ABSTRACT. Multiple complications of varicella have been described. Musculoskeletal complications (osteomyelitis, septic arthritis, and necrotizing fasciitis) as well as neurologic complications (ataxia, encephalitis, and transverse myelitis) are well-known. We describe the cases of 2 children, ages 18 months and 5 years, who were admitted recently to 2 pediatric hospitals in Montreal with a resolving varicella, abdominal and lumbar pain, and a refusal to walk and in whom a diagnosis of epidural abscess caused by group A streptococcus (GAS) was established. No previous case of epidural abscess caused by GAS in the context of varicella has been reported. Epidural abscesses are rare in pediatrics and are caused mainly by hematogenous spread of Staphylococcus aureus. The diagnosis in pediatrics is challenging because it is rare and does not present as classically as in adults. The prognosis is related to the presence of neurologic deficits before surgery and to the rapidity with which the diagnosis and the intervention are made. These cases highlight a new clinical association in children of epidural abscess caused by GAS and varicella. An early clinical diagnosis requires a high index of suspicion when back or abdominal pain with or without neurologic signs and symptoms occurs during or soon after varicella. Pediatrics 2002;109(1). URL: http://www.pediatrics.org/cgi/content/full/109/1/e14; varicella, spinal epidural abscess, group A streptococcus.

VARICELLA

Varicella is a common childhood viral illness, with a projection of 380 000 cases annually in Canada, approaching the annual birth rate.1 The estimated risk of hospitalization is approximately 1 in 550 cases.2 Multiple complications from varicella have been described. The majority of these consist of secondary skin and soft tissue bacterial infections ranging from 45% to 73% of the reported complications caused mainly by group A beta-hemolytic streptococcus (GAS) and Staphylococcus aureus.2,3 Neurologic complications (18%-21%), such as encephalitis, postinfectious cerebellar ataxia, Reye syndrome, aseptic meningitis, and transverse myelitis, have also been reported.2,4,5 Musculoskeletal complications mostly attributable to GAS (osteomyelitis, necrotizing fasciitis, myositis, and septic arthritis) are well-described,6 but no previous case of spinal epidural abscess (SEA) has been found in the literature. We describe 2 cases of children with a diagnosis of SEA as a complication of varicella.

CASE 1

A previously healthy 1½-year-old boy presented with fever up to 40°C for 4 days and refusal to sit or walk for 3 days. He had developed varicella 6 days before the onset of fever and had a mild case until then. His mother had received a diagnosis of streptococcal pharyngitis the week before admission. On examination, he was irritable. There was no evidence of secondary bacterial superinfection of the skin and soft tissues. He had a stiff neck and bilateral brisk patellar deep tendon reflexes, clonus, and positive Babinski sign. The muscular strength was normal. His peripheral white blood cell (WBC) count was 14.8 x 10^9/L. A lumbar puncture yielded clear cerebrospinal fluid (CSF) containing 50 WBC/mm³, 4.3 mmol/L glucose, and 13.4 g/L proteins. Gram stain and culture of the CSF were negative. Intravenous therapy with cefotaxime and vancomycin was started. A throat culture done by his pediatrician 3 days before admission grew GAS. Magnetic resonance imaging (MRI) of the spine showed a posterior extradural collection pushing the spinal cord forward from T3-T4 to T10-T11 and peripheral contrast enhancement after gadolinium injection (Fig 1). The patient was taken to the operating room (OR) 36 hours after admission, where a T5-T11 laminectomy was performed and revealed 3 epidural abscesses with purulent material posterior to the cord. The pus and the blood culture taken on admission grew GAS (M3/T3). Once the culture results were obtained, the treatment was changed to aminocillin G intravenously on which he remained for a total course of 6 weeks. A brace was installed for 3 months, and he improved gradually. At the end of the intravenous antibiotic course, no sequelae remained and a repeat MRI showed complete healing.

CASE 2

A previously well 5½-year-old boy presented to a pediatric hospital with complaint of left paravertebral lumbar pain and left-sided abdominal pain for the previous 2 days. A history of fever up to 40°C for the preceding 4 days as well as a resolving varicella (day 7 of evolution on admission) was of notice. New lesions appeared until the fifth day of varicella, but no sign of secondary bacterial skin superinfection was present. During his varicella course, he was in contact with a cousin who had streptococcal pharyngitis. On examination, he was not toxic but was irritable with a slightly stiff neck and was refusing to sit, stand, or lie on his back because of pain. On palpation, he localized the pain to the lumbar left paravertebral region. Deep tendon reflexes and strength were normal. A lumbar puncture revealed 112/mm³ WBC, 3.6 mmol/L glucose, 0.42 g/L protein. Gram stain and culture of the CSF were negative. Cefazolin was given intravenously on admission and changed to ceftriaxone after the lumbar puncture was obtained. His peripheral WBC was 23.2 x 10⁹/L, and his throat culture was also positive for GAS. An MRI done 36 hours after admission revealed an epidural collection around the cord extending from T8 to T10 and obliterating the CSF space that enhanced after gadolinium injection. The patient was taken to the OR that same day, where a T9-T11 laminectomy was performed. Pus was drained, the fibrin was cleaned off the

