vomiting and diarrhea. She was found to have fetal demise and was treated with dilation and curettage. She had generalized weakness, which at the time contributed to dehydration and electrolyte imbalance. The patient was later readmitted for progressive generalized weakness and an inability to ambulate. Ultrasound revealed retained fetal products. Results of an initial physical examination revealed proximal lower extremity weakness and areflexia without sensory deficit.

Setting: A tertiary hospital.

Results: Initial nerve conduction studies revealed decreased motor amplitudes in the legs, with relatively preserved sensory responses. The motor and sensory studies in the arms were normal. Initial electromyography showed acute denervation and loss of motor units in proximal lower extremity muscles. Acute denervation was also seen in distal leg muscles. The upper extremities or lumbar paraspinals were electrically normal. Subsequent nerve conduction studies revealed a loss of motor and sensory responses in both arms and legs. Repeated electromyography reflected a similar denervation pattern in the upper extremities. She was diagnosed with acute motor and sensory axonal neuropathy subtype of Guillain-Barré syndrome (GBS). The patient had antecedent diarrheal illness but *Campylobacter jejuni,* antiganglioside GM1 antibodies, monoclonopathy, human immunodeficiency virus, West Nile serologies, and CSF studies were negative.

Discussion: GBS is the most common cause of acute flaccid paralysis in the world. It is considered to be a postinfectious, immune-mediated disease targeting peripheral nerves though immune mimicry. There are several GBS subtypes: acute inflammatory demyelinating polyradiculoneuropathy, acute motor axonal neuropathy, and acute motor and sensory axonal neuropathy. The acute motor and sensory axonal neuropathy variant often presents with rapid and severe paralysis with delayed and poorer recovery. In several studies, *C jejuni* was the most commonly isolated pathogen associated with the axonal subtype.

Conclusions: In the presence of multiple negative serologies, this would be the first case report that suggests a hyperimmune response to fetal demise and retained products as a potential etiology of GBS.

Poster 75

The Importance of Electrodiagnostic Medicine in Diagnosing Compression Neuropathy of the Posterior Tibial Nerve Secondary to a Ganglion Cyst: A Case Report.

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Disclosures: N. Gohari, none.

Patients or Programs: A 19-year-old woman.

Program Description: A 19-year-old female athlete presented with numbness and tingling on the plantar surface of her left foot for a 2-month duration with intermittent weakness of toe flexion. The patient's symptoms were exacerbated with knee flexion. Physical examination revealed weakness of left foot plantarflexion and toe flexion. There was hyperesthesia on the lateral plantar aspect of her left foot and the left ankle reflex was diminished.

Setting: A tertiary care facility.

Results: The patient had a nerve conduction study, which showed focal conduction failure in the left posterior tibial nerve below the innervation of the gastrocnemius soleus complex. There was an amplitude drop on left tibial nerve stimulation at the abductor hallucis from 7.1 ms at the ankle to 0 ms at the popliteal fossa; similarly, the abductor digiti quinti pedis dropped from 1.3 ms to 0 ms. Digital plantar nerve and tibial F wave were absent. Tibial H reflex, peroneal motor, superficial peroneal, and sural nerves were normal. Needle electromyography showed active denervation in the left tibialis posterior, left abductor hallucis, abductor digiti quinti pedis, and first dorsal interosseous muscles. All other muscles tested were normal, including soleus and medial gastrocnemius. This study thus localized the site of the lesion to be between the popliteal fossa to above the ankle. Magnetic resonance imaging showed a multiloculated cystic collection ganglion cyst that compressed the tibial nerve from the medial aspect of the proximal tibiofibular joint. Surgical excision of the ganglion cyst improved the patient's symptoms.

Discussion: Posterior tibial nerve neuropathy secondary to a ganglion cyst is very rare and this is the fifth reported case. Ganglion cyst of a peripheral nerve must be considered in the differential diagnosis of neuropathy.

Conclusions: Although most physicians will agree that magnetic resonance imaging is the diagnostic tool of choice for detecting ganglion cyst, this case clearly proved how invaluable electrodiagnostic medicine is in localizing the site of pathology.

