominal injuries.
Case 8.6

**HISTORY:** Young patient with left upper-extremity weakness after a motorcycle accident

**FIGURE 8.6.1**

**FIGURE 8.6.2**

**FIGURE 8.6.3**
Case 8.6 (Continued)

**FINDINGS:** Frontal view from a cervical myelogram (Fig. 8.6.1) shows a small, contrast-filled, abnormal structure in the lower cervical spine. The postmyelogram axial CT view (Fig. 8.6.2) confirms the presence of the lesion (M). In another patient with right upper-extremity weakness also after a motorcycle accident, an axial T2-weighted MR image (Fig. 8.6.3) shows absence of the right exiting nerve root with a cystic structure in its expected location.

**DIAGNOSIS:** Cervical pseudo-meningocele owing to traumatic nerve root avulsion

**DISCUSSION:** Nerve root avulsions result from severe traction on the exiting nerve roots. They are seen most commonly in the cervical spine in association with traction injuries of the arm but can occur in the lumbosacral region with lumbosacral or pelvic fractures. The typical appearance on myelography, CT myelography, or MRI is that of an absent exiting nerve root at the level of the neural foramen (47,48). The avulsed nerve root often retracts laterally, leaving a CSF-filled cavity or pseudo-meningocele in the lateral aspect of the spinal canal extending into the neural foramen and occasionally extraforaminally into the surrounding paraspinal soft tissues. Although pseudo-meningoceles typically fill with contrast introduced into the subarachnoid space, they can occasionally become walled off and manifest as extradural cystic masses. The absence of pseudo-meningoceles on myelography or CT myelography therefore does not exclude an avulsed nerve root. A T2-weighted MR image can demonstrate all pseudo-meningoceles. In rare cases, pseudo-meningocele formation may be associated with spinal cord herniation (49–51). Although many nerve root avulsions occur in association with motor vehicle accidents, they also occur during birth from excessive traction on the shoulder (52). With complete nerve root avulsion, nerve regeneration is impossible. However, through recent advancements in surgical techniques, it is possible with microsurgery to reinnervate the brachial plexus by nerve transfer from other peripheral nerves (53).

**Aunt Minnie’s Pearls**

Severe traction injuries of the arm can lead to nerve root avulsion and pseudo-meningocele formation.

Traumatic avulsions of the cervical nerve roots are more common in newborns and in young men (who are more prone to motor vehicle accidents).
Case 8.7

HISTORY: Two patients with cervical spine trauma after motor vehicle collision

FINDINGS: A lateral radiograph of the cervical spine in one patient (Fig. 8.7.1) demonstrates a bilaminar fracture of the axis with 3-mm anterolisthesis of C2 on C3. In the other patient, lateral radiograph of the cervical spine (Fig. 8.7.2) demonstrates posterior angulation of C2 in relation to C3 with anterolisthesis of 2 mm of C2 on C3. In this same patient, an axial CT image (Fig. 8.7.3) demonstrates bilateral pedicle fractures with involvement of the left transverse foramen. A sagittal T2-weighted MR image (Fig. 8.7.4) demonstrates edema of the spinal cord at the level of the body of C2.

DIAGNOSIS: Traumatic spondylolisthesis of the axis (hangman’s fracture)

DISCUSSION: Traumatic spondylolisthesis of the axis (TSA) is a bilateral fracture of the posterior elements of the C2 vertebra that comprise the neural arch. The degree of instability of the injury is related...
to the degree of disruption of the anterior longitudinal ligament and the C2/C3 disc.

Termed a “hangman’s fracture” by Schneider et al. (54), owing to the similarity of the fracture pattern seen in judicial hangings, the mechanism and expected injuries in TSA are quite different from that seen in judicial hangings. Today, motor vehicle accidents, falls, and diving accidents are the most common situations resulting in this type of injury. The most common mechanism is hyperextension with axial compression. Hyperflexion also plays a role, particularly in the more severe injuries. In contrast, the mechanism in judicial hangings using a submental knot is hyperextension with distraction.

A number of radiographic classification systems based on the lateral cervical spine radiograph have been developed to predict degree of instability. The presumption is that a nondisplaced fracture indicates that the anterior longitudinal ligament and disc are intact and that as displacement and angulation increase, the degree of disruption of these structures increases thereby leading to increasing instability. In the two most widely used systems, those of Effendi et al. (55) and Levine and Edwards (56), fractures are graded from 1 to 3 with grade 3 representing the most unstable injury. In both, a type 3 TSA indicates bilateral jumped facets. As positioning after trauma can artifactually reduce the grade of injury, clinical judgment should also be used to determine the degree of instability.

The role of imaging is to determine the type of injury and the presence of complications (57). The lateral cervical spine radiograph is usually sufficient to make the diagnosis of a TSA. The primary role of computed tomography is to evaluate for potential vertebral artery injury and to help identify additional fractures. MRI is used to identify or confirm injury to the spinal cord depending on the clinical situation. Occasionally, lateral flexion and extension radiographs may be of use in determining the stability of the fracture.

Treatment is based on the determination of stability as determined by the classification system in use and the clinical picture (58). Those fractures deemed stable are treated with external fixation: rigid collar or halo vest. Those fractures deemed unstable are treated with anterior cervical fusion.

**Aunt Minnie’s Pearls**

Traumatic spondylolisthesis of the axis is commonly the result of hyperextension injuries with axial compression. Flexion plays more of a role in severe injuries.

The mechanism in judicial hangings with a submental knot is hyperextension with distraction.

Potential complications include vertebral artery injury and spinal cord injury.
Case 8.8

**HISTORY:** A 35-year-old man with a 3-month history of low back pain and saddle-like distribution anesthesia (Figs. 8.8.1 and 8.8.2). Figures 8.8.3 to 8.8.5 show an example of another subtype of this diagnosis. (Case courtesy of Dr. Aquilla Turk, III)
Case 8.8 (Continued)

**FINDINGS:** Sagittal, T2-weighted (Fig. 8.8.1) and post-contrast, T1-weighted (Fig. 8.8.2) images show an enhancing mass in the region of the conus medullaris with some enhancement of the distal nerve roots. In a different patient, a sagittal T1-weighted post-contrast (Fig. 8.8.3) image shows an enhancing lesion within the distal cord also at the level of the conus medullaris. Axial T1-weighted pre-contrast (Fig. 8.8.4) and post-contrast (Fig. 8.8.5) images demonstrate this lesion to be intramedullary.

