Guillain–Barré Syndrome Mimicking Nerve Injury After Total Hip Arthroplasty

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Abstract *Background* Guillain–Barré syndrome (GBS) is a rare, acute, inflammatory demyelinating polyneuropathy with a presentation of progressive ascending motor weakness of the extremities, which may extend to the respiratory muscles and require mechanical support. *Case Description* This case describes a healthy male who developed GBS 1 week following otherwise an uncomplicated bilateral total hip arthroplasty surgery. The diagnosis was made based on physical exam significant for bilateral lower extremity weakness and diffuse areflexia, and confirmed by nerve conduction studies. There were no effects on respiratory function, and the patient underwent a gradual recovery, with near complete return of motor function by 10 weeks following surgery and no functional deficits at 1 year following surgery.

Functional activity levels are maintained 4 years post-operatively. *Literature Review* A literature review is presented. While GBS has been described in the literature following several different forms of surgery and anesthesia, cases after orthopedic surgery are exceedingly rare. *Purposes and Clinical Relevance* This case demonstrates that GBS may occur following orthopedic, including total joint arthroplasty, surgery. The presentation of GBS can be variable, but the hallmarks are areflexia and diffuse ascending weakness, with or without sensory symptoms. Rapid diagnosis and treatment are critical for preventing disability and life-threatening sequelae.

Introduction

Guillain–Barré syndrome (GBS) is a rare, acute inflammatory demyelinating polyneuropathy that affects approximately 0.6 to 4 out of every 100,000 people [8]. Patients usually present with progressive, ascending motor weakness of the extremities, areflexia, and sensory disturbances. Weakness may extend to the muscles of respiration, resulting in the need for mechanical ventilatory support. Approximately 20% of patients are left with a chronic disability, while mortality rates range from 4% to 15% [3, 8, 16]. The exact etiology of GBS remains unknown, but several triggers have been identified, such as infection, pregnancy, vaccination, posttransplant immunosuppression, transfusion and systemic illnesses, such as systemic lupus erythematosus or Hodgkin's lymphoma.

A number of reports of GBS following different types of surgery and anesthesia have been presented [1, 2, 5, 6, 10, 14, 15, 19, 20], but few have been published in the orthopaedic literature [4, 14, 17]. The current case report describes a healthy 64-year-old male who developed Guillain–Barré syndrome approximately 2 weeks following single-stage bilateral total hip replacement.

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Each author certifies that his or her institution has approved the reporting of this cases, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participating in the study was obtained.

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Case Report

A 64-year-old male with medication-controlled hypertension presented with a history of chronic severe bilateral hip pain refractory to conservative treatments and radiographs demonstrating severe bilateral hip osteoarthritis. The patient underwent uncomplicated single-stage bilateral total hip arthroplasty under combined spinal–epidural anesthesia, in the left and right lateral decubitus positions, respectively. Cemented cobalt chromium femoral stems, femoral heads, and press-fit titanium acetabular components with highly cross-linked, high-molecular-weight polyethylene liners were implanted on both sides through a posterolateral approach. Cumulative estimated blood loss for the bilateral surgery was 400 ml. Postoperative anterior–posterior radiograph of the pelvis revealed good prosthetic component alignment fixation.

On the evening following the surgery, the patient was started on warfarin for DVT prophylaxis. Thromboembolism deterrent stockings were worn at all times, and sequential compression devices were used while in bed. A patient-controlled epidural analgesia infusion (bolus doses only; no basal rate) was used for pain management in the immediate postoperative period. He received two units of autologous blood the day of surgery and two units of homologous blood for mild anemia on postoperative day 1. The patient was ordered daily Nexium for ulcer prophylaxis, ferrous sulfate for anemia, and colace for prevention of constipation. Due to the need for extensive osteophyte resection, 600 cGy of radiation was administered to each hip for heterotopic ossification prophylaxis.

On postoperative day 1, the patient was able to stand at the bedside with the physical therapist. On postoperative day 2, gait training with a walker was initiated, the epidural catheter was removed, and the patient was treated with oral analgesics. The patient remained neurologically intact from the time the spinal–epidural anesthesia wore off until the time of discharge. On postoperative day 5, the patient was transferred to a rehabilitation center. He continued to progress well with therapy and was able to ambulate and climb one flight of stairs with a cane prior to discharge home on postoperative day 14.

