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Radiology Section

Contrast MRI findings of Guillain-Barré Syndrome

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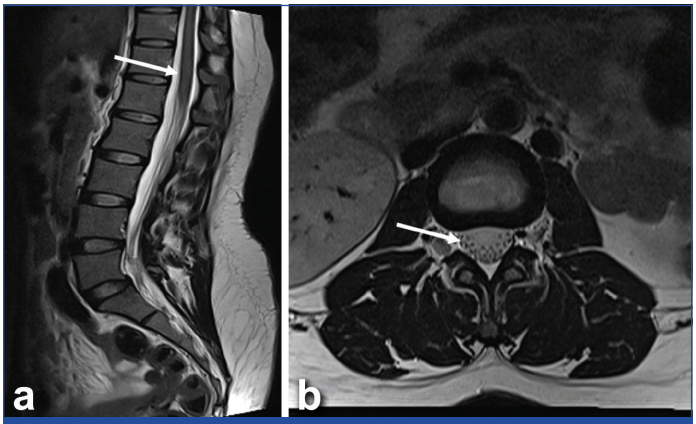
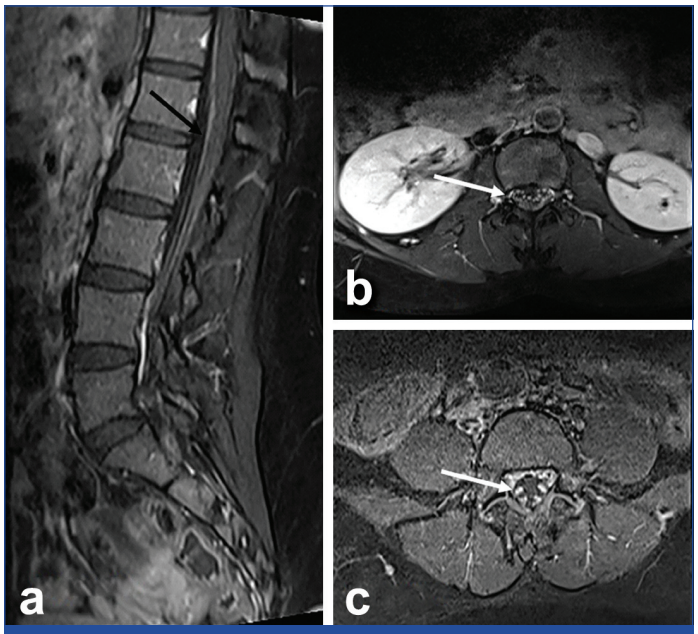
A 28-year-old female patient presented with complaints of progressive bilateral lower limb weakness for the past seven days, which had increased to involve bilateral upper limbs for the past two days. She also presented with difficulty in speaking for five days, associated with exhaustion after speaking more than 10 words. The patient also complained of diarrhoea for four days with bladder disturbances. Difficulty in initiation of micturition with incomplete evacuation of urine associated with dribbling was noted. The patient had a history of high-grade fever three weeks prior due to respiratory illness, which subsided with medications in three days. The patient also reported a history of tingling sensation and paresthesia in both lower limbs. There was no history of bronchial asthma, tuberculosis, cardiovascular abnormalities, chronic kidney disease, migraine, trauma, epilepsy, or transient ischaemic attacks.

On clinical examination, the patient was conscious, oriented, and afebrile. Deep tendon reflexes were absent. No signs of cyanosis, icterus, oedema, pallor, rash, or eschar were found. The patient had reduced finger grip in both toes and difficulty in squatting, suggestive of both proximal and distal muscle weakness in bilateral lower limbs. There was no history of cranial nerve involvement. The patient was normotensive with normal blood sugar levels. Clinically, a diagnosis of Guillain-Barre Syndrome (GBS) was considered. Blood and urine investigations were normal. A 24-hour urinary protein test was within normal limits. Urine culture and blood culture were negative. Cerebrospinal Fluid (CSF) analysis was normal with no evidence of albumin-cytological dissociation. Electrophysiological and nerve conduction studies were not classical of GBS and were inconclusive. Although GBS was suspected clinically, the CSF and nerve conduction studies did not contribute to the diagnosis.