Poster 101

Pediatric Mononeuritis Multiplex Secondary to a Nonsystemic Vasculitis: A Case Report.

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Disclosures: E. Y. Ben-Ozer, none.

Patients or Programs: A 16-year-old girl with 3-4 months of symptoms.

Program Description: She had right arm and then leg paresthesias that improved. After, she developed left-sided symptoms that evolved into pain and weakness. On manual muscle examination, she had greater deficits on the left and distally. Left finger flexion, finger abduction, wrist extension, ankle dorsiflexion and plantarflexion were all 1/5. Electrodiagnostic studies revealed absent responses of the left median sensory, sural sensory, peroneal motor, and tibial motor nerves. The left ulnar sensory nerve had decreased amplitude and prolonged latency. The right median motor, peroneal, and tibial motor nerve amplitudes were reduced, with normal latencies and conduction velocities. The right median sensory, ulnar sensory, and median motor nerves were normal. Electromyography demonstrated active denervation and decreased numbers of motor unit potentials in an asymmetric distribution. These findings showed a severe sensorimotor axonal polyneuropathy consistent with a clinical diagnosis of mononeuritis multiplex. Sural nerve biopsy revealed a small-vessel vasculitis. Magnetic resonance angiography of the brain, chest, abdomen, and extremities showed no areas of vasculitis.

Setting: A university hospital electrodiagnostic laboratory.

Results: She was started on oral prednisone and Cytoxan therapy and was later switched to CellCept. She participated in aggressive physical therapy with a full recovery other than trace left ankle dorsiflexion weakness.

Discussion: This represents a case of a nonsystemic vasculitis that caused a mononeuritis multiplex confirmed by sural nerve biopsy. It is a very uncommon form of childhood-acquired polyneuropathy

with less than a handful of cases reported in the medical literature. In addition, a nonsystemic vasculitis in childhood is even rarer.
Conclusions: Asymmetric sensory or motor symptoms that develop in previously healthy children may suggest an acute vasculitis. Electrodiagnostic evaluation can determine the extent and nature of the possible neuropathic process. Sural nerve biopsy can identify vasculitic disorders. Prompt initiation of immunosuppressive therapy with a rehabilitation program offers the best opportunity of functional recovery.

Poster 102

Lumbosacral Plexopathy After Gunshot Wounds: A Case Report.

Zahava T. Traeger, MD (NYU Medical Center, New York, NY, United States); Joan Gold, Charity K. Hill, MD, Dong Ma, Adam Silver, DO.

Disclosures: Z. T. Traeger, none.
Patients or Programs: A 14-year-old boy with lumbosacral plexopathy after gunshot wounds.
Program Description: The patient presented to acute care after 2 gunshot wounds to the abdomen, which required multiple procedures, including a laparotomy and left thoracotomy for abdominal aortic repair. He had an L3 fracture and paraspinal hematoma, with paresthesias and left lower extremity (LLE) weakness. He was given a lumbar sacral orthosis and an ankle-foot orthosis. Upon admission to acute rehabilitation, his LLE strength was 2+/5 to 3–/5 except 0/5 for dorsiflexion. He had LLE tingling. Electrodiagnostic testing revealed positive sharp waves in the left tibialis anterior, peroneus longus, tibialis posterior, tensor fascia lata, gastrocnemius, vastus medialis and rectus femoris, and gluteus maximus with reduced recruitment patterns on strong effort. There was no response on sensory nerve action potentials in the left superficial and sural nerves. Findings were consistent with a left lumbosacral plexopathy with the L5 root most severely involved.
Setting: A pediatric rehabilitation unit in a tertiary care hospital.
Results: The patient made significant functional gains, with improvements in bed mobility, transfers, and ambulation. Upon discharge, he was independent in activities of daily living and ambulated 100 ft modified independent with a cane. He was without further improvement of innervation of LLE or gait pattern as an outpatient. He is pending reimaging and electrodiagnostics and possible plexus exploration for adhesions.
Discussion: The patient presented with a lumbosacral plexopathy after trauma, an L3 fracture, paraspinal hematoma, and abdominal surgery. Lumbosacral plexopathy is relatively uncommon because the plexus has a rich blood supply. It presents with motor and sensory deficits in a distribution of multiple nerves that originate from the plexus. Etiologies include pelvic injuries or tumors, hemorrhages, trauma, ischemia, inflammation, and postpartum injury.
Conclusions: Lumbosacral plexopathy is rare, has varying etiologies, and may cause severe neurologic deficits. Electrodiagnostic testing can be diagnostic.

