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To avoid renal injury after LPB, several precautions might be considered: (1) the block needle must be introduced at the level of L4 or L5,1 (2) the location (depth and laterality) must be visualized by encountering the fourth or fifth lumbar transverse process,1,2 (3) confirmation of the nerves by electrical stimulation is helpful,1 and (4) performing block procedures under x-ray fluoroscopy may be desirable. These procedures may be appropriate for other nerve blocks, such as lumbar sympathetic ganglion block, LPB, and celiac ganglion block, to avoid renal injury.

References


Postoperative Onset of Idiopathic Brachial Neuritis

E. E. Fibuch, M.D.,† Janet Mertz, M.D.,† Bruce Geller, M.D.†

THIS report describes a case of postsurgical idiopathic brachial neuritis (IBN) in an otherwise healthy female physician. The recognition of this entity as a potential cause of symptoms of peripheral neuropathy in the postoperative patient is important for anesthesiologists to know because of the possibility that it may be misdiagnosed as a brachial plexus stretch injury or ulnar nerve compressive injury.

Case Report

A 4-yr-old, ASA physical status I woman was scheduled to undergo a diagnostic hysterectomy, dilatation, and curettage because of menometrorrhagia. Preoperatively, there was no history of trauma or viral or bacterial infection. Her review of systems, medical and surgical history, and family history were noncontributory. She had no allergies to medications and was taking only vitamins. She was 162.5 cm tall and weighed 49.9 kg. Her vital signs (blood pressure 124/70 mmHg) and physical examination results were within normal limits.

In the operating room, standard monitoring was applied. While breathing oxygen, she was given atropine (0.5 mg), fentanyl (100 μg), and ketorolac (60 mg) intramuscularly in the left deltoid muscle and metaclopromide (10 mg) intravenously before induction. Anesthesia was induced with propofol (1 mg/kg) and maintained with a propofol infusion (2 mg·kg⁻¹·min⁻¹). The head was maintained in a neutral position, with the intravenous catheter in the right arm, abducted to approximately 70 degrees from the thorax. The left arm was tucked to the left side of the patient. At no time did her systolic blood pressure intraoperatively decrease to less than 95 mmHg. Anesthesia and surgery were uneventful. The anesthetic lasted approximately 34 min. The patient emerged from general anesthesia without sequelae and was taken to the recovery room, where she had an uneventful anesthetic recovery.

Because the patient was doing well after discharge, she elected to go to a restaurant for dinner that evening (12 h postoperatively).

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During the course of the dinner, she complained to her husband (a physician) of an acute burning pain in both shoulders, radiating into the neck and scapula. The pain was so uncomfortable that she became nauseated and terminated eating her meal. That evening she could not get comfortable, despite the use of heat, massage, a cervical collar, and analgesics (naproxen sodium and chlorzoxazone). The following morning, the pain persisted, and weakness in the shoulder girdle had developed. Later that day, she noticed a tingling sensation radiating into her left lateral posterior arm and into the thumb and first and second fingers. The pain gradually subsided in 2 weeks, but the weakness on the left side persisted. This weakness made it difficult for her to perform certain procedures in the operating room.

Approximately 4 weeks after the onset of symptoms, she was evaluated by a neurologist, who noted that her review of systems was unremarkable except for the above noted complaints. On physical examination, her cranial nerves were intact. Manual muscle testing revealed normal trapezius bilaterally (5/5) and diminished strength in the left deltoid (3/5), left supraspinatus (2/5), left infraspinatus (2+/5), and left teres minor (3–4/5) muscles and the left rhomboids (2+ to 3–/5). The left triceps muscle was slightly weak (5/5). The remainder of the manual muscle testing was almost normal on the left and normal on the right. There was mild atrophy of the left infraspinatus muscle. Reflexes in the upper and lower extremities were normal and equal bilaterally. Sensory examination to pinprick was normal except for a decreased appreciation of pinprick in the left second digit. Results of coordination and gait testing were unremarkable.

