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Acute Guillain-Barré Syndrome After Sacroiliac Joint Fusion

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Background: Guillain-Barré syndrome (GBS), also called acute inflammatory demyelinating polyradiculopathy (AIDP), is one of the most common causes of acute, acquired weaknesses. GBS is an acute immune-mediated polyneuropathy that presents with progressive weakness of the arms or legs. GBS presents after infection; however, there are few reports that describe acute GBS after chronic pain procedures.

Case Report: A 70-year-old man with a past medical history of spinal stenosis status post anterior cervical discectomy and fusion, sacroiliac joint (SIJ) dysfunction status post SIJ fusion presented with imbalance, weakness, and difficulty walking. The patient underwent SIJ fusion one week prior; his weakness progressively worsened, requiring a walker. He underwent a series of labs and diagnostic tests, which were consistent with AIDP/GBS. He was placed on respiratory and cardiac monitoring and started on intravenous immunoglobulin treatment. He started developing bilateral facial palsies and started on plasmapheresis. His symptoms have improved and he was discharged from our inpatient rehab facility on after 28 days.

Conclusions: This case report aims to highlight a rare, but potentially dangerous, complication of AIDP/GBS following an SIJ fusion.

Key words: Sacroiliac joint fusion, sacroiliac joint pain, Guillain-Barré syndrome, case report

BACKGROUND

Guillain-Barré syndrome (GBS), also called acute inflammatory demyelinating polyradiculopathy, is a commonly acquired weakness that is often provoked by a preceding infection. The acute polyneuropathy is triggered by an immune response to a previous infection, or other inflammatory events that cause a cross-reaction with shared epitopes on peripheral nerves (1,2). It can affect all myelinated nerves: including motor, sensory, cranial, and sympathetic nerves (1-3). The most common antecedent infectious agents are campylobacter jejuni gastroenteritis, followed by influenza A and B. However, other known causes of GBS are surgical procedures, the postpartum period, as well as several medications such as tacrolimus and isotretinoin (3,4).

The pathophysiology behind GBS is that a widespread inflammatory response develops against myelin-producing Schwann cells or peripheral myelin (4-6). Demyelination starts at the nerve root where the blood-brain barrier is deficient (4,5). The breakdown of the barrier allows for infiltration of T cells and macrophage-mediated demyelination, leading to evidence of complement activation and immunoglobulin deposition on myelin and Schwann cells (5,6).

Clinical features of GBS vary depending on the severity of the syndrome. The common clinical features of GBS include progressive, usually ascending, and symmetric muscle weakness with absent or depressed deep tendon reflexes (4,5). However due to demyelination of the autonomic nervous system, patients can present with dysautonomia (4,5). The most frequent autonomic symptoms are ileus, hypertension, or hypotension. Initial

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symptoms typically present within a few days to a week. The peak of the disease is usually reached around 4 weeks postinfection (6,7). The initial diagnosis is based upon clinical features. Clinical features include progressive weakness of the arms and/or legs, ranging from minimal weakness of the legs to complete paralysis of limbs, bulbar and facial muscles, and possibly, external ophthalmoplegia.

Diagnostic tests can aid diagnosing, but are not required to diagnose GBS. If patients do not meet diagnostic criteria, then tests can be ordered. Cerebrospinal fluid analysis should be performed to confirm a GBS diagnosis and to exclude other sources to symptoms. The typical finding with a lumbar puncture in patients with GBS is an elevated cerebrospinal fluid protein with a normal white blood cell count (8,9). This finding is called albuminocytologic dissociation (7-9). Similarly, electromyography and nerve conduction studies are performed to support a diagnosis and provide prognostic information regarding GBS. On electromyography, a sural sparing pattern, when noted, also reinforces the suspicion for GBS since this finding is usually not observed in length-dependent neuropathies (9,10). Other diagnostic tests to rule out other causes of polyneuropathy include a complete blood count test, a comprehensive metabolic panel, an erythrocyte sedimentation rate test, and a serum glucose test. Diagnostic imaging is reserved for atypical symptoms, such as prominent early bowel or bladder dysfunction, and/or those who present with a heightened sensory level loss (8).

Treating GBS is based on its clinical severity and disease presentation. Baseline neurologic, respiratory, and hemodynamic monitoring should be performed to assess the disease's progression and clinical deterioration. Ventilation status with forced vital capacity, respiratory rate, and maximal expiratory pressure should be measured to access ventilatory function (9). If patients present with signs of a rapid decline in respiratory function, then Intensive Care Unit admission is warranted without delay in intubation. Symptomatic treatment is recommended.

