two Solusan injections at the Olson's point at weekly intervals. The elbow was splinted at 90° of flexion.

Pain, numbness, and Tinel's sign persisted, and he was unable to work. Diagnosis with sensory nerve conduction studies could not be done because he had severe pain and burning in the distribution of the lateral antebrachial cutaneous nerve, and declined any electrical studies. The patient was treated by surgical release of the lateral antebrachial cutaneous nerve. At surgery, the nerve was found compressed between the brachialis and biceps tendon as it emerged laterally under the tendon (Fig 2). The nerve was freed between the biceps tendon and the brachialis muscle. The bicipital tendon was neither incised nor partially excised. The severe pain at the elbow and forearm disappeared. The patient regained full range of motion of the elbow, Tinel's sign became negative, and he returned to work in 3 weeks. At 18-month follow up, he was asymptomatic.

**DISCUSSION**

The lateral antebrachial cutaneous nerve emerges at the lateral side of the elbow between the bicipital tendon and brachialis muscle at their intersection with the elbow crease. The nerve supplies cutaneous sensation to the radial half of the volar forearm.

When this nerve is compressed, patients present with pain at the elbow, positive Tinel's sign at Olson's point, pain, numbness, tingling, or burning in the radial half of the volar forearm. Symptoms last from 3 days to 12 weeks in patients with acute injuries who forcefully extended the elbow in pronation. Bassett noted that the nerve is under more compression in pronation than supination. Our patient's main complaint was severe pain at the elbow and inability to extend the elbow beyond 45° with burning pain over the radial aspect of the volar forearm.

We confirmed the diagnosis in our patient with a 1 mm injection of Xylocaine at the Olson's point. This relieved the patient's burning pain at the elbow and forearm. Since the condition is rare, being the first case we saw in 16 years, the results of the injection were helpful in confirming diagnosis. This test has not been reported previously.

Sensory conduction studies of the lateral antebrachial cutaneous nerve have been described by Trojaborg, and Felsenthal. Trojaborg stimulated the lateral antebrachial cutaneous nerve with needle electrodes below the elbow, and recorded action potentials in the axilla and Erb's point in the anterior cervical triangle. Spindler stimulated the lateral antebrachial cutaneous nerve at the Olson's point and recorded action potentials in the distal forearm using surface electrodes with antidromic technique. This technique is simpler and less painful to the patient. Felsenthal reported a decrease in amplitude of the evoked response in three patients, but prolonged distal latency was found in only one.

Conservative treatment was successful in four of 11 patients treated by Bassett and one of three patients treated by Felsenthal. The conservative treatment consisted of oral inflammatory medications, rest, restriction of activities, sling, posterior splint, ultrasound, TENS, and local cortisone injections over a 12 week period.

Surgical treatment described by Bassett consisted of release of the LACN at the elbow between the bicipital tendon and brachialis muscle, and wedge resection of the bicipital tendon. Felsenthal released the nerve as above and partially incised the bicipital tendon. In both instances, the patients had induration of the scar at the elbow. We believe that violation of the bicipital tendon is not necessary as the tendon continues to rest on the nerve.

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**ORTHOPEDIC SEQUELAE OF MENINGOCOCCEMIA**

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In 1887, Weichselbaum showed that the cause of meningitis was *Neisseria meningitidis*, a gram-
negative coccus.\textsuperscript{1} Infections by this organism may affect any organ system and are widely varied in their clinical manifestations. Approximately 60\% of meningococcal infections occur in patients <15 years old, and military recruits are especially vulnerable. Ninety percent to 95\% of patients with meningococcal infection have meningococcemia or meningitis. Thirty percent to 35\% of patients have meningococcemia without meningitis. Onset is abrupt, but is often preceded by a short period of coughing, cephalgia, or sore throat, followed suddenly by high fever, chills, myalgia, and mild hypotension. About 75\% of these patients develop the petechial rash during the bacterium. Chronic meningococcemia is a rare form of the infection, seen with intermittent fever and maculopapular rash. Joint involvement occurs in about 20\% of these patients, and meningitis, carditis, and nephritis may occur if diagnosis and treatment are delayed.\textsuperscript{2}