Electromyographic and nerve conduction studies demonstrated, approximately 1 month after the onset of pain, a chronic left C7 radiculopathy and an acute and chronic denervation in muscles sharing innervation through the C5 nerve root and the upper trunk of the brachial plexus. The cervical paraspinal muscles did not demonstrate acute denervation. This pattern noted on electromyographic and nerve conduction studies, in view of the clinical history and examination findings, suggested a left neuralgic amyotrophy.

Because of the persistence of left upper extremity weakness, approximately 2 months after the onset of symptoms, laboratory evaluation for serum proteins and thyroid function was performed, and results were within normal limits. However, when ganglioside antibody titers were evaluated, they were noted to be increased, suggestive of an ongoing immune phenomenon (table 1).

A magnetic resonance imaging study of the brachial plexus and neck was performed and found to have normal results, except for a mild posterior disc bulge at C5–C6 that caused minimal impingement anteriorly on the thecal sac.

Finally, urine evaluation results for heavy metals (arsenic, lead, mercury, and cadmium) were noted to be normal.

Based on the above findings, the diagnosis of postoperative IBN was made. At 11 months after the initial onset of pain, the patient has recovered full function and is painfree.

**Table 1. Ganglioside Antibody Titers Measured in Our Patient**

<table>
<thead>
<tr>
<th>Type</th>
<th>Patient Titer (units)</th>
<th>Normal (units)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgM–GMI</td>
<td>1.030</td>
<td>&lt;600</td>
</tr>
<tr>
<td>IgG–GMI</td>
<td>2.500</td>
<td>&lt;800</td>
</tr>
<tr>
<td>IgM–Asialo-GMI</td>
<td>Normal</td>
<td>&lt;1,000</td>
</tr>
<tr>
<td>IgG–Asialo-GMI</td>
<td>1.400</td>
<td>&lt;1,000</td>
</tr>
<tr>
<td>IgM–Sulfadate</td>
<td>4.300</td>
<td>&lt;1,500</td>
</tr>
<tr>
<td>IgG–Sulfadate</td>
<td>2.500</td>
<td>&lt;1,500</td>
</tr>
<tr>
<td>IgM–g–tubulin</td>
<td>1.400</td>
<td>&lt;1,500</td>
</tr>
<tr>
<td>IgG–g–tubulin</td>
<td>2.370</td>
<td>&lt;1,250</td>
</tr>
</tbody>
</table>

IgM = immunoglobulin M, GMI = ganglioside M, IgG = immunoglobulin G; Asialo = without monosialic acid.

*Review of autoantibodies in neurological diseases can be found in reference 1.

First described by Spillane in 1943,14 this disorder has had many names, such as “brachial plexus neuropathy,” “acute brachial radiculitis,” “nerveitis of the shoulder girdle,” “neurologic amyotrophy,” “shoulder girdle syndrome of Parsonage and Turner,” “paralytic brachial neuritis,” “sensory brachial neuritis,” and “nontalgic paresthesia.”15

IBN is characterized by patchy weakness and atrophy of muscles of the shoulder girdle.2,9 The weakness often is preceded by intense and unbearable pain, maximum in intensity at the onset of symptoms,10 which gradually recedes, leaving variable muscle dysfunction of the proximal muscle group. The muscles most often involved, in decreasing frequency, are the deltoid, supraspinatus, infraspinatus, serratus anterior, biceps, and triceps.10 Sensory disturbance occurs in about two-thirds of the patients10 but tends to be minimal and generally resolves in time.7 Seventy percent of the patients have involvement of the axillary nerve.10 Overall recovery may take 2 yr.15 A complete review of the natural history of IBN has been published.8

Using the Mayo Clinic data base for the period 1970–1981, Beghi et al.15 noted an incidence of 1.64 cases per 100,000 in the population of Rochester, Minnesota. Eleven cases fulfilled their criteria for IBN, and of these, six occurred after an antecedent event of flu-like symptoms (four cases) and tetanus toxoid immunization (two cases). In addition, two others included a previous neurologic disorder (encephalitis and peripheral neuropathy). Although the authors could not arrive at a specific etiology for IBN in their study, they suggested that an immune or inflammatory process may be the

Discussion

Postoperative IBN is a well recognized clinical syndrome in the orthopaedic and neurologic literature3–13 but has not received wide attention in the anesthesiology literature.