Hypotension can be treated with intravenous fluids or phenylephrine. Hypertension can be treated with labetalol, nicardipine or nitroprusside. Serious or life-threatening cardiac arrhythmias, including atrioventricular block and asystole, can occur with GBS and may require intervention with administration of atropine or cardiac pacing (9). Intravenous immunoglobulin (IVIG) or plasma exchange (PLEX) is indicated for patients who are nonambulatory and unable to walk 10 meters independently within 4 weeks of neuropathic symptom onset. Available evidence suggests that the efficacy of IVIG is like that of PLEX (9). One beneficial advantage of IVIG is that it is easier to administer, and therefore treatment can be implemented quicker with fewer adverse effects (7,8).

We obtained telephone consent from our patient to present this case report.

CASE PRESENTATION

A 70-year-old man with a past medical history of spinal stenosis status post an anterior cervical discectomy and fusion and sacroiliac joint (SIJ) dysfunction presented with imbalance, weakness, and difficulty walking after his SIJ fusion. He initially presented with chronic sacroiliac pain that failed conservative measures. Given significant, but unsustained, relief (> 75% for 2 weeks) of his axial low back pain after SIJ injections, he underwent SIJ fusion (Figs. 1 and 2).

At his postoperative visit a week later, he reported numbness and tingling in his hands and feet with subjective fevers and chills. He denied any recent infections, vaccinations, or sick contacts. One week later, the weakness progressively worsened, requiring a walker.

The patient called the clinic and was instructed to go to the emergency department. He was evaluated and admitted by the neurology service. He was intermittently hypertensive; otherwise, all other vital signs were within normal limits. On physical examination the cranial nerves were intact. By the time the neurology service evaluated him his weakness had progressed to his upper extremities. The strength of his upper and lower extremities was 3 out of 5 proximally and 4 out of 5 distally. His sensation to light touch and pinprick was intact throughout. His reflexes were 1+ symmetrically.

He had extreme difficulty standing up from a seated position and was not able to walk. He underwent a series of lab testing, including but not limited to, a complete blood count, a comprehensive metabolic panel, B12 level, folate level, human immunodeficiency virus test, and thyroid-stimulating hormone test; all were within normal limits, thus ruling out other potential causes of his polyneuropathies.

Magnetic resonance imaging of the brain and spine demonstrated no acute infarct or intracranial hemorrhage and no critical narrowing of the spinal canal at any level as well as no suspicious marrow lesions. Electromyography (Figs. 3 and 4) demonstrated sural sparing,

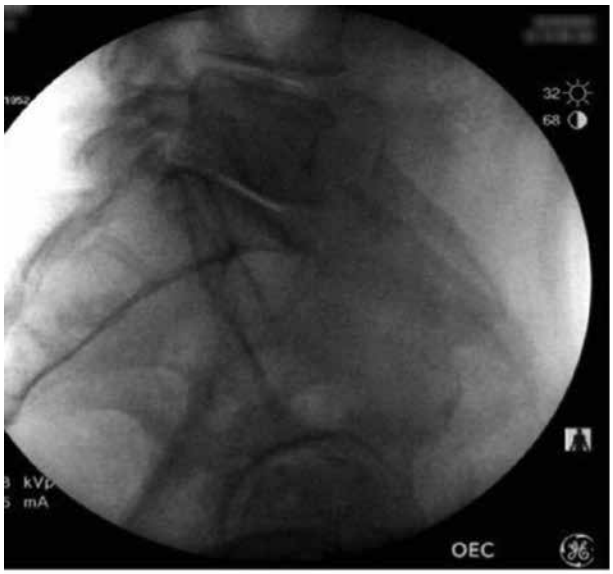

Fig. 1. Lateral hip x-ray of right sacroiliac joint bone graft placement.