**DIAGNOSIS:** Spinal cord ependymoma, myxopapillary ependymoma

**DISCUSSION:** Ependymomas are the most common intramedullary tumors in adults, followed by astrocytomas and hemangioblastomas (59). In children, astrocytomas are more common than ependymomas (60). Ependymomas arise from ependymal cells lining the central spinal canal and usually cause symmetric expansion of the cord. Eighty percent of cord ependymomas have associated cysts (61). These are considered to be reactive cysts and are not lined by neoplastic cells but by gliosis. Sixty-seven percent of intramedullary ependymomas occur in the cervical region (62), with extension of the solid portion of the tumor for an average of four vertebral body segments (61,63,64). Unlike astrocytomas that demonstrate infiltrative pathology, ependymomas are generally well-circumscribed lesions, making surgical resection possible (65,66). The myxopapillary variant of ependymoma generally involves the filum terminale (65). Clinically, these patients present with nonspecific back pain or focal neurologic symptoms. Ependymomas are isointense to hypointense on unenhanced, T1-weighted images and enhance after gadolinium administration (63,64). Sharply marginated enhancement is typical of ependymomas, and this correlates surgically with the margins of the tumor (61). Ependymomas can hemorrhage, and if bleeding occurs along the periphery of the tumor, a hypointense hemosiderin ring will be seen on T2-weighted images. Ependymomas may show blood-fluid levels in their cysts. It has been reported that cervical ependymomas are more likely to hemorrhage (63). Myxopapillary ependymomas have a typical sausage shape, are located in the proximal filum terminale, do not bleed, may extend into the nerve roots of the cauda equina or the conus medullaris, and show enhancement.

**Aunt Minnie’s Pearls**

A well-enhanced, sharply marginated, centrally located lesion, in the cervical spine in particular, is most likely an ependymoma.

A spinal cord tumor containing a rim of chronic blood products or intratumoral blood is most often an ependymoma.

A sausage-shaped, enhancing tumor in the filum terminale is nearly always an ependymoma. The differential diagnosis includes astrocytoma, paraganglioma, and metastasis.
**Case 8.9**

**HISTORY:** A 26-year-old female presents with recurrent lower back pain, extending into bilateral thighs.

**FINDINGS:** Sagittal T1-weighted, T2-weighted, and contrast-enhanced images show two heterogeneous, low signal on T1-weighted images, predominantly high signal on T2-weighted images, enhancing intradural round lesions at the level of L4–L5 and the conus with a thin filum terminale connecting them (Figs. 8.9.1 to 8.9.3). Additional axial T2-weighted image of the sacrum (Fig. 8.9.4) demonstrates a subcutaneous dermal sinus tract.

**DIAGNOSIS:** Spinal dermoid

**DISCUSSION:** Spinal dermoids are rare, slow-growing dysontogenic tumors that arise from ectopic ectoderm and mesoderm embryonic rests inclusion within the spinal canal at the time of neural tube closure (third–fifth weeks of fetal life) (67). Accounting for approximately 1% to 2% of intraspinal tumors, they occur predominantly in
Case 8.9 (Continued)

the lumbosacral region (60%), followed by the thoracic region (10%)—usually residing in an intradural, extramedullary location (68,69). Approximately 20% of spinal dermoids are associated with a dermal sinus tract (67).

With a slight increased predominance in males, spinal dermoids are usually diagnosed between second and third decades (70). Symptoms result from the lesion’s location, owing to its compressive effect on adjacent structures. The most common presenting symptom is lower back or sciatic pain. Spinal dermoids and tethered cord have been associated with progressive lower neurologic and bladder dysfunction—including postoperative cases of myelomeningocele repair (71). Excluding spina bifida repair, acquired dermoid cysts have also occurred with other procedures (e.g., spinal surgery or lumbar puncture)—likely a result from implantation of epidermal tissue into the subdural space. Rupture of these lesions may cause seizures, chemical (aseptic) meningitis, or arachnoiditis—owing to the spread of lipid droplets throughout the subarachnoid spaces (72).

The combination of fluid, fat, solid tissue, and calcium is diagnostic of a dermoid tumor (67,73). Based on its composition, these lesions have a heterogeneous appearance on MRI. High signal on T1-weighted images correlates with fatty secretions of sebaceous glands or cholesterol from degenerating epithelial cells. However, the intensity of the dermoid cystic components varies, usually hypointense on T1-weighted and iso- to hyper-intense on T2-weighted sequences, relative to the spinal cord. Noncontrasted CT easily confirms the presence of fat and calcifications within the lesion. Fat-fluid levels are occasionally seen on CT. In addition to spina bifida, vertebral anomalies associated with spinal dermoids, radiographs may also demonstrate erosion (scalloping) of the posterior vertebral body walls and widened interpedicular widths (74).

There is presentation and imaging overlap between spinal epidermoids and dermoids. Neuroenteric or arachnoid cysts should also be considered in the differential; however, they are more easily differentiated from dermoids.

Surgery is the primary treatment modality, with good outcomes upon excision. Malignant transformation of spinal dermoids is extremely rare (75). Steroids may be beneficial for treatment of meningitis-type symptoms.

Aunt Minnie’s Pearls

Although rare, spinal dermoids should be considered in spina bifida patients or young adults with intraspinal masses.

CT and MRI imaging play complementary roles in its characterization, particularly MRI given its superior visualization of other spinal structures.
Case 8.10

HISTORY: A 38-year-old man with progressive weakness of the legs

FIGURE 8.10.1

FIGURE 8.10.2

FIGURE 8.10.3

FIGURE 8.10.4
Case 8.10 (Continued)

**FINDINGS:** Axial (Fig. 8.10.1) and sagittal T2-weighted (Fig. 8.10.2) images of the spine show abnormal increased T2-weighted signal associated with the conus medullaris consistent with venous congestion. In addition, there are multiple prominent intradural, extramedullary flow voids. A 3D reconstruction from MR angiography of the spine (Fig. 8.10.3) shows the anterior spinal artery arising at the L2 level and extends superiorly to the level of T12. The artery then feeds an arteriovenous malformation. Multiple irregular draining veins are also seen extending superiorly and inferiorly. These findings were confirmed at catheter angiography (Fig. 8.10.4) (Case courtesy of Dr. Vittoria Spampinato).

**DIAGNOSIS:** Spinal arteriovenous malformation

**DISCUSSION:** Vascular malformations of the spinal cord have been classified according to the anatomic location and characteristics of the malformation nidus (shunt). Intramedullary, pial, and mixed lesions occur. In one classification scheme, type I malformations are glomus-like, with a small, compact nidus and a few feeding vessels. Type II malformations are juvenile and have a larger nidus with multiple feeders from the anterior and posterior spinal arteries. Type III malformations, which can be seen in association with Cobb syndrome, are metameric, with an extensive lesion extending outside the cord and involving the meninges, epidural space, and the adjacent vertebral body. These lesions are congenital. A similar lesion is the dural arteriovenous fistula (dAVF) with a direct communication between arteries and draining veins and no intervening nidus. The dAVFs are the most common spinal vascular malformations, found in a slightly older patient population and are more likely acquired lesions (76,77).

Patients with spinal arteriovenous malformation (AVMs) present with progressive neurologic deficits. Acute deficits may occur after hemorrhage of the lesion, leading to hematomyelia or subarachnoid hemorrhage, which has a high mortality rate. Additional pathology includes venous congestion, mass effect, and “steal phenomena” (76). Spinal AVMs may also undergo spontaneous thrombosis. Myelography usually demonstrates enlarged, tortuous vessels on the surface of the spinal cord associated with these vascular malformations. On MRI, these vessels appear as serpentine flow voids (78). Abnormal cord signal occurs if there has been intramedullary hemorrhage or ischemia or infarction from chronic venous congestion resulting in edema. All symptomatic arteriovenous dural fistulas result in high-intensity T2 signal in the spinal cord. Although dynamic gadolinium-enhanced MRA has been shown to be useful in the evaluation of spinal AVMs (79–81), spinal arteriography remains the gold standard. Therapy comprises intravascular embolization and surgery (82,83).

