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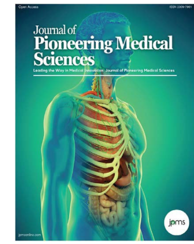

Hip Heterotopic Ossification and Pellegrini-Stieda Disease After Guillain-Barré Syndrome: A Rare Case Report

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Abstract: Heterotopic ossification (HO) is the formation of extraskeletal mature bone in periarticular soft tissues, whereas Pellegrini-Stieda disease (PSD) refers to ossification or calcification adjacent to the proximal attachment of the medial collateral ligament. Both entities are uncommon in Guillain-Barré syndrome (GBS), and their coexistence in the same patient is exceptionally rare. We report a 27-year-old man with prior severe GBS who required a 4-month intensive care unit stay, mechanical ventilation, and prolonged immobilization, and who later presented with left hip pain, reduced hip range of motion, and subsequent right medial knee pain during rehabilitation. Radiography and non-enhanced computed tomography confirmed mature HO around the left hip, while knee radiography demonstrated a characteristic ossified fragment adjacent to the proximal medial collateral ligament, consistent with PSD. Serum calcium and phosphate were normal, whereas alkaline phosphatase was mildly elevated initially and normalized on follow-up, supporting maturation of the ossific process. This single case highlights that persistent pain or stiffness during rehabilitation after severe GBS should prompt early imaging evaluation for HO or PSD so that conservative management and timely surgical referral, when needed, are not delayed.

Key Words: Heterotopic Ossification, Pellegrini-Stieda Disease, Guillain-Barré Syndrome, Rehabilitation, Case Report

INTRODUCTION

Heterotopic ossification (HO) is the formation of mature lamellar bone in periarticular soft tissues and skeletal muscle. The pathophysiology is multifactorial and is thought to involve tissue injury, local inflammation, mesenchymal cell differentiation, and abnormal osteogenic signaling. In neurogenic settings, prolonged immobilization, critical illness, tissue hypoxia, and aggressive rehabilitation may further increase risk. The incidence of neurogenic HO is reported to be approximately 10%-25% in major central neurologic injuries, but it is much less frequently described after peripheral neuropathies such as Guillain-Barré syndrome (GBS) [1-4].

Pellegrini-Stieda disease (PSD) refers to ossification or calcification near the femoral attachment of the medial collateral ligament. Although classically regarded as post-traumatic, medial femoral condylar ossification has radiologic differential diagnoses, including enthesopathic calcification, old avulsion injury, and calcified soft-tissue lesions, so clinicoradiologic correlation is important when assigning the diagnosis [4].

GBS is an acute autoimmune polyradiculoneuropathy characterized most commonly by progressive symmetrical weakness, areflexia, and variable sensory or autonomic involvement [5,6]. Severe cases may require prolonged intensive care, mechanical ventilation, and extended rehabilitation, thereby creating a clinical context in which delayed musculoskeletal complications can be overlooked.

This report describes a rare combination of hip HO and PSD in a young man recovering from severe GBS. The purpose of this case report is to emphasize early clinical suspicion, appropriate imaging, functional implications, and the relevance of ICU-related immobility and rehabilitation exposure when unexplained pain or joint stiffness appears during recovery. This case report was prepared with attention to CARE reporting principles.

CASE REPORT

A 27-year-old man presented to the orthopedic clinic with left hip pain and restricted hip rotation. He had been diagnosed with Guillain-Barré syndrome (axonal polyneuropathy variant) more than two years earlier. During the acute illness he required admission to the intensive care unit for 4 months, had quadriplegia for several months, received plasma exchange and intravenous immunoglobulin

therapy, and was later enrolled in prolonged physiotherapy. At presentation, the upper limbs had normal strength and sensation. The lower limbs showed residual weakness, graded as 4/5 proximally and 2-3/5 distally. Left hip range of motion was restricted, with internal and external rotation approximately 25-30 degrees, flexion 85 degrees, and extension 25 degrees. These limitations interfered with daily activities and slowed rehabilitation progress. Pain severity had not been recorded on a formal numeric scale at the original visit.

Plain radiography and non-enhanced computed tomography confirmed heterotopic ossification within the soft tissues around the left hip joint (Figures 1 and 2).