Poster 103

Mononeuritis Multiplex in Leprosy. A Case Report.

Jorge Diaz-Ruiz, Professor (Universidad Nacional de Colombia, Bogota, Colombia); Camilo Mendoza-Pulido, Fernando Ortiz-Corredor.

Disclosures: J. Diaz-Ruiz, none.
Patients or Programs: A 44-year-old woman with borderline tuberculoid leprosy that was treated 15 years ago.
Program Description: This patient was referred to the physiatrist with 1-month severe neuropathic pain in her left foot. She had paresthesia in her hands and right foot. She had no weakness. On physical examination, she had hypoesthesia, allodynia, and areflexia in her left foot. She also had tender and enlarged the ulnar and posterior tibial nerves. Her strength was normal, and she had no nasal mucosa or skin lesions. Autoimmune, infectious, and metabolic diseases were ruled out. Electrodiagnostic evaluation showed mild axonal disease in the right ulnar nerve and severe axonal loss in the posteriottibial, peroneal, and sural nerves of the left foot. Needle electromyography showed denervation in abductor hallucis, abductor digitiminimi, and extensor digitorum brevis of the left foot. Leg muscles were normal. Sural nerve biopsy indicated perivascular and endoneural inflammatory infiltrates with severe axonal loss. No foamy macrophages or acid-fast bacilli were observed. Pure neuritic leprosy was diagnosed and was treated with prednisolone, dapsone, rifampicin, and minocycline, with symptoms improvement.
Setting: A university hospital.
Results: One year after treatment, she did not have pain or paresthesia. On physical examination, hypoesthesia without allodynia was found on the sole of the left foot. Electrodiagnostic assessment was normal for the ulnar nerve, with mild improvement of amplitude of the motor responses of the peroneal and posterior tibial nerves. Some denervation was found in abductor hallucis muscle.
Discussion: This is a mononeuritis multiplex in a patient with leprosy. The pure neuritic form exhibits no skin lesions and, unlike this particular case, is more frequent in males. In general, it damages the small nonmyelinated fibers that carry temperature and pain.
Conclusions: Leprosy has different neurologic manifestations that may appear many years after the onset of the clinical picture.

Poster 104

Chronic Hoarseness as Isolated Presentation for Early Motor Neuron Disease: A Case Report.

Amanda Farag, MD (Kessler, West Orange, NJ, United States); Jeffrey L. Cole, MD.

Disclosures: A. Farag, none.
Patients or Programs: A 55-year-old man with progressive hoarseness and positional shortness of breath.
Program Description: The patient presented with a 5-year history of positional shortness of breath exacerbated by lying supine as well as progressive hoarseness and subjective sensation of throat closure exacerbated by jogging. He denies any history of dysphagia. He was recently diagnosed with left diaphragm paralysis on a diaphragm sonogram. Pulmonary function tests revealed severe restrictive ventilatory defect with severe reduction in lung volume and air trapping. On physical examination, while supine, the patient was in respiratory distress with oxygen desaturation that resolved when upright. Testing of manual muscle strength, sensation, reflexes, and gait revealed no deficits. On electrodiagnostic testing, the left phrenic nerve showed prolonged onset latency of 9.14 ms (normal, approximately 8.0 ms) in association with a significantly decreased CMAP amplitude of 0.09 mV (normal, >0.2 mV) as well as sustained pseudomyotonia, sustained dystonic firing, spontaneous denervation potentials, and sustained firing at a rate approaching tetany. Left infrahyoid-omohyoideus muscle showed complex repetitive pseudomyotonic discharges at rest, whereas right crico-