**Aunt Minnie’s Pearls**

In the presence of hematomyelia, a spinal vascular malformation should be sought.

With AVFs, MRI shows high-intensity T2 signal in the cord and enlarged blood vessels (draining veins) on the surface of the cord.

With AVMs, MRI shows a nidus containing flow voids inside the cord.
**Case 8.11**

**HISTORY:** Patient on long-term steroid therapy presenting with lower thoracic back pain

**FINDINGS:** The lateral radiograph (Fig. 8.11.1) shows a wedge compression fracture of L1. The vertebral body contains a gas-filled cleft.

**DIAGNOSIS:** Vertebral osteonecrosis (i.e., Kümmell disease)

**DISCUSSION:** Kümmell disease refers to osteonecrosis of a collapsed vertebral body. The most common cause of vertebral body osteonecrosis is posttraumatic, typically following an osteoporotic vertebral body compression fracture. However, other causes may lead to osteonecrosis first prior to vertebral body collapse, including neurologic, vasomotor, and nutritional deficiencies as well as administration of exogenous steroids.

Theoretically, an episode of trauma leads to ischemia and delayed vertebral body collapse. An association between vertebral body ischemia and the presence of gas (nitrogen) within the vertebral body, also known as an intravertebral vacuum cleft, is characteristic of this entity. A radiographic and histologic study of patient's undergoing kyphoplasty for vertebral compression fractures found that the intravertebral vacuum cleft has a sensitivity of 85% and a specificity of 99% for osteonecrosis (84). In an analysis of 1,272 patients, near equally divided among patients with osteoporotic vertebral fractures, spinal infections, spinal metastases, and multiple myeloma, Feng et al., demonstrated that the vacuum phenomenon was seen in 18.9% in patients with osteoporotic fractures, 6.4% in patients with multiple myeloma, and in only one patient with tuberculous spondylitis (85). With the exception of the patient with the spinal infection, the morphology of the vacuum phenomenon was that of a linear cleft. In the case of the infection, the intravertebral gas had a more bubble-like and diffused appearance. It is important to recognize the linear cleft of intravertebral gas because this finding effectively excludes metastatic disease or infectious involvement of the vertebral body (86,87). Radiographically, the cleft appears as a radiolucent transverse band in the centrum of the collapsed vertebra or adjacent to one of its end plates. The cleft may increase in size with spinal extension and be inhomogeneous on CT.
scans. On MRI, the intravertebral cleft appears as a linear signal void on T1-weighted and T2-weighted sequences. Prolonged positioning of the patient in a supine position during MRI may lead to displacement of the gas within the cleft by fluid, with high-intensity T2 signal appearing on delayed sequences. When seen centrally in the vertebrae, this change appears to be specific for osteonecrosis also (88).

**Aunt Minnie’s Pearls**

A linear cleft of gas seen in the centrum of a collapsed vertebral body is diagnostic of vertebral osteonecrosis. The cleft of gas effectively excludes metastasis and infection, although, it is seldom seen in multiple myeloma.
Case 8.12

**HISTORY:** A 71-year-old male presented with intermittent, yet worsening lower back and buttock pain. Physical examination demonstrated “fullness” in skin in the coccygeal region.

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**FINDINGS:** Lateral lumbar radiograph (Fig. 8.12.1) shows ill-defined soft tissue opacity overlying the distal coccyx with tiny calcifications in it. A coronal CT image of the pelvis (Fig. 8.12.2) demonstrates a partially calcified soft-tissue mass slightly eroding into the adjacent coccygeal bone. Sagittal T1-weighted, T2-weighted, and contrast-enhanced images show predominantly low T1 and high T2 signal intensity heterogeneously enhancing lobulated mass encompassing the coccyx (Figs. 8.12.3 to 8.12.5).
Case 8.12 (Continued)

**DIAGNOSIS:** Sacrococcygeal chordoma

**DISCUSSION:** Chordomas are rare, accounting for ~2% to 4% of bone tumors and 20% of primary spine tumors (89,90). Arising from notochordal remnants, these slow-growing lesions develop along the axial skeleton—usually involving the sacrum and coccyx (50% of reported cases), skull base (35%), or mobile spine (15%, most commonly the C2 vertebral body) (2).

Rare in children, these lesions are typically diagnosed in the sacrococcygeal region between 40 and 60 years of age—presenting 10 to 20 years earlier in cases that involve the skull base. Chordomas are more common in males (2:1 ratio). Symptoms at the time of presentation greatly depend on tumor location. They usually present with back pain owing to bone destruction and compression of adjacent structures. Additional symptoms may include constipation, peri-rectal or lower extremity paresthesia, neurogenic bladder, or fecal incontinence.

Chordomas in the sacrococcygeal region classically present with lytic destruction of several sacral vertebrae, combined with a soft-tissue mass anterior to the sacrum. Surprisingly, the intervertebral discs and posterior elements are usually spared. The reported radiographic findings of bone expansion, rarefaction, calcification, and trabeculation associated with chordomas (91) are nonspecific and other entities—including chondrosarcoma, lymphoma, plasmacytoma, teratoma, and metastases—should be considered during diagnostic workup.

Radiographs consistently underestimate a tumor’s soft-tissue component. Therefore, CT and MRI are recommended for better delineation of its extra-vertebral extent. On CT, chordomas appear as centrally located, well-circumscribed, expansive, enhancing soft-tissue masses with intratumoral calcifications or bone fragments (2). Up to 50% of chordoma have intratumoral calcifications (92).

Given potential extension into the epidural space or adjacent structures, MRI is frequently obtained owing to its greater soft tissue contrast properties. On MRI, sacrococcygeal chordomas are lobulated tumors, with low-to-intermediate T1-weighted and high T2-weighted signal intensity on pre-contrast sequences—exhibiting a heterogeneous honeycomb enhancement pattern on post-contrast T1-weighted images (2,92,93). Chordomas may demonstrate high T1-weighted intensity owing to high protein content. Blooming artifact on gradient echo sequences results from intralesional hemorrhage.

The primary treatment for chordoma is surgery. En-bloc surgical resection may be followed by radiation, in cases of incomplete removal. For nonsurgical candidates (with large invasive tumors), radiotherapy may suppress or slow down tumor growth. Despite slow growth, chordomas

![FIGURE 8.12.5](image-url)
have a poor long-term prognosis, which varies depending on degree of resection (94,95). The overall median survival time is 7 to 9 years—less in patients with metastatic disease at time of diagnosis. The instances of metastatic disease range between 30% and 43% in the literature (96,97). The most common sites of metastatic disease are adjacent pelvic lymph nodes, the lungs, or bones—usually treated with either surgery or radiotherapy. Chemotherapy may have a beneficial role in these patients, based on active ongoing multicenter clinical trials (98).