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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case, specifically citing the similarities between IBN and the neuropathy of serum sickness, although no immunologic studies were performed in this series of patients.

In addition to the possibility of an immunologic etiology for IBN, cases have been reported after trauma, pregnancy, and surgery and in a hereditary form. A review of 111 cases of IBN noted the following antecedent events: acute trauma (n = 37), recurrent trauma (n = 21), following infection (n = 16), postpartum (n = 7), following injections (n = 12), unrelated surgical procedures (n = 7), flu-like symptoms (n = 13), and idiopathic (n = 18).

Recently, Malamut et al. reported an additional six cases of IBN after unrelated surgical procedures. None of their cases had the risk factors for IBN. These cases were similar in presentation to our report. Five of the six patients had general anesthesia. These authors noted that, when upper extremity pain and weakness occurred postoperatively without obvious etiology, it often is related to compressive neuropathies, cervical radiculopathy, or brachial stretch injuries. All of these latter conditions can place the anesthesiologist at legal risk.

The reason some cases of IBN occur after surgery and general anesthesia is speculative. Autoimmune neuropathies have been reported to occur in the postoperative period. Wilson et al. have suggested that the stress of surgery may activate an unidentified virus lying dormant in nerve roots, a circumstance, we speculate, similar to the onset of herpes zoster after surgery.

Laboratory (including cerebrospinal fluid analysis) and radiologic studies (including myelography) are generally of minimal help in making the diagnosis. In our patient, however, antibody titers for immunoglobulin IgM and IgG ganglioside proteins were increased, suggesting an ongoing immune phenomenon. There was no evidence in the preoperative history of our patient nor in the postoperative interview for this case report that any of the reported etiologic risk factors were present.

The use of electrodiagnostic studies, consisting of electromyography and nerve conduction analysis, is probably the most helpful laboratory test in securing the diagnosis, in conjunction with a complete clinical evaluation. Weikers and Matsson reported their results of electrodiagnostic studies in 10 of 11 patients with the diagnosis of IBN. In each case, the study results were abnormal, showing evidence of denervation, with positive waves and fibrillations seen on needle electrode examination. In our case, the electromyography was performed approximately 1 month after the onset of symptoms and demonstrated that acute and chronic denervation were present. Nerve conduction velocities generally were slowed maximally in the distal part of the peripheral nerve, despite clinical weakness in the proximal muscle bed. This pattern was similar in our patient.

To our knowledge, this is the first case report of IBN to measure elevated ganglioside titers after the onset of IBN. The measurement of ganglioside antibody titers may represent a better laboratory test to differentiate postoperative cases of IBN from those patients presenting with symptoms of peripheral neuralgia secondary to a stretch or compressive injuries of the brachial plexus. Further study is required to substantiate this impression.

We have presented a case of IBN in an otherwise healthy woman, that occurred spontaneously 12 h postoperatively after an outpatient gynecologic procedure under general anesthesia. Although the etiology is unknown, a strong link to an immune reactive phenomenon can be made and is noted in our patient’s laboratory evaluation. Recovery is generally complete using conservative treatment but may take up to 2 yr for resolution. An understanding by anesthesiologists of the possibility that IBN may occur spontaneously in the postoperative period is important because of the serious medicolegal implications of this diagnosis being missed and one of brachial stretch injury or compressive neurapraxia being made or compressed.

Practice Guidelines for Non-Anesthesia: A Report by the Task Force on Sedation and Analgesia by Anesthesiologists

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Accepted for publication by the American Society of Anesthesiologists: Jeffrey J. Ackerman, M.D., B. Sehgal, Washington, Woodville, Washington; Fred G. Davis, M.D., B. Arents, M.D., Washington, D.C.; John M. Zervas, Jr., M.D., Cleveland, Ohio. A report by the Task Force on Sedation and Analgesia by Anesthesiologists. Approved by the House of Delegates of the American Society of Anesthesiologists. Address correspondence (M/C 2015) to Universitas Corporation, 520 North North 25th, Milwaukee, Wisconsin, 53202. Key words: Analgesia, consciousness.