In 10\% to 20\% of patients with diffuse meningococcal infection, fulminant meningococcemia may occur.\textsuperscript{4} This may result in massive purpura, hypotension, encephalitis, adrenal failure, consumptive coagulopathy, and mortality due to circulatory collapse. Bacterial meningitis is one of the more well-known infections caused by this organism. Arthritis occurs in approximately 5\% of meningococcal infections and may present as a purulent synovitis, sterile polyarthritis,\textsuperscript{5} monoarthritis, or as a tenosynovitis. Signs and symptoms of joint involvement may not occur until after treatment of meningitis or meningococcemia. This may occur as an immune phenomenon, and permanent joint changes have been reported.\textsuperscript{4,5}

The skin lesions seen in meningococcemia result from damage to skin blood vessels. The organism can often be found in aspirates from involved skin. Immunglobulin and complement are present even in early vascular lesions.\textsuperscript{9} Edema, infarction of overlying skin, and extravasation of red blood cells are responsible for skin lesions. Similar lesions occur in other tissues.\textsuperscript{10} In addition, many cutaneous hemorrhagic lesions may be caused by a dermal Schwartzman reaction due to a lipopolysaccharide endotoxin of meningococcus.\textsuperscript{11} This endotoxin may also be responsible for a generalized Schwartzman reaction contributing to the severity of the systemic disease.\textsuperscript{11}

At our institute, we have identified three patients presenting with rare and severe orthopedic sequelae of meningococcal infection: purpura fulminans resulting in gangrenous extremities; premature epiphyseal arrest resulting in severe angular deformity; and shortening of the involved extremity. These cases are presented to remind the clinician of the detrimentally capricious nature of meningococcal infections.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image1.png}
\caption{Case 1: Extensive skin ecchymosis and necrosis over the face and extremities due to purpura fulminans.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image2.png}
\caption{Extensive debridement for skin graft of same case as in Figure 1.}
\end{figure}

\section*{CASE REPORTS}

\textbf{Case 1.} A 3-year-old boy presented with a 3-week history of fever and neck soreness. On the day of evaluation and admission, he developed a diffuse ecchymotic rash and generalized seizures. Meningococcal meningitis was diagnosed and confirmed by culture; appropriate antibiotic therapy began immediately on admission. Within the first hospital week, the patient developed patchy areas of ischemic necrosis on the face and extremities. Purpura fulminans secondary to meningococcal meningitis was diagnosed (Fig 1). The child was subsequently transferred to our institute for orthopedic treatment, multiple debridements
Fig 3: Case 2: Anterior view demonstrates severe varus deformity of both knees (A). Posterior view (B).

Fig 3A.

Fig 3B.

Fig 4: AP radiograph of case 2.

of ischemic skin, and subsequent grafting, in addition to further antibiotic treatment (Fig 2).

Case 2. A 6-month-old girl was treated successfully with appropriate antibiotic therapy for culture-proven meningococcal meningitis. Three months posttreatment, the patient's parents noted a mild genu varum, which progressed with growth. She was referred for evaluation of this progressive angular deformity. History was otherwise noncontributory. Physical examination showed marked genu varum (Fig 3). Radiographs of the lower extremities showed irregularity and asymmetric closure of her proximal and distal tibial physes (Fig 4). Brace treatment was unsuccessful and the patient required multiple bony procedures, including osteotomies, limb lengthenings, and subsequent epiphysiodesis to halt further deformity.