**Aunt Minnie’s Pearls**

- Consider chordomas for midline sacrococcygeal masses, particularly adults.
- Although usually visualized on radiographs as a destructive lesion, CT and MRI imaging are essential for adequate evaluation of the soft tissue component.
- Although slow growing, chordomas have a high recurrence rate and intermediate survival rate despite treatment.
Case 8.13

HISTORY: Elderly Japanese man with progressive myelopathy (Figs. 8.13.1 and 8.13.2). Another case is illustrated in Figures 8.13.3 and 8.13.4.
Case 8.13 (Continued)

**FINDINGS:** A sagittal, T2-weighted (Fig. 8.13.1) image shows a thick, dark, linear structure (arrows) posterior to the C2–C6 vertebrae. This results in narrowing of the spinal canal and compression of the cord, which also contains abnormally high signal intensity. On an axial, T2-weighted image (Fig. 8.13.2), the thickened ligament (arrow) is seen narrowing the canal and compressing the cord. In the second patient, axial CT (Fig. 8.13.3) and T2-weighted (Fig. 8.13.4) images demonstrate a central ossified structure projecting posteriorly from the vertebral body resulting in effacement of the ventral thecal sac and flattening of the cord.

**DIAGNOSIS:** Ossification of the posterior longitudinal ligament in the setting of diffuse idiopathic skeletal hyperostosis (DISH) in the first patient and in isolation in the second patient

**DISCUSSION:** DISH, or Forestier disease, is a bone-forming diathesis (enthesopathy) affecting 10% to 20% of the elderly population. It is not considered an arthropathy because the articular cartilage, adjacent bone marrow, and synovium are not affected. Although DISH has been called senile ankylosing spondylitis, it has no association with HLA-B27 antigen and is easily differentiated from ankylosing spondylitis radiographically by thicker, more disorganized paravertebral excrescences; lucency between the excrescences and the vertebral bodies; and no involvement of the posterior elements (99,100).

The spine-related findings of DISH are normal mineralization, flowing ossification of the ligaments of at least four contiguous vertebral bodies, and preservation of the disc or joint space. The middle-to-lower thoracic spine is most commonly affected, although the lumbar and cervical spine may also be involved. Other prominent findings of DISH include ossification at tendinous and ligamentous insertions without intrinsic joint abnormalities and the absence of apophyseal joint ankylosis or sacroiliac joint disease.

The posterior longitudinal ligament may be calcified or ossified. In Western countries, ossification of the posterior longitudinal ligament is commonly associated with DISH, whereas in Eastern countries, it may occur in an isolated form. The most common symptom is that of a myelopathy generally resulting from narrowing of the cervical canal to <10 mm in its anteroposterior dimension. Surgery is the mainstay of management for symptomatic disease. Disease spanning just one or two segments can be treated via an anterior approach with corpectomy, whereas longer segment disease requires decompressive laminectomy (101).

**Aunt Minnie’s Pearls**

Ossification at four contiguous vertebral body levels + preservation of the disc space + no ankylosis in an elderly patient = DISH.

DISH is a common disease found in patients with ossification of the cervical posterior longitudinal ligament.
Case 8.14

HISTORY: A 32-year-old man with chronic low back pain and right footdrop (Figs. 8.14.1 and 8.14.2). Figure 8.14.3 is another patient with the same pathology.
Case 8.14 (Continued)

**FINDINGS:** A sagittal, T2-weighted image (Fig. 8.14.1) shows a dark epidural abnormality at the L5–S1 disc space that projects posteriorly and narrows the spinal canal. An axial, T2-weighted image (Fig. 8.14.2) of the same patient shows that the abnormality is contiguous with the disc. The degree of narrowing of the canal is better appreciated in this image. In a different patient with the same type of pathology, an axial, T1-weighted image (Fig. 8.14.3) shows a hypointense lesion in the right lateral recess of S1.

**DIAGNOSIS:** Central disc herniation (first patient) and sequestered herniated disc fragment (second patient)

**DISCUSSION:** Standard nomenclature for describing disc pathology was introduced in 2001 and should be used in imaging reports (102). Extension of disc material beyond the vertebral end plates for >50% of the disc circumference is defined as a bulge. A disc bulge is usually circumferential and symmetric but can be asymmetric as commonly seen in scoliosis. It may result from generalized relaxation of the annulus fibrosus and radial tears and is very common at the L5–S1 level. A localized extension of disc material beyond the limits of the intervertebral disc space is called disc herniation, which can be focal or broad-based. If <25% of the disc circumference is involved, the herniation is considered focal, but if 25% to 50% of the disc circumference is involved, the herniation is broad-based. A disc herniation may be considered a disc protrusion if it has a wide margin of attachment to the underlying disc. If in any plane, including the sagittal plane, the size of the disc fragment is wider than its base, then it is considered an extrusion. The location of the herniation is described as central, right or left central, foraminal, or extraforaminal. A herniation may become separated from the original disc, becoming a sequestration or free fragment. A free disc fragment extends through a tear in the posterior longitudinal ligament and may remain localized or migrate superiorly or inferiorly within the ventral epidural space. In addition to disc herniations, other degenerative changes of the spine include osteophytes, facet joint disease, and thickened ligamentum flavum. These can also result in compressive sequelae and there are systematic methods for reporting spinal and neuroforaminal stenosis (103).

An MRI study of 60 asymptomatic volunteers between the ages of 20 and 50 years found a disc bulge in 62% of subjects and a disc protrusion in 67% of subjects. Disc extrusions and free fragments with nerve root compression were found in 18% of the subjects and may be more predictive of low back pain (104). Similar findings were seen in a study of 98 asymptomatic subjects: 52% of the subjects had a bulge at least one level, 27% had a protrusion, and 1% had an extrusion (105). Treatment is usually initially conservative; however, there is current debate in the literature concerning the adequacy of randomized, controlled trials in assessing the efficacy of treatment approaches (106). Failure to relieve symptoms or recurrent symptoms after surgery is referred to as failed back syndrome, and its causes include recurrent disc herniation, scar formation, arachnoiditis, and surgery at the wrong level. Postoperative MRI is usually equivocal initially, but it is useful in differentiating recurrent disc herniation from epidural scar 6 to 8 weeks after surgery. After contrast administration, peripheral enhancement is consistent with herniation because the disc material is not perfused; however, granulation tissue and scar demonstrate prominent enhancement.

**Aunt Minnie’s Pearls**

Disc bulges are diffuse and result in stenosis when accompanied by osteophytes, degenerative facet joint disease, and thickened ligamentum flavum.

Disc herniations are focal or broad-based, and many patients respond to conservative treatment.

Postoperative recurrent or residual disc herniations show no significant or peripheral contrast enhancement, whereas scar and granulation tissue show prominent enhancement.
**Case 8.15**

**HISTORY:** A 40-year-old man with neck pain (Figs. 8.15.1 to 8.15.3). Two more patients with the same diagnosis, one with low back pain (Fig 8.15.4) and the other with cauda equina syndrome (Figs. 8.15.5 and 8.15.6).

