A 40-year-old female presented with 10 days history of bilateral lower limb weakness, numbness difficulty walking and loss of balance with intact sensation in lower limb and upper limb. There was no history of fever, diarrhoea, vomiting or abdominal pain. There was no recent vaccination. Bilateral lower limb weakness was followed by left sided facial palsy, on admission she appeared alert, oriented and non toxic. Vital signs were normal. Facial palsy was noticed after 3 days of onset of areflexia and hypotonia. On examination, the patient was found to have areflexia and hypotonia in bilateral lower limbs. There was no neck stiffness, no evidence of meningismus. There was no history of headache or change in sensorium and no evidence of cranial nerve palsy. The patient had a history of intramuscular injection of vaccines around 2 weeks prior to the onset of symptoms. The patient was afebrile with normal blood pressure. There was no history of recent infectious illness. A review of past literatures showed controversy regarding early intervention with intravenous immunoglobulin for unilateral facial palsy. Early intervention with intravenous immunoglobulin may benefit these patients.

**DISCUSSION**

GBS in this patient was unusual because it was associated with unilateral facial nerve palsy. Incidence of GBS with unilateral palsy is rare. GBS is characterized by acute inflammatory demyelinating polyneuropathy characterized by progressive bilateral lower limb weakness mediated by autoimmune response commonly triggered by viral or bacterial infections. There were several theories about unilateral facial palsy. It can be caused by antibody-mediated autoimmune responses, inflammatory demyelination which is possible explanation in our patient. There was no evidence of viral or bacterial infection which could have caused the unilateral facial weakness. The patient was treated with IV Ig 1g/kg for 5 days with glucocorticoids improvement started in lower limb within 1 week of treatment.
CONCLUSION
The presence of facial palsy in GBS has also been identified as a potential indicator for anticipating more complicated recovery. GBS should be considered as a possible cause of unilateral peripheral facial palsy. GBS patients with facial nerve palsy may develop bulbar weakness require close monitoring as they are at risk of developing acute respiratory failure and early intervention with IV Ig and glucocorticoid may hasten the recovery.

REFERENCES
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