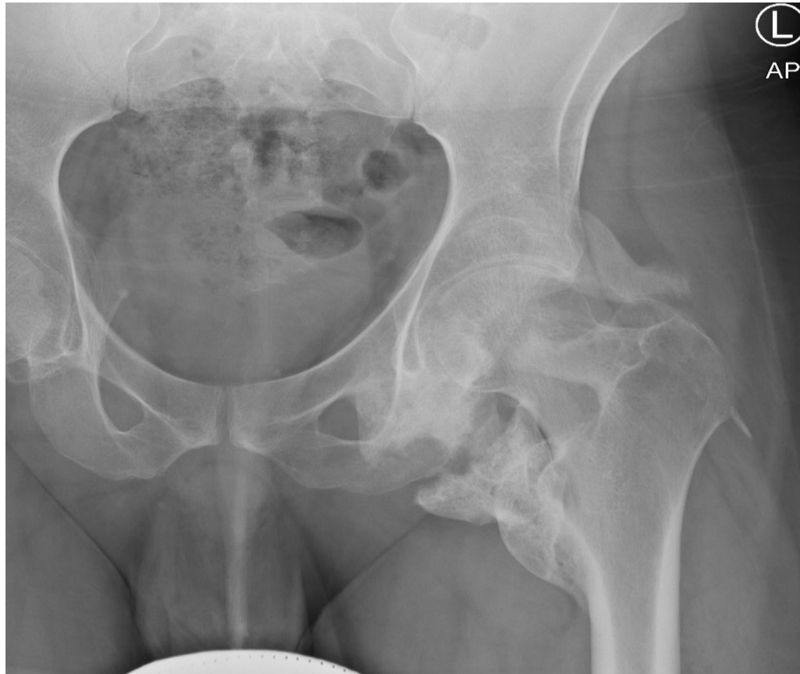

Figure 1: Anteroposterior Radiograph of the Left Hip Demonstrating Periarticular Soft-Tissue Ossification Consistent with Heterotopic Ossification

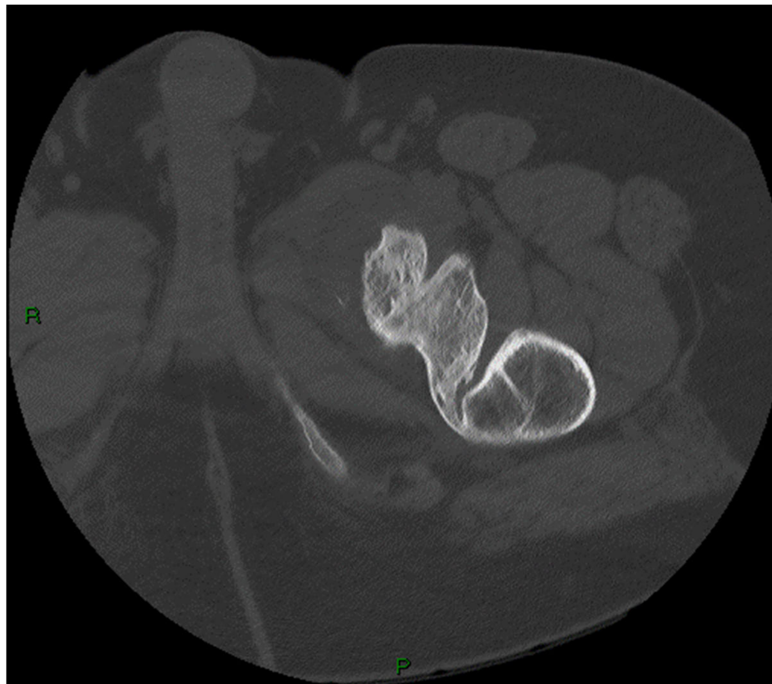

Figure 2: Selected Non-Enhanced Axial CT Image showing Lamellated Mature Bone Formation within the Proximal Left Adductor Musculature, Confirming Heterotopic Ossification