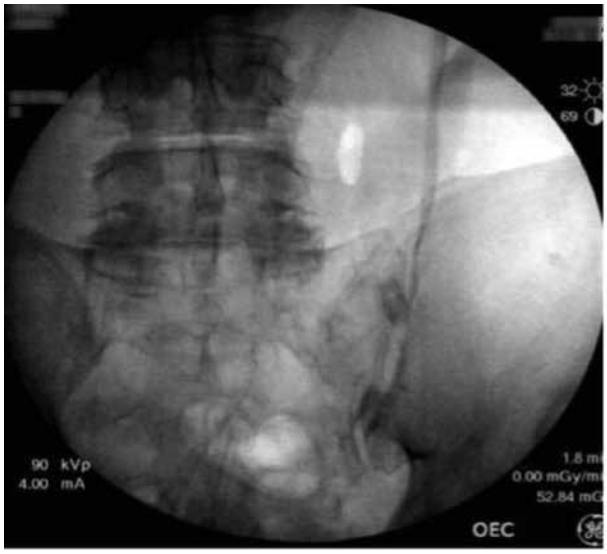

Fig. 2. Posterior hip x-ray of right sacroiliac joint bone graft placement.

EMG

EMG Summary Table											
			Spontaneous					MUAP			Recruitment
Muscle	Nerve	Roots	IA	Fib	PSW	Fasc	H.F.	Amp	Dur	PPP	Pattern
R. Tibialis anterior	Deep peroneal (Fibular)	L4-L5	N	None	None	None	None	N	N	N	*Red (mild)*
R. Gastrocnemius (Medial head)	Tibial	S1-S2	N	None	None	None	None	*1+*	*1+*	*1+*	*Red (moderate)*
R. Vastus medialis	Femoral	L2-L4	N	None	None	None	None	*1+*	*2+*	N	*Red (moderate)*
R. Deltoid	Axillary	C5-C6	N	None	None	None	None	*1+*	*1+*	N	*Red (moderate)*
R. Triceps brachii	Radial	C6-C8	N	None	None	None	None	N	N	N	*Red (mild)*
R. First dorsal interosseous	Ulnar	C8-T1	N	None	None	None	None	N	N	N	*Red (mild)*

Fig. 3. Electromyograph demonstrating R median, ulnar and tibial nerve latency.

prolonged distal motor latencies and reduced nerve conduction velocities. These findings are supportive of acute inflammatory demyelinating polyradiculopathy also known as GBS.

He was placed on continuous electrocardiogram telemetry and pulse oximetry. His maximal inspiratory pressure and negative inspiratory pressure were measured every 4 hours via respiratory pressure manometry.

For treatment, he was started on IVIG therapy at 0.4 g/kg/d. On length of stay 3, he started developing facial droop and difficulty swallowing. This was consistent with his bilateral facial palsies. We began plasmapheresis treatment due to his clinical worsening. He received 5 treatments of PLEX and demonstrated clinical improvement with dysphagia and ambulation. He tolerated PLEX, with no symptoms of hypotension, arrythmias or paresthesia secondary to treatment. He was admitted to an acute inpatient rehabilitation facility. At the acute rehabilitation facility, he demonstrated significant improvement in physical, occupational, and speech language therapy goals. Twenty-eight days following admission he was discharged with intermittent supervision of his activities of daily living as well as outpatient physical and occupational therapy.

Nerve / Sites	Muscle	Latency ms	Amplitude mV	Rel Amp %	Segments	Distance cm	Velocity m/s	Stim Int	Stim Dur ms
R Median - APB									
Wrist	APB	***9.64***	***0.7***	100	Wrist - APB	7		100mA	0.2
Ref.		≤4.40	≥4.0		Ref.				
Elbow	APB	14.38	0.6	87	Elbow - Wrist	22	***46***	100mA	0.2
Ref.					Ref.		≥49		
R Ulnar - ADM									
Wrist	ADM	***4.69***	***2.6***	100	Wrist - ADM	7		84.3mA	0.1
Ref.		≤3.60	≥5.0		Ref.				
B.Elbow	ADM	8.44	2.2	86.3	B.Elbow - Wrist	20	53	100mA	0.1
Ref.					Ref.		≥49		
A.Elbow	ADM	10.73	1.7	67	A.Elbow - B.Elbow	8.5	***37***	100mA	0.1
Ref.					Ref.		≥49		
R Peroneal - EDB									
Ankle	EDB	5.63	3.7	100	Ankle - EDB	8		100mA	0.1
Ref.		≤6.20	≥2.0		Ref.				
Fib head	EDB	15.42	3.0	81.2	Fib head - Ankle	31.5	***32***	100mA	0.1
Ref.					Ref.		≥39		
Pop fossa	EDB	17.66	2.9	80.3	Pop fossa - Fib head	9	40	100mA	0.1
Ref.					Ref.		≥39		
R Tibial - AH									
Ankle	AH	***8.54***	***3.0***	100	Ankle - AH	7		100mA	0.3
Ref.		≤6.00	≥3.0		Ref.				
Pop fossa	AH	18.28	2.0	67.9	Pop fossa - Ankle	34	***35***	100mA	0.3
Ref.					Ref.		≥39		

F Wave

Nerve	F Lat ms	M Lat ms	F-M Lat ms
R Ulnar - ADM	***35.0***	5.1	29.9
R Peroneal - EDB	24.8	6.3	18.5

Fig. 4. Electromyograph results demonstrating R median, ulnar and tibial nerve latency.