![Figure 8.15.1](image1)

![Figure 8.15.2](image2)

![Figure 8.15.3](image3)
Case 8.15 (Continued)

FINDINGS: Lateral cervical spine radiograph (Fig. 8.15.1) demonstrates delicate syndesmophytes bridging the cervical vertebral bodies and fusion of the facet joints. A fracture through the C6/C7 level is present. The sagittal CT reformat (Fig. 8.15.2) illustrates the gracile syndesmophytes anteriorly and a fracture through the C6 spinous process and the superior body of C7. The coronal CT reformat (Fig. 8.15.3) demonstrates the multilevel fusion of the facets and the fracture through the C6/C7 level. Frontal lumbar spine radiograph (Fig. 8.15.4, left) and select coronal reformat from an abdominal CT (Fig. 8.15.4, right) in the second patient demonstrate thin syndesmophytes spanning the lateral aspects of the lumbar vertebral bodies, best seen at the L2/L3 level. In the third patient, axial CT (Fig. 8.15.5)
and T1-weighted MR images (Fig. 8.15.6) show CSF-filled diverticula eroding the posterior elements.

**DIAGNOSIS:** Ankylosing spondylitis

**DISCUSSION:** Ankylosing spondylitis is a seronegative spondyloarthropathy associated with HLA-B27. This entity affects men 3 to 10 times more often than women, with an age of onset between 15 and 35 years (107). Imaging findings include bilateral, symmetric sacroiliac joint erosions and sclerosis progressing to complete fusion late in the disease. Fine, marginal syndesmophytes (i.e., ossification of the outer fibers of the annulus fibrosus) ascend the spine, giving the appearance of a “bamboo” spine. Typically, spinal changes follow sacroiliac disease (107). Erosions occur at the corners of vertebral bodies, leading to “squared vertebral bodies.” The presence of destructive changes at the superior and inferior portions of the vertebral bodies is known as a Romanus lesion and is an early sign of ankylosing spondylitis. The erosions later become sclerotic, giving the appearance of “shiny corners” (108).

The spine in ankylosing spondylitis patients is prone to injury. Minor stress or trauma may result in fractures through the intervertebral disc or through the vertebral bodies adjacent to the end plates and can extend through the posterior elements (109,110). Delayed identification and treatment of these fractures, with continued motion, leads to the formation of pseudoarthrosis at the fracture site. If the fracture occurs through the cervical spine, instability with cord compression may result, leading to severe neurologic complications.

A 10-year retrospective review of spinal cord injury in patient’s with ankylosing spondylitis by Thumbikat et al. (111) noted that most of these patients were able to walk after the initial injury and then subsequently declined neurologically. In particular, overcorrection of the exaggerated kyphosis that develops in ankylosing spondylitis was identified as the cause of subsequent neurologic decline in 40%. In one patient, this occurred during positioning for an MR scan. Another cause for delayed neurologic decline was the presence of a spinal epidural hematoma.

In the setting of trauma, initial radiographs may be difficult to interpret or nondiagnostic owing to severe osteopenia and obscuration of the cervicothoracic junction. As a result, one can have a low threshold for performing CT (112) and possibly MRI. Late in the disease, additional MR findings may include erosion of the posterior vertebral elements, dural diverticula, and arachnoiditis (i.e., nerve root clumping and cord tethering). A sterile discitis known as an Anderson lesion can occur. The cause of these late findings is unknown, but decreased elasticity of the meninges combined with ligamentous inflammation has been proposed as leading to chronic arachnoiditis and diverticula formation (113–115).

**Aunt Minnie’s Pearls**

Fine, confluent syndesmophytes are characteristic of ankylosing spondylitis.

In the setting of trauma, one can have a low threshold for CT and possible MRI of the spine.

Causes of subsequent neurological deterioration after the initial injury include iatrogenic overcorrection of the patient’s normal hyperkyphosis (including positioning for imaging exams) and spinal epidural hematoma.

On MRI, chronic cases of ankylosing spondylitis can have prominent dural diverticula, clumping of nerve roots, and tethering of the spinal cord.
**Case 8.16**

**HISTORY:** A 28-year-old male presented with neck pain, persisting after a recent motor vehicle collision. (This case courtesy of Dr. Mauricio Castillo)

**FIGURE 8.16.1**

**FIGURE 8.16.2**

**FINDINGS:** Right posterior oblique cervical spine radiograph (Fig. 8.16.1) shows a widened left neural foramen and a nonvisualized left pedicle with well-corticated, smooth appearance of adjacent osseous structures. An axial T2-weighted image (Fig. 8.16.2) shows absence of the left pedicle, enlarged left neural foramen, and hypertrophy of the contralateral articulating facets.

**DIAGNOSIS:** Congenital absence of the pedicle (pedicle agenesis)

**DISCUSSION:** Absence of a cervical spine pedicle is a rare congenital anomaly (116,117) that results from early developmental failure in formation or subsequent ossification of a vertebral chondrification center during the fourth to eighth weeks of gestation. Recognition of this entity is important as misdiagnosis may result in unnecessary invasive interventions (116–118). In one study, 18% of patients with pedicle agenesis received inappropriate therapy (halo immobilization, surgical exploration, or radiation) owing to an initial misdiagnosis of trauma or neoplasm (117).

The most frequent region of pedicle agenesis is the cervical spine, followed by the lumbar and thoracic spine. The C6 vertebra is the most commonly reported cervical site, with L4 being the most site of occurrence in the lower spine (119). In symptomatic cases, presenting symptoms are usually neck pain radiating to the back and shoulders, or upper extremity sensory deficits.

Radiographic signs of pedicle agenesis on the AP radiograph include loss of the pedicle contour, apparent separation of the ipsilateral transverse process from the vertebral body, and deviation of the spinous process away from the affected side. On the oblique radiograph (Fig. 8.16.1), the left neuroforamen appears enlarged owing to the absent pedicle. The presence of compensatory hypertrophy and sclerosis of the contralateral pedicle supports a diagnosis of unilateral pedicle agenesis. Additional articular pillar dysplasia and spinal segmentation anomalies may also be present (117,118,120).

In cases of a radiographically nonvisualized unilateral pedicle, one should consider other benign (aneurysmal bone cyst, osteoblastoma, or osteoid osteoma) and malignant (metastases or myeloma)
Case 8.16 (Continued)

lesions. In cases of minimal compensatory hypertrophy in the contralateral pedicle, metastatic disease will often be the primary consideration until it is excluded with further workup. Additional considerations for an absent pedicle include erosion from a spinal canal or bone tumor, tortuous vessel, or extradural cyst (meningocele).

Despite pedicle agenesis, the spine is considered stable as the anterior column and ipsilateral posterior elements are intact. If there is uncertainty regarding the diagnosis of pedicle agenesis, computed tomography, with or without 3D reconstructions, should be performed. In cases of metastatic disease or primary neoplasm of bone, a nuclear bone scan will demonstrate intense uptake in the region of the interest (121,122). High radionuclide uptake on bone scan in the contralateral pedicle would be seen in pedicle agenesis, owing to stress hypertrophy (119).

Treatment of pedicle agenesis is conservative. Complications are rare, usually sequelae of nerve compression.

Aunt Minnie’s Pearls

Pedicle agenesis is a rare, congenital anomaly, which should be considered in the presence of contralateral compensatory pedicle hypertrophy.