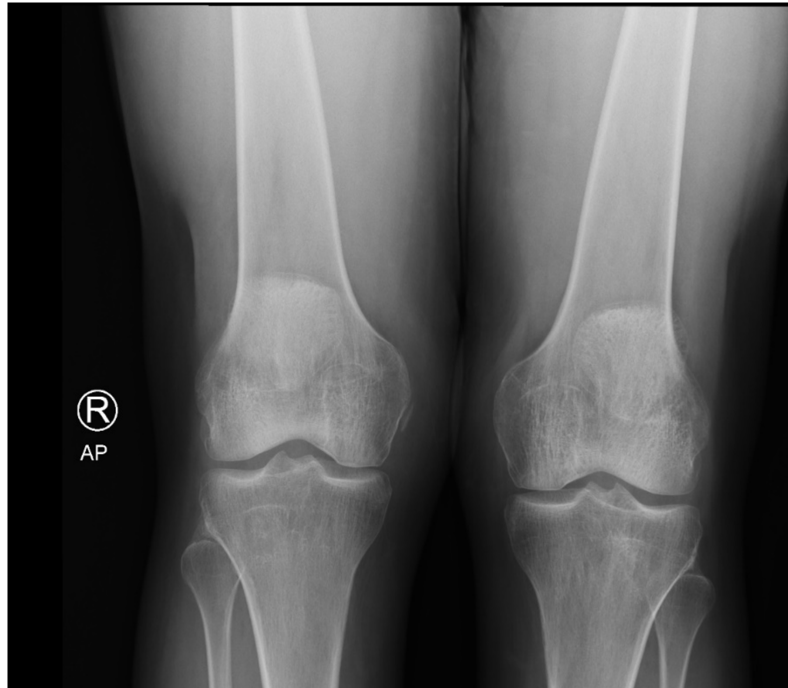

Figure 3: Anteroposterior Radiograph of Both Knees Showing a Small Ossified Fragment Adjacent to the Proximal Attachment of the Right Medial Collateral Ligament, Compatible with a Pellegrini-Stieda Lesion

Table 1: Clinical Timeline

Time point	Clinical events	Key findings / actions
Acute GBS phase	ICU admission for 4 months	Mechanical ventilation, quadriplegia for several months, plasma exchange, IVIG.
Recovery phase	Prolonged rehabilitation	Gradual neurologic recovery during physiotherapy.
Orthopedic presentation	Left hip pain and reduced motion	X-ray and CT confirmed left hip HO; ALP 126.67 U/L with normal calcium/phosphate.
10-month follow-up	Persistent recovery with new knee symptoms	ALP normalized to 111.76 U/L; knee radiograph showed right-sided PSD lesion.

At the time of imaging, serum calcium and phosphate levels were within the normal range, whereas serum alkaline phosphatase was mildly elevated at 126.67 U/L. No surgical intervention was undertaken, and the patient continued supervised physiotherapy and conservative management. Ten months later, serum alkaline phosphatase had returned to the normal range (111.76 U/L), suggesting maturation of the ossific process.

At follow-up, the patient also reported right medial knee pain during daily activities. Examination showed mild quadriceps atrophy without major restriction of knee motion or instability. Radiography of the knee demonstrated a small ossified fragment adjacent to the proximal attachment of the medial collateral ligament, in keeping with a Pellegrini-Stieda lesion (Figure 3). The patient denied prior knee trauma and had not previously reported knee symptoms, making a post-rehabilitation neurogenic association plausible, although definite causation cannot be proven in a single case report (Table 1).

DISCUSSION

Guillain-Barré syndrome is an immune-mediated inflammatory polyradiculoneuropathy that may involve motor, sensory, and autonomic fibers and remains the most common cause of rapidly progressive flaccid paralysis worldwide [5,6]. In severe cases, intensive care admission, assisted ventilation, and prolonged immobilization create a biologically plausible setting for neurogenic heterotopic ossification. Accordingly, this case is best interpreted through the neuro-ICU and rehabilitation pathway rather than through trauma alone.

HO represents ectopic mature bone formation in soft tissues. Proposed mechanisms include local inflammation, tissue hypoxia, osteogenic signaling, and mesenchymal stem-cell differentiation after neurologic injury [7]. In the present patient, prolonged ICU stay, mechanical ventilation, severe paresis, and extended immobility were the main risk indicators. The hip is the most frequently involved joint in previously reported GBS-related HO cases, which is consistent with our findings [8-11].

Published literature suggests that HO after GBS is uncommon but clinically important because it can be mistaken for contracture, generalized deconditioning, or pain related to rehabilitation. In our review table, most reported cases were associated with severe neurologic disease, intensive care treatment, and prolonged immobilization. Imaging modalities used across published cases have included plain radiography, CT, MRI, bone

Table 2: Reported Cases of Heterotopic Ossification in Patients with Guillain-Barré Syndrome

Study	# of cases	Affected joint
Ryu SR *et al.*	1	Hip
Zeilig G *et al.* [8]	4	Hip (all cases)
Vaishya R *et al.* [2]	1	Knee
Shawgi M	1	Hip
Kerdoncuff V *et al.* [10]	3	Shoulder, knee and hip
Ohnmar H *et al.*	1	Shoulder, elbow and hip
Bernard V	1	Hand
Hung JCC *et al.*	1	Hip
Ploumis A *et al.*	1	Hip
Coppens E *et al.*	1	Hip
Nalbantoglu M *et al.* [9]	1	Hip
Abid H *et al.*	1	Hip
Gulati R *et al.* [4]	2	Hip
Sivakumaran D *et al.* [11]	1	Hip

