Intramedullary Spinal Cord Abscess

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Intramedullary spinal cord abscesses are uncommon, especially in pediatric patients. Since the first description by Hart in 1830, fewer than 100 cases have been reported in the literature, with less than 40 cases reported in pediatric patients. This article describes a child with known spinal dysraphia and other spinal congenital anomalies who presented with loss of lower extremity function and an intramedullary spinal cord abscess. The etiology, pathophysiology, diagnosis, and treatment of spinal cord abscess are outlined, and differences between adults and children with this condition are discussed.

CASE PRESENTATION

Initial Presentation and History

A 2-year-old male child with a past medical history of myelomeningocele, sacral epidermoid cyst, spinal cord syrinx, and hydrocephalus with ventriculoperitoneal shunt presented with decreased ambulation, progressive lower extremity weakness, and new purulent drainage from a sacral epidermoid cyst. Three weeks prior to admission, the patient had difficulty walking independently and had a marked decrease in lower extremity tone. He underwent ventriculoperitoneal shunt revision and treatment for a sacral epidermoid cyst. The patient's lower extremity function did not return to normal, but the patient was discharged home without fever, irritability, decreased appetite, neck stiffness, rash, or other neurologic symptoms. Magnetic resonance imaging (MRI) performed prior to discharge revealed an unchanged spinal cord syrinx and a stable spinal dysraphia. On the day of the current admission, 10 mL of purulent material was drained from the sacral wound, and the patient was transferred to the pediatric tertiary care hospital for further evaluation and management.

Pertinent past medical history included spina bifida defect at L5, myelomeningocele at level L1-2, sacral epidermoid cyst, and spinal cord syrinx extending throughout the thoracic spine (most pronounced from T3–T8.) The patient had been receiving outpatient physical therapy for developmental delays associated with these conditions. Acetaminophen was the patient's only medication. Family medical history was negative for serious bacterial infections, progressive neuromuscular disorders, and childhood cancers. The patient lived in a rural area with his mother, had not traveled, and had no ill contact or tick/insect exposure.

Physical Examination

Physical examination revealed an irritable, ill but non–toxic-appearing child. The patient had a temperature of 38.6°C (101.5°F), but his other vital signs were otherwise normal. Examination of the head and neck was unremarkable with no nuchal rigidity and full range of motion of the neck. He had no photophobia or papilledema. Chest examination revealed clear breath sounds bilaterally with no murmur; abdominal examination was negative for masses or organomegaly. On the lower back in the mid-line, a 7-cm area of erythema with a small amount of brownish-yellow drainage was noted. The area was slightly fluctuant and very warm. Lower extremity examination revealed no spontaneous movements, no response to pain, and marked decreased tone. Toes were upgoing, and muscle bulk was within normal limits. Upper extremity examination and the remainder of the neurologic examination were unremarkable.

Laboratory and Imaging Studies

The patient's laboratory results revealed a leukocyte count of $17.6 \times 10^3/\mu L$ with a normal differential and a normal platelet count. Inflammatory markers were mildly elevated, including an erythrocyte sedimentation rate of 26 mm/h and a C-reactive protein level of 1.6 mg/dL. Results of wound cultures (prior to antibiotic therapy) showed pansensitive *Proteus mirabilis* and methicillin-resistant, vancomycin-sensitive *Staphylococcus epidermis.* All intraoperative wound cultures (after antibiotics commenced) and blood cultures (prior to antibiotic therapy) were negative for bacterial growth.

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Initial gadolinium-enhanced MRI revealed a sacral epidermoid cyst with rim-like enhancement extending superiorly within the spinal cord and into the syrinx (**Figure**); this was a significant change from a scan performed 3 weeks prior.

Clinical Course

Incision and drainage of the infected epidermoid cyst and spinal cord abscess occurred on hospital day 2. The postoperative course was complicated by urinary retention that required intermittent catheterization. The patient was started on vancomycin and ceftazidime. Because the *P. mirabilis* was pansensitive, ceftazidime was changed to ceftriaxone for ease of dosing. Parenteral antibiotic therapy was administered for 8 weeks. Serial MRI showed gradual improvement and eventual resolution of the intraspinal abscess. The patient gradually recovered lower extremity motor function. At discharge on hospital day 42, he was able to partially weight bear but was unable to ambulate independently, and he was urinating normally.