Case 3. In 1954, an 11-year-old boy developed sudden onset of vomiting, diarrhea, and fever to 106°F orally. This was followed shortly thereafter by a "convulsion of short duration and peculiar movements." On admission, he was obtunded and noted in the first 48 hours to develop diffuse petechia, jaundice, and hepatomegaly. He remained obtunded and was
treated with IV fluid support and blood transfusions for a hemoglobin of 9.6 mg/ml. Initially, he was treated empirically with chloromycetin followed by sodium sulfadiazine. This was discontinued after 3 days due to developing gross hematuria. On the sixth hospital day, he was begun on IV penicillin. His condition seemed to stabilize after several doses of the penicillin; however, during this time, it was noted that he began sloughing the skin of his progressively ischemic, gangrenous extremities. By the end of the first month of hospitalization, he had autoamputated distal aspects of his left foot and fingers of both hands. Blood and urine cultures reported as negative.

After slightly more than 2 months of hospitalization, he was transferred to the authors’ institute for definitive orthopedic care of his dry, gangrenous, ischemic extremities. On his 11th hospital day, he was to undergo debridement of his extremities under general anesthesia, but suffered cardiopulmonary arrest from which he was not able to be resuscitated. This occurred during administration of anesthesia and was prior to any surgical procedure. Results of any post-mortem examination were not available.

**DISCUSSION**

Sequelea of the endotoxin *Neisseria meningitides* are varied. In meningococcemia, the essential lesion is vascular, resulting in damage to vessel walls with subsequent necrosis, thrombosis, or hemorrhage in the involved tissue. Rapidly spreading hemorrhage in the skin is the characteristic feature of purpuric fulminans. Hypofibrinogenemia and thrombocytopenia may be seen. Similar involvement of deeper tissues may result in irreversible ischemic changes and gangrene. With larger vessel involvement, an entire extremity may be affected. Immune-related vasculitis due to a Schwartzman-like reaction has been implicated. The mortality rate in this condition may be as high as 50%. Steroids, heparin, and immune-mediating agents have all been tried with uncertain benefit. The course of the disease is rapid with evolution occurring over days.

Surgical treatment of these later changes may necessitate amputation, which may be performed after the line of demarcation has been established in about 3 or 4 weeks. Emergent amputation may become necessary in the case of progressive septicemia or myoglobinuria. Open amputation with delayed closure has been advocated in the actively infected extremity. In the well-demarcated “dry” gangrenous extremity without active infection, primary closure may be performed. It should be remembered that septic embolization is a continued vascular threat in meningococcal infection.

Premature epiphyseal arrest may occur and result in angular deformity and limb-length discrepancy. Three mechanisms of physeal arrest are postulated. The most likely mechanism is ischemia due to disseminated intravascular coagulopathy. The second most likely mechanism is septic embolization leading to septic arthritis or osteomyelitis, and the third, direct extension of overlying infection to the growth plate.

Epiphyseal arrest may not manifest for several years after the initial physeal insult; therefore, growth should be monitored closely by the orthopedic surgeon after any such severe illness. Growth plate arrest, as evidenced by radiographic abnormalities, may present in three general types:

1. Asymmetrical peripheral physeal destruction, causing angular deformity.
2. Central arrest producing a ball-and-socket deformity.
3. Complete physeal arrest, resulting in shortening of a bone or limb.

Management of physeal arrest is dependent on type of arrest, amount of deformity, and amount of growth remaining. Physeal bar resection may be useful to restore growth to a partial premature arrest. Percutaneous selective epiphysiodesis may be indicated to correct angular deformity and equalize limb-length discrepancy. Limb-lengthening procedures offer a potential solution to the problem of limb-length discrepancy >5 cm.

**CONCLUSION**

Meningococcal infection may be severe and multisystemic. Management requires expertise of medical and surgical specialties in severe cases. Orthopedic management needs to pay particular attention to the control of limb-threatening sequelae. In cases where limb salvage is achieved, continued vigilance is needed to monitor the growth of the affected extremities. Treatment of growth disturbance may be needed at a later date.

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