Table 3: Reported Cases of Pellegrini-Stieda Disease in Patients with Spinal Cord or Brain Insults

Study	No. of cases	Type of injury
Altschuler EL *et al.* [13]	1	Spinal cord
Yildiz N *et al.* [14]	1	Brain
Yavuz F *et al.*	1	Cauda equina syndrome
10-month follow-up	1	Spinal cord

scintigraphy, and SPECT/CT. In our patient, plain radiography and CT were sufficient to demonstrate mature periarticular ossification, while the biochemical pattern of initially elevated and later normalized alkaline phosphatase was also compatible with maturation of the lesion [8-13].

HO is considered a rare complication of central nervous system disorders. It is increasingly reported in traumatic brain and spinal cord injuries. Peripheral nerves disorders, including GBS is also linked to HO formation. The incident of HO in patients with GBS can reach up to 6% [9]. We found in the literature 20 cases of HO reported as a complication of GBS (Table 2). The most common affected joint is the hip (18 out of 20 reported cases). The mean age of heterotopic ossification in patients with GBS as per reported cases was 42 years. The most common risk factors of HO are GBS patients were mechanical ventilation/ICU stay and prolonged immobilizations which were seen in almost all reported cases [4, 10]. Other risk factors of HO in GBS in reported cases were respiratory failure/hypoxic conditions and encephalopathy. In our case, the risk factors of HO were mechanical ventilation/ICU stay and prolonged immobilizations. In the reported cases, several radiological modalities were used for diagnoses, including plain radiograph, non-enhanced CT, 3-phase bone scan, MRI and SPECT/CT. Simple radiograph and non-enhanced CT were utilized in our case. Lab investigations used in the reported cases were serum calcium, phosphate and alkaline phosphatase. Serum alkaline phosphatase level can be normal initially but will gradually increases with the new bone formation and returns to normal as the bone becomes mature [11].

Management of neurogenic HO depends on symptoms, maturity of ossification, and functional limitation. Conservative management may include careful rehabilitation, non-steroidal anti-inflammatory drugs, or other prophylactic measures in selected settings, whereas surgical excision is generally reserved for persistent pain, progressive functional compromise, neurovascular compression, or failure of conservative care. In this case, the patient was managed conservatively because the lesion appeared mature, symptoms were tolerable, and no acute neurovascular compromise was documented.

PSD is another periarticular ossific disorder that is usually described after trauma at the femoral attachment of the medial collateral ligament. However, it has occasionally been reported in patients with spinal cord or brain injuries, suggesting that immobilization, tissue hypoxia, altered sympathetic activity, and repetitive microtrauma during rehabilitation may also contribute [12,14-15]. In the present patient, the radiographic location and absence of a prior trauma history supported the diagnosis of PSD. Nevertheless, the differential diagnosis includes enthesopathic ossification, remote avulsion injury, and calcified soft-tissue lesions, so the diagnosis should remain clinicoradiologic rather than purely label-based.

Pellegrini-Stieda disease (PSD) is the other entity of soft tissue ossification/calcification seen in our patient. PSD is defined as ossification of the medial collateral ligament (MCL) of the knee (mainly at it its proximal femoral attachment). It is presumed to be a result of mechanical avulsion injury of the MCL femoral attachment. Very few reports (a total of 4 cases) were found in the literature of PSD in patients with brain/spinal cord insults (Table 3). We believe that our case is the first reported case of PSD as a complication of GBS. This report has limitations. It is a single case report, so no causal relationship can be proven. Exact ventilation duration beyond the documented ICU stay was not available in the original chart summary, pain scores were not formally recorded, and no dedicated follow-up imaging of the knee was available. Similarly, the absence of a structured functional outcome scale limits quantitative assessment. Despite these limitations, the case remains educational because it illustrates dual-site ossification after severe GBS and highlights the importance of early imaging when rehabilitation progress is unexpectedly limited.

Several risk factors were linked to the development of PSD in patients with nervous system insults including tissue hypoxia, hypercalcemia, changes in sympathetic nerve activity and prolonged immobilization [12]. However, some reports from physiatrists have suggested micro-repetitive trauma from therapeutic manipulation of a restricted knee joint to be possible etiologies of Pellegrini-Stieda Disease [14-16].