Over the ensuing 2 days at home, the patient began to experience paresthesias and slight weakness in his hands and feet, in addition to altered taste (dysgeusia). The senior surgeon was contacted and saw the patient in his office on postoperative day 17 and immediately readmitted him to the hospital. Examination was significant for bilateral lower extremity areflexia and grade 4/5 motor strength of foot dorsiflexion and plantar flexion. Upper motor neuron reflexes were intact. On hospital day 2, the weakness had progressed in both the lower and upper extremities. Foot plantar flexion and dorsiflexion were graded as 3+/5 bilaterally while the quadriceps, hamstrings, and iliopsoas were 4+/5. Laboratory studies were within normal limits except for a platelet count of 743 per nanoliter. It has been postulated in the literature that myelodysplasia (including thrombocytosis) is associated with Guillain–Barré syndrome [12]. Myers et al. [11] reported a series of patients

who were noted to have, among other hematologic abnormalities, elevated platelet counts both preceding and during the course of Guillain–Barré syndrome.

A neurology consultation and nerve conduction studies were obtained, which showed prolonged distal latencies of 238% and 139%, the upper limit of normal in the right and left median nerves, consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP), a subtype of Guillain–Barré syndrome. A spinal tap was not performed because the patient was anticoagulated with coumadin. The patient was transferred to a monitored setting and administered a 5-day course of IV immunoglobulin (0.4 mg/kg), during which time there was no change in his neurological exam. Pulmonary medicine was consulted, and pulmonary function tests revealed no evidence of respiratory compromise. The patient developed autonomic dysreflexia on postoperative day 21, which led to hypertensive attacks and was treated with antihypertensive medication. In addition, the patient developed anxiety, which resolved following treatment with a selective serotonin receptor inhibitor and a benzodiazepine. There was minimal residual hip pain, which was relieved with acetaminophen. Two weeks after the symptoms began, the patient had motor strength of 3+/5 in most muscle groups in the lower extremities and 4/5 in the upper extremities. At 10 weeks postoperatively, nearly all muscle groups were graded as 4/5, and the patient was able to ambulate with Canadian crutches.

At 1-year follow-up, the patient was able to walk 1 mile and ascend stairs with a banister. The neurological examination revealed that patellar reflexes had returned to normal bilaterally, while Achilles reflex remained absent. He complained of mild hypoesthesia below the ankles and bilateral lateral thigh hypoesthesia in the distribution of the L2 and L3 dermatomes. The motor examination revealed complete (5/5) recovery of bilateral hamstring and tibialis anterior strength and 4/5 iliopsoas, abductor, and quadriceps strength bilaterally. Gastrocnemius–soleus strength was 4/5 on the left side and 3/5 on the right side. He had no complaints of hip pain bilaterally and reported no functional deficits with his activities of daily living. Range of motion was symmetrical, revealing 110° of flexion, 40° of abduction, 10° of adduction, 10° of internal rotation, and 25° of external rotation bilaterally.

The patient was contacted 4 years postoperatively for telephone follow-up. At this time, the patient reported “excellent” mobility and ambulatory function, and he was able to walk unlimited distances without assistive devices, ascend and descend stairs without the use of a railing. He exercises daily, including playing golf and lifting weights. He denied hip pain and reports full strength in all extremities, with the exception of mildly weak right ankle plantar flexion and weakness with toe extension in multiple toes bilaterally. He reported mildly decreased sensation in both feet and paresthesias bilaterally distal to the knees approximately once per week. His sensory function is otherwise intact. The Modified Harris Hip Score was 100% at 4 years postoperatively.

Discussion

This case describes a healthy 64-year-old male who developed Guillain–Barre syndrome (GBS) approximately 2 weeks following uneventful bilateral total hip replacement. While bilateral primary total hip arthroplasty (THA) is associated with higher complication rates compared with unilateral primary THA, both have relatively low overall complication rates. Nerve injury following total hip arthroplasty has been reported to be 0.8–1.3% in large series [13, 18].