Appropriate knowledge and awareness of such an anomaly can prevent inappropriate management.
**Case 8.17**

**HISTORY:** A 2-month-old girl with a subcutaneous mass on her back

**FINDINGS:** A sagittal, T2-weighted image (Fig. 8.17.1); axial, T1-weighted image (Fig. 8.17.2); and axial, T2-weighted image (Fig. 8.17.3) show a fluid-filled meningocele at the posterior sacrum. The sac contains portions of unfolded spinal cord and nerve roots. The spinal cord is dysplastic and tethered. There is a posterior spinal bifida, and the mass is covered by subcutaneous fat.

**DIAGNOSIS:** Lipomyelomeningocele

**DISCUSSION:** Spinal dysraphisms can be divided into open and closed types, depending on whether neural tissue is exposed or covered by skin (123–125). For example, myelomeningoceles and myeloceles are open dysraphisms. Lipomyelomeningoceles and lipomyeloceles are examples of closed spinal defects.
Case 8.17 (Continued)

These probably arise from premature disjunction of the neuroectoderm from cutaneous ectoderm, with entrapped ectodermal elements differentiating into lipomatous tissue. Lipomyelomeningoceles account for 20% of skin-covered lumbosacral masses and up to one-half of occult spinal dysraphisms (126). The spinal cord is dorsally contiguous, with a lipomatous mass that extends to the subcutaneous tissues. Nerve roots do not course through the fatty mass, which is dorsal to the cord and extradural in location. In lipomyelomeningoceles, the dysplastic and unfolded spinal cord, called the placode, lies superficial to the level of the bony defect owing to expansion of the subarachnoid space ventrally (127). In a lipomyelocele, the placode lies inside of the spinal canal and therefore deep to the bony defect. A tethered cord is almost always present, and up to 25% of patients have associated hydrosyringomyelia (128). Skin tags or dimples, dermal sinuses, and other cutaneous defects are found in up to 50% of patients. Lipomyelomeningoceles should be differentiated from the less common intraspinal lipomas, because the clinical outcome after surgery is better for intraspinal lipomas (129,130). This disorder is not associated with the Chiari II malformation, a congenital hind brain malformation associated with open spinal defects.

**Aunt Minnie’s Pearls**

A lipomyelomeningocele is a closed spinal dysraphism in which the distal spinal cord is contiguous dorsally with a large fatty mass.

A tethered cord, hydrosyringomyelia, and cutaneous abnormalities are common findings associated with lipomyelomeningocele.

This lesion is not associated with the Chiari II malformation.
**Case 8.18**

**HISTORY:** A 20-year-old woman with mild lower-extremity weakness

**FINDINGS:** Axial, T1-weighted (Fig. 8.18.1) and T2-weighted (Fig. 8.18.2) images show that the spinal cord is divided by a fluid-filled cleft. The left hemicord is smaller than the right one.

**DIAGNOSIS:** Diastematomyelia

**DISCUSSION:** Diastematomyelia is a closed dysraphic state in which the spinal cord is divided into two hemicords by a sagittal cleft (131). This congenital anomaly most commonly occurs in the female population (80%–85%) and usually involves the low thoracic or upper lumbar regions (132). The term *split cord malformation* also applies to this spectrum of abnormalities, the outcome of which is determined by the fate of intervening primitive streak tissue (127). In one-half of the cases, each hemicord has its own dural sac i.e., type 1); in the other half, (i.e., type 2) the hemicords share a common dural sac (133). If the primitive streak tissue forms a bony or cartilaginous spur, the hemicords will be contained within two dural sacs. If a fibrous septum develops or the primitive streak tissue is resorbed, the hemicords will be contained within one dural sac (127). A fibrous or bony spur separating the two hemicords is present in 50% of cases and is multiple in about 6% of cases (134). Evaluation of the bony spur and associated bony abnormalities, including scoliosis and vertebral segmentation anomalies, is best accomplished with CT (135,136). MRI is useful in demonstrating associated spinal anomalies, including Chiari II malformation, tethered cord, and hydromyelia (which may be confined to one hemicord). Treatment is surgical, with removal of the bony spur, which may tether the cord.

*Aunt Minnie’s Pearl*

Diastematomyelia is a closed dysraphic state in which the spinal cord is divided into two hemicords by a sagittal cleft.
**Case 8.19**

**HISTORY:** A 1-month-old girl with clubfeet and bladder incontinence

**FINDINGS:** Sagittal, T1-weighted (Fig. 8.19.1), and T2-weighted (Fig. 8.19.2) images show a conus medullaris that is truncated, a sparse cauda equina, and only one sacral vertebrum.

**DIAGNOSIS:** Caudal regression syndrome with partial sacral agenesis

**DISCUSSION:** The caudal regression syndrome is a congenital anomaly in which there is abnormal formation of the lower portion of the spine and spinal cord (60). There is a spectrum of abnormalities with various degrees of lumbosacral hypoplasia or aplasia, ranging from only partial malformation of the sacrum to fused lower extremities (i.e., sirenomelia or mermaid syndrome; 123–125). The caudal regression syndrome is frequently associated with abnormalities of the genitourinary system and with lower-extremity sensory and motor abnormalities. Caudal regression syndrome can be seen as part of other syndromic complexes such as VACTERL (vertebral abnormality, anal imperforation, cardiac malformation, tracheoesophageal fistula, renal abnormalities, limb abnormalities), OEIS association (omphalocele, exstrophy of the cloaca, imperforate anus, spinal anomalies), and the Currarino triad (partial sacral agenesis, anorectal malformation, and presacral mass) (127).
A high percentage of infants with this condition are born to diabetic mothers. The distal spinal cord typically has a blunted appearance (137). Occasionally, the cord is tethered at the level where the spine terminates, and a blunted (or truncated) conus medullaris is not seen. The cause of this condition is unknown but appears to be the result of some disturbance in the mechanism of retrogressive differentiation that is responsible for the formation of the conus medullaris and the cauda equine (138,139). Because the notochord, which is responsible for the formation of the distal spinal column, is affected, the distal spinal cord, the sacrum, and coccyx also fail to form normally. Motor impairment may occur, but sensation is usually relatively spared (140).

**Aunt Minnie’s Pearls**

In the presence of a truncated-appearing conus medullaris, always count the sacral segments (there should be five).

The caudal regression syndrome is rare, and many patients are the offspring of diabetic mothers.
**Case 8.20**

**HISTORY:** A 59-year-old man with a known disorder and new right-sided weakness.

**FINDINGS:** Axial T2-weighted (Fig. 8.20.1), T1-weighted (Fig. 8.20.2), and T1-weighted post-contrast (Fig. 8.20.3) images demonstrate a large heterogeneous mass with both intraspinal and paraspinal extension widening the neural foramen and displacing the thoracic cord to the right. The mass demonstrates heterogeneous enhancement. Also noted are innumerable subcutaneous lesions. A sagittal reconstruction obtained as part of a CT myelogram (Fig. 8.20.4) demonstrates levoscoliosis of the thoracic spine as well as the mass seen on the axial MR images centered at the apex of the scoliotic.
Case 8.20 (Continued)

Deformity. An axial image from the CT myelogram (Fig. 8.20.5) demonstrates lateral meningoceles.