From the *Division of Infectious Disease, Department of Pediatrics, Sainte-Justine Hospital, University of Montreal, Quebec, Canada; and †Division of Infectious Disease, Department of Pediatrics, Montreal Children’s Hospital, McGill University, Montreal, Quebec, Canada. Received for publication Jun 5, 2001; accepted Aug 21, 2001. Reprint requests to (B.T.) Sainte-Justine Hospital, 3175, Côte Sainte-Catherine, Montreal, Quebec, Canada, H3T 1C5. E-mail: bruce_tapiero@ssss.gouv.qc.ca or tapiero@cedep.net PEDIATRICS (ISSN 0031-4005). Copyright © 2002 by the American Academy of Pediatrics.
dural surface, and irrigation with an antibiotic solution was done. The patient improved gradually and defervesced after the surgery. The OR specimen as well as the blood culture grew GAS (M12/T12). The treatment was then changed to intravenous aqueous penicillin G, but for convenience, the patient was sent home on ceftriaxone to complete a 4-week course of intravenous antibiotics. The patient remains free of sequelae, and a repeat MRI after the completion of the intravenous antibiotics showed complete healing.

**DISCUSSION**

SEAs in the adult population occur mainly in patients with underlying conditions—diabetes, intravenous drug use, immunodeficiency, previous spinal surgery—and are increasing in frequency. The incidence of SEA varies from 0.2 to 1.2 per 10,000 admissions in all age groups. A recent review of the literature in children revealed that SEA is rare in the pediatric age group and that the predominant pathogen found was *S. aureus* with methicillin-resistant organisms becoming an increasingly important causative agent. A review done by Rubin et al on SEA in the pediatric age group also found that the main causative agent was *S. aureus* (79%) followed by *Streptococcus viridans* and *Streptococcus pneumoniae* (4% each), and *Salmonella enteritidis* (2%). In our review of the literature, we only found 1 other case of SEA caused by GAS; it was reported by Nussbaum et al in a previously healthy 7-year-old girl with no previous history of varicella.

One case of SEA in the context of varicella was reported previously in a 2-year-old girl who developed varicella while admitted. Her skin lesions became secondarily infected with *S. aureus*, with associated sepsis and pneumonitis. Skin abscesses were incised and drained and grew *S. aureus*. SEA was diagnosed after the incision and drainage were performed. We are not aware of any cases of SEA in varicella caused by GAS.

Aebi et al reviewed bacterial complications of primary varicella in children. Of the 84 patients with a bacterial complication, 36 (43%) had a GAS infection. Eighteen patients of 84 had invasive infections. Only 2 patients had acute pharyngitis. Nonetheless, 17 of 36 patients with GAS infection had a throat culture done, 7 of which were positive for GAS.

GAS is a known cause of serious infections that occur during the course of varicella. Law et al reviewed chickenpox admissions to 11 pediatric Canadian hospitals from 1991 to 1996 and found that 49% of the serious bacterial infections were caused by GAS, but no cases of SEA were found.

SEAs are mainly secondary to hematogenous seeding of the epidural space in pediatrics with the primary focus being mainly skin and soft tissue infection but could also be upper respiratory tract infections, urinary tract infection, and osteomyelitis. The primary focus for our cases was probably the pharyngitis (throat culture positive in 2 of 2 patients) because there was no evidence of skin superinfection.

The classical symptomatology of SEA was described by Heusner. First, back pain and fever appear and progress to spinal ache and root pain and can mimic acute abdomen. The reflexes can be altered before motor weakness is seen. Increasing weakness of the muscles and bowel as well as bladder incontinence will be followed by total paralysis. The differential diagnosis includes myelitis caused by bacterial meningitis, syphilis, viral meningitis, and parainfectious process, as well as the syndrome of acute transverse myelopathy of unknown cause. Rarely, a lymphoma may mimic a SEA. Spinal cord tumors, vascular malformations, and arachnoiditis are to be thought of when evidence of sepsis is minimal or absent.

Rubin et al looked at the CSF abnormalities in children with SEA and found that 22 of 42 cases (52%) had a purulent CSF, 26% had signs of suspected meningeval infection (mild leukocytosis or low glucose levels), and 12% had increased proteins as the only CSF abnormality. Ten percent had normal lumbar punctures. Danner and Hartman found that a CSF protein content of >3.5 g/L was associated with a complete block of the spinal canal. Rockney et al also found that 9 of 17 (53%) of the CSF abnormalities in children with SEA and found that 22 of 42 cases (52%) had a purulent CSF, 26% had signs of suspected meningeval infection (mild leukocytosis or low glucose levels), and 12% had increased proteins as the only CSF abnormality. Ten percent had normal lumbar punctures. Danner and Hartman found that a CSF protein content of >3.5 g/L was associated with a complete block of the spinal canal. Rockney et al also found that 9 of 17 (53%) of the
patients with SEA had elevated CSF protein values and 41% had a mildly elevated WBC count. These descriptions fit with our patients’ lumbar puncture profiles.

Treatment lies in prompt surgery, and the outcome has been reported to be directly related to the preoperative neurologic impairment. Some studies in adults showed that certain categories of patients could be treated only with intravenous antibiotics, but no data are available in children.  

CONCLUSION

SEA in children is rare. These cases highlight a new clinical association in children who have SEA caused by GAS and varicella. Prompt clinical diagnosis and treatment are mandatory. It requires a high index of suspicion because of variable nonspecific presentation but should be looked for when a child with varicella presents with abdominal or back pain with or without neurologic findings, regardless of the lumbar puncture results.

Furthermore, these cases emphasize the importance of a vaccination program against varicella. With the increasing use of varicella vaccine in Canada since its recent licensure, the incidence of such bacterial complications of varicella is expected to decrease.

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