Guillain–Barre syndrome is an acute, inflammatory demyelinating polyneuropathy that affects approximately 0.6 to 4 out of every 100,000 people [8, 9]. Four subtypes have been described, of which the AIDP is the most common. While extremely rare in the postoperative period, it has been described following a diverse range of procedures, including pancreatectomy [1], cranial surgery [5], mandibular surgery [19], gastrectomy [15], spine surgery [17], and transplant surgery [2]. Koc et al. [10] contended that surgical stress may be a key triggering factor in the etiology of GBS. The authors suggest that the physiological alterations to the immune system may be responsible for the development of the condition rather than the surgery itself. The etiological contribution of surgical procedures versus the anesthesia used during the surgery is unknown, as one case of GBS has followed anesthesia performed for obstetrical labor [6]. Hebl et al. presented convincing evidence that patients with preexisting peripheral sensorimotor neuropathy or diabetic polyneuropathy are at significantly increased risk of neurologic complications after neuraxial anesthesia or analgesia [7], which may include GBS. One theory implicates the administration of epidural anesthesia and the interaction between the anesthetic agents and peripheral nervous system myelin or local trauma to nerve roots, initiating a cascade of immunologic events that result in the demyelinating neuropathy 1–2 weeks after anesthetic delivery [20]. This idea remains controversial considering the lack of tangible evidence supporting this relationship; however, the case presented here followed a similar timeline. Our patient received combined spinal–epidural anesthesia before the surgery and patient-controlled epidural analgesia (bolus doses only, no basal rate) following the surgery and had no evidence of preoperative or early postoperative neurological deficits until 14 days postoperatively. The most common pathological conditions preceding GBS are flu-like illnesses and gastroenteritis, with approximately two thirds of patients having an infection within 6 weeks prior to the diagnosis [3]. Vaccinations have been associated with GBS in the past, but little confirmatory evidence exists for vaccines being etiological factors [3]. Our patient had neither a recent illness nor any vaccinations in the months prior to surgery. One case report documented only a blood transfusion prior to the onset of GBS [11], suggesting that the devastating neurologic outcomes associated with GBS may follow only minor physiologic perturbations. Our patient received two units of autologous blood and two units of homologous blood which is not an abnormal transfusion requirement for bilateral THA at our institution.

In one of the few reports of GBS in the orthopaedic literature, Reibel et al. [17] describe a 62 year-old woman who underwent lumbar spine decompression and fusion for degenerative lumbar scoliosis and spinal stenosis. Despite complaining of distal lower extremity paresthsias in the early post-operative period, no formal neurologic workup was initiated until 25 days after surgery, by which time GBS with diffuse areflexia, numbness, and bilateral upper and lower extremity weakness had rendered the patient non-ambulatory. While De Decker et al. [4] described GBS in a patient who had previously undergone arthroplasty, the etiology was felt to be instead the development of syndrome of inappropriate anti-diuretic hormone and the resulting extreme hyponatremia. One recent report in the French literature [14] describes a patient with GBS with an onset two weeks following hiparthroplasty, but this was in conjunction with an upper respiratory infection as well. A second patient in the same report [4] underwent a hemiarthroplasty for treatment of a femoral neck fracture and developed GBS in the early post-operative period with no other causes.

While the patient in the current report had a very similar presentation to that in the patient who underwent lumbar spine fusion [17], our patient was diagnosed expeditiously following readmission with nerve conduction studies. While neither the patient in the report by Reibel et al. nor the patient in the current report had significant respiratory symptoms, up to 20% of patients may remain chronically disabled following GBS, with mortality rates ranging from 4% to 15% [3, 8, 16].

In conclusion, while more common causes of nerve injury following orthopedic procedures must be ruled out in patients with neurological signs or symptoms in the early postoperative period, orthopedic surgeons must be familiar with other pathological conditions of both the central and peripheral nervous systems, such as Guillain–Barré syndrome. Speculative causes include epidural anesthesia, which is used commonly during orthopedic procedures of the lower extremities. This case following bilateral total hip arthroplasty demonstrates that GBS may occur following orthopedic, including total joint arthroplasty, surgery and is the first case of its kind in the English language literature. Regardless of molecular etiology, presentation has been shown to occur approximately 2 weeks postoperatively, roughly the interval required for the immunologic cascade resulting in demyelinating polyneuropathy. The presentation of GBS can be variable, but the hallmarks are areflexia and diffuse ascending weakness, with or without sensory symptoms. Other factors may be related to the condition, such as preceding systemic illnesses and infections, immunological compromise, anesthesia, and perhaps blood transfusion. Rapid diagnosis and treatment are critical for preventing disability and life-threatening sequelae.

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