DISCUSSION

While an uncommon trigger, a small percentage of patients develop GBS after other triggering events, such as surgical procedures. A retrospective case series from Mayo Clinic reported surgery antedated GBS in 9.1% of patients diagnosed with GBS January 1995 through June 2014 (9). Gensick, et al (11) reported that the relative risk of contracting GBS was 13.1 times higher in patients who had surgery compared to the general population (11). The median postsurgery onset of symptoms was 15 days (10,12). The main types of surgeries preceding GBS were gastrointestinal, neurosurgical, and cardiac (9,13,14). In the study by Aranson et al (10), an association was found with malignancy and comorbid autoimmune disease and postsurgical GBS. There have been minimal case reports of GBS after pain procedures. There was a case report of acute GBS after lumbar epidural steroid injection (15), in that study, the cause of GBS cannot be directly tied to the procedure as the patient presented with a gastrointestinal infection prior to injection.

GBS's pathophysiology in patients postsurgery is not well understood, but there are proposed mechanisms that could explain the presentation. One mechanism, described by Steiner, et al (15), is that the reaction between local anesthetics and peripheral nerve myelin or local trauma to the nerve roots could trigger a cascade of immunological events that result in demyelinating neuropathy. SIJ innervation is complex. The SIJ's innervation is from the ventral rami of L4 and L5, the superior gluteal nerve, and the dorsal rami of L5-S2 (16). The nerve supply can vary between individuals. Localized trauma to the nerve roots with SIJ bone graft placement

leading to a demyelinating immunological cascade is one hypothesized mechanism (17).

Furthermore, the lipid soluble local anesthetics, containing an aromatic ring, allows them to pass through the myelin of the peripheral nerves and induce damage to the nerves through the interaction between the anesthetics and the myelin of the peripheral nerves (16,18). Further research is recommended to investigate this hypothesized correlation between local anesthetics and myelin.

Another proposed mechanism is through activating the endocrine stress systems. Surgery activates this stress response which leads to transient immunosuppression by altering cellular/humoral immunity. promoting autoantibodies to attack peripheral nerves (18,19). Exposure to certain viruses, bacteria, vaccines, or even myelin itself may sensitize the body's immune systems, which can trigger an autoimmune response on the nervous system (19,20).

In our case, our patient presented with mild symptoms of progressive upper and lower extremity weakness that progressed to facial muscle weakness. He presented with symptoms about 14 days after his procedure with a preceding trigger. He did not respond to IVIG therapy, was transitioned to PLEX, and demonstrated recovery after acute inpatient rehabilitation hospitalization. There have been previous case reports of acute GBS after chronic pain procedures; however, this is the first with SIJ fusion. As previously mentioned, 2 hypothesized mechanisms of this clinical presentation are interactions between the anesthetics and peripheral myelin, and the modulation of the immune system secondary to surgical stress. While case series and reports demonstrate postsurgical GBS after cardiac, neurosurgical, and gastrointestinal procedures, there is a similar risk factor associated with pain procedures as these procedures can promote a similar stress response (9,13,14).

CONCLUSION

GBS is an acute immune-mediated polyneuropathy that is a common cause of acute acquired weakness. While GBS has an overall positive prognosis, it can present with dangerous complications that can lead to death. We report a case of a patient who presented with progressive right upper extremity and right lower extremity weakness with electromyography studies consistent with GBS after SIJ fusion. To our knowledge, this is the first case of GBS after SIJ fusion. There are hypothesized mechanisms on the cause of GBS after SIJ fusion; however, we cannot definitively conclude that the procedure is the cause of GBS in this case; further investigation is warranted. Surgery is a potential independent risk factor for the occurrence of GBS. This case report reinforces the need for providers to be aware of this potentially reversible condition. While it is a rare surgical complication, early diagnosis and prompt treatment are imperative to reduce mortality and improve outcomes.

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