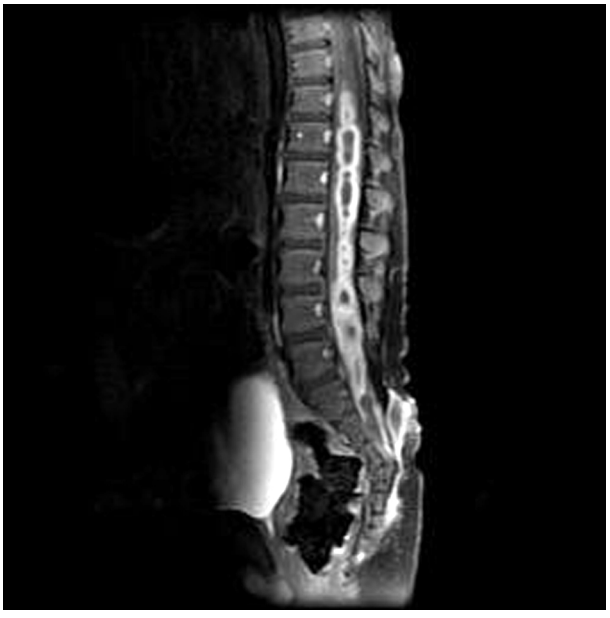

Figure. Sagittal view of a gadolinium-enhanced T2-weighted magnetic resonance image of the case patient. Multiple ring-enhancing collections are noted from T10 to the sacral spinal cord, and there is a ring-enhancing lesion posterior to the spinal cord that represents the infected dermoid cyst.

DISCUSSION

Epidemiology

Intramedullary spinal cord abscesses are very rare,[1] especially in children. More common central nervous system (CNS) infections in pediatric patients include meningitis, encephalitis, intracerebral abscess, and intracranial/spinal epidural abscess. Spinal epidural abscesses occur at a rate of 0.2 to 2 per 10,000 hospital admissions[2–4] and are more common than intramedullary spinal abscesses in both children and adults. Since 1830, fewer than 100 cases of spinal cord abscess have been reported in the literature.[5] Males have a higher predilection for spinal cord abscesses than females (3:2),[5] occurring more commonly in the first and third decades of life.[5,6] A higher incidence of spinal cord abscess has also been shown in intravenous drug users.[7]

Etiology

The bacteria most frequently found in spinal cord abscess aspirates are gram-positive cocci, such as *Staphylococcus* species (ie, *S. aureus*) and *Streptococcus* species (ie, *S. milleria*).[8–10] The second most common organisms are gram-negative rods, including *Escherichia coli*.[7] Cases of atypical organisms including *Proteus* species, *Listeria* species, *Brucella* species, *Mycobacteria* species, *Schistosoma mansoni*, *Actinomyces* species, and parasites have been reported.[5,8,10–12] *S. mansoni* is more commonly isolated in spinal cord aspirates of children as compared with adults. Spinal cord abscesses are not commonly caused by multiorganism infections.[9,13]

Pathophysiology

Spinal cord abscesses originate from hematogenous spread[8] or local extension.[14] The Batson plexus, the area where spinal epidural veins coalesce, is a rich vascular bed that may harbor bacteria.[7] Spinal cord abscesses are categorized as primary, when no other focus or entry of infection is noted, or secondary. Secondary spinal cord abscesses occur in 86%[9] of cases and are most frequently caused by infections that originate in the genitourinary tract, bony spine, lung, or heart valves.[7–9,13] Spinal cord abscesses are generally associated with congenital dermoid lesions or sinus tracts,[1,8,9] most commonly presenting in the lumbar or midthoracic cord[15] but may also occur in conjunction with congenital dermal sinuses or trauma.[5,6]

Intramedullary spinal cord abscesses are classified as acute or chronic and can occur in isolation or with several distinct abscesses within the spinal cord.[14] While adults tend to have more insidious symptoms and signs of spinal cord abscesses (ie, chronic abscess), children experience rapid progression of symptoms (as quickly as several hours).[15]

Diagnosis

Usual clinical symptoms of spinal cord abscesses include back pain with pinpoint tenderness.[5,15] In

nonverbal patients, lower extremity weakness (resulting in impaired ambulation) or loss of bowel and bladder control are frequently the presenting symptoms. Most patients with acute spinal cord abscesses report fever, chills, myalgias, and malaise; however, pediatric patients may present without signs of systemic infection. Although the differentiation between spinal cord and epidural abscesses is difficult, spinal cord abscesses illicit less tenderness with percussion of the vertebral column.