**DIAGNOSIS:** Neurofibromatosis type 1 (NF1)

**DISCUSSION:** NF1 is the most common neurocutaneous syndrome, with an incidence of 1 in every 2,500 to 5,000 individuals (141–147). It is an autosomal dominant disorder caused by a mutation in the NF1 tumor suppressor gene located on chromosome 17, which codes for the protein neurofibromin (148). A list of clinical criteria has been established for the diagnosis of NF1, and including cutaneous lesions, nervous system tumors, Lisch nodules of the iris, osseous lesions, and a first-degree relative with the disorder. Two or more of these criteria must be met for the diagnosis (141).

Prominent spinal involvement can be seen in NF1 patients. The most common skeletal abnormality in NF1 is scoliosis most commonly involving the thoracic region. Other bony spinal abnormalities include scalloping of the posterior aspect of vertebral bodies, posterior element hypoplasia, and bony remodeling caused by compression from expanding soft-tissue tumors. Spinal tumors include neurofibromas (the characteristic lesion of NF1), schwannomas, and intrinsic spinal cord tumors. Neurofibromas can be localized, diffuse, or plexiform. Some consider plexiform neurofibromas pathognomonic of NF1 (143), which occur in ~50% of patients with NF1 (146). These benign tumors diffusely involve a long segment of a nerve and its branches in a tortuous manner and can extend beyond the nerve sheath into the surrounding tissues (143). The natural history of most tumors is slow growth, but degeneration into malignant nerve sheath tumors is the dreaded complication with a 7% to 13% lifetime risk in NF1 patients (146). Other spinal abnormalities seen in NF1 include dural ectasia and lateral meningoceles.

On plain-film, scoliosis, vertebral body scalloping, dysplastic posterior elements, widened interpedicular distances, and neuroforaminal enlargement can be seen (144). A “ribbon” deformity of the ribs can also be seen owing to multiple neurofibromas of intercostal nerves. CT imaging is useful in evaluating vertebral anomalies and bony changes associated with adjacent tumor growth. CT myelography may be helpful in assessing lateral meningoceles. MRI, however, is the optimal technique for evaluating the soft-tissue abnormalities of NF1.

Paraspinous and intraspinous neurofibromas are well visualized on MR. Although commonly asymptomatic in NF1, in the younger patients in particular, spinal neurofibromas can be seen at all segments of the spine at imaging (149). Spinal tumors in NF1 are primary intraforaminal extending into the canal but can also be intradural and seldom intramedullary (149). When small, tumors can be seen as nodules along the nerve roots of the cauda equina, but as they enlarge, they can widen the neural foramen.
and expand outside the canal in a “dumbbell” pattern (150). Tumors may also displace and compress the cord and cauda equina. On T1-weighted images, the tumors have signal intensity similar to muscle and they are hyperintense on T2-weighted images (143). Contrast enhancement is variable. Signs suggestive of malignant degeneration include new neurological symptoms associated with a lesion, increase in the size of a previously stable neurofibroma, and central necrosis with irregular peripheral enhancement (143).

**Aunt Minnie’s Pearls**

The majority of neurofibromas are solitary and not associated with NF1.

The majority of spinal nerve sheath tumors in NF1 are asymptomatic, particularly in children.

A previously stable lesion that becomes symptomatic or increases in size should raise suspicion for malignant degeneration.
**Case 8.21**

**HISTORY:** Two young patients (Fig. 8.21.1 is one patient and Figs. 8.21.2 and 8.21.3 are another patient) with weakness in all extremities, but predominantly in the lower ones.

**FIGURE 8.21.1**

**FIGURE 8.21.2**

**FIGURE 8.21.3**
Case 8.21  (Continued)

FINDINGS: A sagittal, T1-weighted image (Fig. 8.21.1) shows a low-intensity mass anterior to a compressed spinal cord at the C6–T2 levels. There is widening of the canal and scalloping of the posterior surface of the vertebral bodies in the region of the mass. In a different patient, a sagittal, T2-weighted image (Fig. 8.21.2) shows a hyperintense mass in the lower cervical spinal canal. The mass results in a wide canal and compression of the cord. There is fusion of the vertebral bodies anterior to the mass. An axial, T1-weighted image (Fig. 8.21.3) in the same patient shows that the mass is hyperintense.

DIAGNOSIS: Neurenteric cysts

DISCUSSION: About the third week of development, the amniotic cavity and the yolk sac are temporarily connected by the neurenteric canal that allows for transient contact of the endoderm with the neural ectoderm, which later forms the notochord. Incomplete separation of the notochord from the endoderm (precursor of the foregut) may result in formation of neurenteric cysts (151). These cysts are rare but have a fairly typical imaging appearance. Histologically, neurenteric cysts are derived from the gastrointestinal or respiratory tract. The cysts may be lined with mucus-secreting columnar epithelia resembling the lining of the gastrointestinal tract or pseudostratified ciliated epithelium resembling the respiratory tract lining (152). The cysts are usually found in the cervical or thoracic regions or in the posterior cranial fossa and are intradural, extramedullary masses. Neurenteric cysts may manifest at any time, depending on the severity of associated congenital malformations and the degree of compression of the spinal cord. In numerous cases, cyst decompression has provided dramatic resolution of symptoms such as paresthesias or paraplegia (151).

In 50% of cases, radiographs demonstrate vertebral anomalies such as fusion abnormalities or anterior or posterior spinal dysraphism (152). Other findings include widening of the spinal canal, diastematomyelia, and the Klippel–Feil anomaly. CT myelography generally demonstrates an intradural, extramedullary mass located in the ventral epidural space (153). MRI better delineates the contents of the cyst. Because of the high protein content of the cyst, T1 and T2 relaxation times are shortened. On T1-weighted sequences, neurenteric cysts demonstrate signal intensity higher than CSF. They are also bright on T2-weighted images (152,154).

Aunt Minnie’s Pearl

When vertebral body anomalies are seen along with cystic intraspinal masses, include neurenteric cysts in the differential diagnosis.
**Case 8.22**

**HISTORY:** A 53-year-old woman on chronic steroid treatment with low back pain

**FINDINGS:** Sagittal (Fig. 8.22.1) and axial (Fig. 8.22.2), T1-weighted images of the lumbar spine demonstrate circumferential, high signal-intensity tissue surrounding the distal thecal sac and compressing the nerve roots.

**DIAGNOSIS:** Epidural lipomatosis

**DISCUSSION:** Epidural lipomatosis comprises excessive accumulation of unencapsulated fatty tissue in the epidural space, generally in the thoracic or lumbar regions. Patients are usually on high-dose steroid therapy, are morbidly obese, or have endocrinopathies such as Cushing syndrome, although the disease may be idiopathic (155). The majority of patients with epidural lipomatosis are asymptomatic or have alternate explanations for their symptoms (156). For those who are symptomatic, the presenting symptom is usually chronic back pain; however, radiculopathies and myelopathy can occur (157). Treatment can be medical or surgical. Decreasing steroid dosages and instituting weight management have been effective for some patients (158). Decompressive surgery has also been successful at alleviating symptoms (155,159–161). Epidural lipomatosis generally does not occur in the cervical region unless it were to exist elsewhere too in the spine (156,161). In the thoracic region, fat accumulates more dorsally, whereas in the lumbar region, fat accumulation is circumferential (161).