CONCLUSION

Hip HO and PSD can occur as late musculoskeletal complications after severe Guillain-Barré syndrome, particularly in patients with prolonged ICU stay, assisted ventilation, and extended immobilization. Persistent pain, stiffness, or failure to progress during rehabilitation should prompt timely radiographic assessment so that HO or PSD is

not mistaken for contracture alone. Management should be individualized, with conservative treatment for stable lesions and surgical referral considered when pain, functional decline, or neurovascular compromise persists.

Conflicts of Interest

The author declares that there is no conflict of interest.

Informed Consent

Written informed consent was obtained from the patient for publication of the clinical data and radiological images. A copy of the consent is available on request.

Funding

No financial support has been received for this work.

Ethics and Reporting

This manuscript describes a single anonymized case managed during routine clinical care. Written informed consent for publication of the clinical information and radiological images was obtained from the patient. The report was prepared in line with CARE guidance for case reports [15,16].

REFERENCES

[1] Garland, D.E., "A clinical perspective on common forms of acquired heterotopic ossification." *Clinical Orthopaedics and Related Research*, no. 263, 1991, pp. 13–29.
[2] Vaishya, R. *et al.* "Heterotopic ossification circumferentia articularis (HOCA) of both knee joints after Guillain-Barré syndrome." *Cureus*, vol. 8, no. 2, 2016, e480.
[3] Gulati, R. *et al.* "Heterotopic ossification in Guillain-Barré syndrome – A dual case report." *Journal of Clinical and Diagnostic Research*, vol. 15, no. 1, 2021, pp. YD01–YD04.
[4] Mendes, L.F.A. *et al.* "Pellegrini-Stieda disease: a heterogeneous disorder not synonymous with ossification/calcification of the tibial collateral ligament – anatomic and imaging investigation." *Skeletal Radiology*, vol. 35, no. 12, 2006, pp. 916–922.
[5] Yuki, N. and Hartung, H.P., "Guillain-Barré syndrome." *New England Journal of Medicine*, vol. 366, no. 24, 2012, pp. 2294–2304.
[6] van Doorn, P.A. *et al.* "Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome." *Lancet Neurology*, vol. 7, no. 10, 2008, pp. 939–950.
[7] Huang, H. *et al.* "Relationship between heterotopic ossification and traumatic brain injury: Why severe traumatic brain injury increases the risk of heterotopic ossification." *Journal of Orthopaedic Translation*, vol. 12, 2018, pp. 16–25.
[8] Zeilig, G. *et al.* "Heterotopic ossification in Guillain-Barré syndrome: Incidence and effects on functional outcome with long-term follow-up." *Archives of Physical Medicine and Rehabilitation*, vol. 87, no. 1, 2006, pp. 92–95.
[9] Nalbantoglu, M. *et al.* "Neurogenic heterotopic ossification in Guillain-Barré syndrome: A rare case report." *Journal of Musculoskeletal and Neuronal Interactions*, vol. 20, no. 1, 2020, pp. 160–164.
[10] Kerdoncuff, V. *et al.* "Paraostéoarthropathies dans le syndrome de Guillain-Barré." *Annales de Réadaptation et de Médecine Physique*, vol. 45, no. 5, 2002, pp. 198–203.
[11] Sivakumaran, D. *et al.* "Myositis ossificans of the hip joint causing sciatic nerve palsy following Guillain-Barré syndrome: A case report." *International Journal of Surgery Case Reports*, vol. 84, 2021, 106110.
[12] Ploumis, A. *et al.* "Pellegrini-Stieda disease in a severely injured patient with spinal cord injury." *Journal of Musculoskeletal and Neuronal Interactions*, vol. 23, no. 3, 2023, pp. 365–367.
[13] Aldhilan, M.M. *et al.* "Heterotopic ossification post-Guillain-Barre syndrome in Saudi Arabia: A case report." *Annals of Medicine and Surgery*, vol. 86, no. 9, 2024, pp. 4981–4985.
[14] Altschuler, E.L. and Bryce, T.N., "Pellegrini-Stieda syndrome." *New England Journal of Medicine*, vol. 354, no. 1, 2006, e1.
[15] Yildiz, N. *et al.* "Pellegrini-Stieda disease in traumatic brain injury rehabilitation." *American Journal of Physical Medicine and Rehabilitation*, vol. 87, no. 6, 2008, p. 514.
[16] Riley, D.S. *et al.* "CARE guidelines for case reports: explanation and elaboration document." *Journal of Clinical Epidemiology*, vol. 89, 2017, pp. 218–235.