When there is clinical suspicion for a spinal cord abscess, laboratory data are helpful. Leukocytosis with a left-shifted differential and increased inflammatory markers (eg, platelets, C-reactive protein, and/or erythrocyte sedimentation rate) are typical.[1,15] As with other CNS infections, cerebrospinal fluid may reveal elevated leukocytes, decreased glucose, increased protein, and positive Gram stain. However, cerebrospinal fluid studies may be unremarkable in cases of spinal cord abscess.[16] Because the risk of seeding the spinal column is high, lumbar puncture is contraindicated when cellulitis or a skin abscess overlies the lumbar spine or when an epidural abscess is suspected.[6,15]

Gram stain and cultures should be obtained from wound or abscess fluid during evacuation. Because atypical organisms can be isolated, slides for parasites and cultures for anaerobic bacteria, *Mycobacteria* species, and fungi must be obtained.

MRI with gadolinium enhancement allows better differentiation of the affected spinal planes, visualization of abscess cavities, and localization of boney destruction or sinus tracts.[7] Therefore, post-gadolinium MRI is preferred to contrast computed tomography.[15] Because MRI poorly differentiates abscess from tumor, surgical exploration is always indicated. Findings on MRI suggestive of an abscess include high signal on T2-weighted images with poorly defined enhancement on T1-weighted images.[8] If MRI or computed tomography are not diagnostic, metrizamide myelography may be considered.[1,5,15]

Differential Diagnosis

Differential diagnoses of spinal cord abscesses include epidural abscess,[14] discitis, osteomyelitis with bony destruction, meningitis, spinal cord hematoma, Guillain-Barré syndrome,[8] transverse myelitis, para- or intraspinal tumors[8] including sarcomas or neuroblastomas, and herpes zoster infection.[15] Acute presentation of decreased lower extremity motor function, especially in conjunction with fever or other signs of infection, are most consistent with abscesses, neurologic conditions (eg, Guillain-Barré syndrome, transverse myelitis), and other infectious processes. Progressive onset is more typical of tumors, which cause compression or destruction of spinal parenchyma with resultant loss of motor function.[5,7]

Treatment

Surgical treatment uniformly requires exploration and evacuation of abscess pockets, usually via laminectomy above and below the abscess, incision of the dura, and visualization and aspiration of abscess cavity.[13] Many neurosurgeons advocate for intraspinal antibiotic flushes after abscess drainage, but most antibiotics have excellent CNS penetration.[15] Some surgeons place an intraspinal drain to measure fluid output, which can be used to indicate a persistent abscess or failed medical therapy. Other neurosurgeons use glucocorticoids perioperatively to control superimposing spinal cord inflammation.[17] Frequently, these steroids are tapered within 2 weeks.

In addition to complete abscess drainage, an appropriate course of intravenous antibiotic therapy is essential, beginning with broad-spectrum antibiotics to target the most common organisms (ie, *Staphylococcus*, *Streptococcus*, and gram-negative species). Antibiotics are narrowed if an organism is isolated.[13] Most experts advocate a minimum of 4 weeks of intravenous antibiotics after abscess drainage and laboratory data improvement;[5] however, a several-month treatment course is usually recommended at many clinical institutions.[8] For mycobacterial spinal cord abscesses, 1 year of parenteral therapy is indicated.[8]

Follow-up spinal column imaging to assure complete resolution of the abscess cavity is recommended; however, the ring enhancement around the abscess cavity may persist for several weeks after successful treatment.

Outcome

Functional outcome after intramedullary spinal cord abscess is determined by the timeliness and appropriateness of interventions, including surgical débridement and effective antibiotic treatment. Today, patients experience better outcomes because surgical techniques and antibiotics have improved. However, a subpopulation of patients experience marked functional neurologic impairment, including paraplegia, caused by recurrent or chronic abscess formation, or spinal cord infarct, a complication due to inflammation and occlusion of the vertebral vasculature. The mortality rate for intraspinal abscesses approaches 30% due to sepsis, perioperative complications, and comorbidities.[9] Patients with acute presentations (ie, < 7 days of symptoms prior to seeking medical care) have poorer prognoses than those with slowly progressive symptoms.[5,6,8] **HP**

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