Hence, the patient underwent contrast Magnetic Resonance Imaging (MRI) of the whole spine and brain for further evaluation and to rule out other causes. On plain study, the cord revealed normal signal intensity with normal cauda equina nerve roots without clumping [Table/Fig-1a,b]. Postcontrast administration showed smooth enhancement of the pial surface of the conus medullaris with smooth thickening and enhancement of all the cauda equina nerve roots [Table/Fig-2a-c]. Although the differential diagnosis for diffuse cauda equina nerve root enhancement were many, such as GBS, spinal meningitis or arachnoiditis, and chronic inflammatory demyelinating polyneuropathy, most of these were ruled out by correlating with clinical history, and GBS was considered an appropriate diagnosis. The patient then received Immunoglobulin therapy, with five daily infusions for a total dose of 1 g/kg/wt, resulting in the reversal of symptoms subsequently. The patient is presently on follow-up and is doing well.



[Table/Fig-1a,b]: Sagittal and axial T2 Plain MRI of lumbosacral spine shows normal conus medullaris and cauda equina nerve roots (white arrows).



[Table/Fig-2a-c]: Sagittal, axial postcontrast MR images of the lumbosacral spine show the presence of enhancement along conus (black arrow) and all the cauda equina nerve roots which appear smoothly thickened (white arrows).

DISCUSSION

The GBS has been defined as a spectrum of clinical syndromes that manifest as acute inflammatory polyradiculoneuropathy and are associated with weakness. It is typically described as acute, progressive, and symmetrical [1]. The most typical presentation in a patient with GBS is that of Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), which presents 2-4 weeks following a relatively benign respiratory or gastrointestinal illness or vaccination [2]. The present case had a history of fever three weeks prior due to a respiratory illness. However, if bladder involvement is a prominent feature and appears early in the course of the disease, an alternate diagnosis should be considered. More worrying respiratory symptoms include shortness of breath and dyspnoea on exertion, with up to one third of the patients needing ventilatory support throughout the course of the disease due to respiratory failure. The patient had only ascending motor paralysis with no evidence of cranial nerve or respiratory muscle involvement. Bladder involvement, although

present in the present case, was transient and mild, resolving after treatment. Although MR findings are mostly complementary, MRI is preferred in cases with paraesthesia, extremity weakness, or Gastrointestinal (GI)/Genitourinary (GU) dysfunction [3]. MRI findings of GBS report marked enhancement of the thickened nerve roots in the conus medullaris and cauda equina [1]. However, precontrast studies are mostly insignificant and appear normal, as in the present case [4]. Byun WM et al., suggested that enhancement solely of the ventral roots was strongly suggestive of GBS [5]. In the present case, both dorsal and ventral nerve roots showed enhancement, which may explain the sensory symptoms experienced by the patient. The contrast MRI findings of the present case are similar to previously reported cases by Ding X et al., and Fontes CA et al., where there was enhancement of both dorsal and ventral roots of the cauda equina, similar to the present case [6,7]. Our patient had normal imaging findings before contrast. However, on postcontrast imaging, marked enhancement of all cauda equina nerve roots with smooth thickening was noted.

REFERENCES

[1] Alkan O, Yildirim T, Tokmak N, Tan M. Spinal MRI findings of Guillain-Barré Syndrome. J Radiol Case Rep. 2009;3(3):25-28.
[2] Lewis RA. Chronic inflammatory demyelinating polyneuropathy. Neurol Clin. 2007;25(1):71-87. Doi: 10.1016/j.ncl.2006.11.003.
[3] Zapadka M. Diffuse cauda equina nerve root enhancement. J Am Osteopath Coll Radiol. 2012;1(1):34-37.
[4] Baran GA, Sowell MK, Sharp GB, Glasier CM. MR findings in a child with Guillain-Barré syndrome. AJR Am J Roentgenol. 1993;161(1):161-63.
[5] Byun WM, Park WK, Park BH, Ahn SH, Hwang MS, Chang JC. Guillain-Barré syndrome: MR imaging findings of the spine in eight patients. Radiology. 1998;208(1):137-41.
[6] Ding X, Jiang H, Hu X, Ren H, Cai H. Guillain-Barré Syndrome and low back pain: Two cases and literature review. Open Med (Wars). 2018;13:503-08.
[7] Fontes CA, Dos Santos AA, Marchiori E. Magnetic resonance imaging findings in Guillain-Barré syndrome caused by Zika virus infection. Neuroradiology. 2016;58(8):837-38.

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