In a recent meta-analysis of all 104 cases (156), the distribution of epidural lipomatosis was dependent on the underlying etiology. In steroid-induced lipomatosis, 56% of patients demonstrated increased epidural fat only in their thoracic spine, 33% only in the lumbar spine, and 11% affecting both thoracic and lumbar spines. Lipomatosis in the setting of obesity, however, favored the lumbar spine in 70% and the thoracic spine in only 30%. In idiopathic cases, 50% of patients had their disease localized to the lumbar spine, 38% to the thoracic spine, and 12% affecting both the thoracic and lumbar spines. Furthermore, it was noted was that success of therapy was dependent on the underlying etiology. In steroid-induced lipomatosis, both decompressive surgery and medical therapy had a success rate of 77%. In obesity-related lipomatosis, treatment with weight management alone had
improvement in symptoms in 82%. Decompressive surgery was successful in 67%. In the idiopathic group, decompressive surgery had a success rate of 94%. None in this group received conservative management.

On imaging, abnormal soft tissue, which follows the signal intensity characteristics of fat, is seen in the epidural space in the thoracic or lumbar regions. CT demonstrates fatty, low-density tissue filling the epidural space with impression on the thecal sac (160). This may extend for several vertebral levels. On MRI, the fat does not enhance, is homogeneous (a difference between normal fat and angiomyolipomas and angiolipomas), and decreases in signal intensity with the use of fat-suppression techniques. Furthermore, the normal width of the epidural fat is between 3 and 5 mm. More than 6 mm may be considered abnormal. However, there is only a loose link between the thickness of epidural fat and the presence of symptoms, as many asymptomatic people have an epidural fat thickness >6 mm (162). Extension of the fat into the neural foramina correlates with the presence of radiculopathy. In the distal lumbar spine, the lipomatosis surrounds the nerve roots.

**Aunt Minnie’s Pearls**

Epidural fat wider than 6 mm and extending for long segments is typical of epidural lipomatosis. Be sure to evaluate the spine for other causes of back pain as many individuals with an abnormal amount of epidural fat have alternate explanations for back pain.

Epidural lipomatosis is common dorsally in the thoracic region and circumferentially in the lumbar spine.

The signal intensity of epidural lipomatosis is nulled in fat-suppression sequences.
Case 8.23

**HISTORY:** A 58-year-old female presents with lower back pain
Case 8.23 (Continued)

**FINDINGS:** Lateral radiograph (Fig. 8.23.1) and axial/sagittal CT images (Figs. 8.23.2 and 8.23.3) of the lumbar spine demonstrate a lucency through the L5 pars interarticularis bilaterally. In a second patient, a lateral radiograph (Fig. 8.23.4) demonstrates a grade 1 anterolisthesis of L5 on S1 owing to bilateral pars interarticularis defects. The axial CT image through the L5 pars interarticularis in a third patient (Fig. 8.23.5), demonstrates differing appearances of the two pars interarticularis defects.

**DIAGNOSIS:** Bilateral pars interarticularis defects (isthmic spondylolysis)

**DISCUSSION:** Isthmic spondylolysis is an osseous defect of the pars interarticularis, which represents the confluence of the pedicle, lamina, and articular facets. With a general population prevalence of 3% to 6%, spondylolysis is a relatively common condition (163–165). More common in males (2:1) and adolescents or young competitive athletes, it is frequently seen at the level of L5, followed by L4. Although unilateral cases have been reported, most patients have bilateral pars interarticularis defects (166).

Spondylolysis may be asymptomatic, discovered incidentally. When symptomatic, it usually presents with back pain or radiculopathy. Likely a combination of a congenitally dysplastic pars interarticularis and repetitive micro-trauma leading to a stress fracture, spondylolysis may progress to spondylolytic spondylolisthesis (167,168).

With spondylolysis, the posterior elements including the inferior articular processes separate from their anterior counterparts—the vertebra body, pedicle, transverse process, and superior articular processes. On imaging, spondylolysis may present as a hairline fracture, fibrous ankylosis, or a pseudoarthrosis.

Imaging is utilized to detect pars interarticularis defects, establish prognosis, and guide treatment. Radiographs are usually sufficient for the initial diagnosis of spondylolysis or spondylolisthesis. Easily visualized on a lateral radiograph of good quality, oblique projections demonstrate the classic linear lucency in the “neck” of the pars interarticularis—appearing like a lucent collar on the neck of the “Scottie dog.”

Usually reserved for equivocal cases, surgical planning or posttreatment follow-up, CT with multiplanar reformatting is the most accurate imaging modality for pars interarticularis defect detection and healing assessment (169,170). Depending on slice thickness, the pars interarticularis defect may not be readily apparent on axial images. Therefore, the presence of other signs, specifically an enlarged vertebral canal or peri-articular process callus, can aid in diagnosis. Sagittal CT reconstructions can easily confirm pars interarticularis defects.

The appearance of pars interarticularis defects may differ from side to side in people with bilateral spondylolysis. The degree of healing and stress response may differ leading to situations where one side has an overt defect and the contralateral side has a more sclerotic appearance (166).

Given suboptimal visualization of bones, MRI is reserved for patients, mainly children, with
continued back pain yet no clear radiographic or CT findings of spondylolysis (165,170–172). STIR or T2-weighted fat-suppressed sequences are utilized, given their optimal visualization of bone edema. On sagittal images, wedging of the posterior vertebral body and displacement of the posterior elements is usually demonstrated, in patients with minimally subluxed spondylolysis or spondylolisthesis. Furthermore, MRI is superior in the assessment of the disc material, nerve root, and fibrous tissue in these patients. Hollenberg et al. have proposed a classification system for spondylolysis based on marrow signal and the appearance of the pars interarticularis cortical margin (173). Radionuclide bone scintigraphy has been used in the past but has lost favor owing to a relatively high degree of radiation exposure and lack of sensitivity and specificity (166).

Some clinicians would advocate the use of MRI as a secondary imaging modality (over CT), given its lack of ionizing radiation. However, the concomitant presence of degenerative changes in the adjacent facets may limit visualization of isthmic spondylolysis.

Based on work by Wiltse et al., spondylolisthesis is classified by etiology: isthmic (pars interarticularis defects), degenerative, traumatic (excludes pars interarticularis defects), pathologic, dysplastic, or iatrogenic (postsurgical) (174). Spondylolisthesis is also graded, based on the degree of the slippage of one vertebral body, relative to its caudal counterpart. Every 25% increment or fraction thereof of anterior displacement relative to the caudal vertebral body anteroposterior length represents an increase in the grade: grades 1 to 4.

Treatment depends on patient demographics and symptoms, chronicity of the findings, and degree of spondylolisthesis. With spondylolysis or mild spondylolisthesis, most cases are treated conservatively—usually a hard brace for a few months (165,175). In competitive athletes and patients with neurological symptoms or high-grade spondylolisthesis, surgical fusion may be required (176,177).

**Aunt Minnie’s Pearls**

Pars interarticularis osseous defects are common, present early in life, are usually bilateral, and may progress to spondylolisthesis.

Cross-sectional imaging, CT and/or MRI, may be necessary in equivocal, pediatric, or neurologically symptomatic cases.

Most cases can be treated with observation or brace immobilization, but surgical intervention may be required in severe cases or professional athletes.
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