

SKIN RASH AS EARLY PRESENTATION OF GUILLAIN-BARRÉ SYNDROME*

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Abstract

We report an unusual case of Guillain-Barre syndrome in a 36-year old gentleman, diagnosed based on clinical presentation, CSF analysis and nerve study tests findings, who presented to our department for elective cystoscopy and discovered at the day of surgery to have macular skin rash over the trunk and upper limbs, surgery was postponed. Then and after 12 hours he started to develop the classical manifestations of Guillain-Barre syndrome. Asymptomatic skin rash should carefully be investigated as it could be an early presentation of a serious condition.

Key words: Skin Rash, Syndrome, Surgery, Anesthesia, Paralysis.

Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy disorder that affects the peripheral nervous system usually triggered by an acute infection. The most characteristic symptom is ascending paralysis, weakness beginning in the feet and hands and migrating towards the trunk. It can cause life-threatening complications, particularly if the breathing muscles are affected or if there is dysfunction of the autonomic nervous system¹.

Skin rash has been reported as possible manifestations during the course of the disease but not as the first presenting symptoms^{1,2}. We herein reported a rare case of GBS where skin rash was the earliest clinical presenting symptoms.

Case Report

A 36 year old healthy gentleman admitted to our hospital with left sided colicky loin pain, dysuria and urinary urgency. His review of symptoms, past medical history and drug history were within normal except for mild sore throat of 3 day duration. His physical examination was within normal except for tenderness over the costophrenic angle. Urine analysis showed 2-4 WBC, 2-3 RBC and yellowish discoloration. Other laboratory tests were within normal range except for high ESR and creatinine. He was scheduled for elective cystoscopy to extract urinary tract stone.

At the day of surgery the patient developed generalized erythematous maculopapular non-scaly rash over his back, chest and upper arms, Figure 1, numbness in his hands, and minimal upper extremities weakness. The surgery was cancelled for further evaluation of these symptoms. Twenty four hours later the patient developed ascending muscle weakness and shortness of breath. He transferred to the ICU for closed neurological and respiratory monitoring. Lumbar puncture showed:

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albumin-cytological dissociation, elevated protein level, and increased white blood cell count. Nerve conduction studies revealed prolongation of the upper and lower motor action potential latencies, reduced motor conduction velocities and reduced amplitude. Median and ulnar nerves sensory action potentials were absent. The diagnosis of GBS was reached and he was started on intravenous Immunoglobulin 400 mg/kg, Gabapentin 300 mg twice a day and Clexan 40 mg once daily.

Fig. 1
Erythematous maculopapular Rash over the chest



Patient's level of consciousness and respiratory status deteriorated on the next day, he was Intubated and ventilated with mechanical ventilator. Unfortunately 6-days post intubation he died after he developed acute adult respiratory syndrome secondary to generalized sepsis.

Discussion

GBS is an acute immune-mediated polyneuropathy caused by infection, inflammation, tumors, medications, vaccines and surgery with

incidence worldwide of 0.6–4/100,000 persons/year.

Up to two thirds of patients report an antecedent bacterial or viral illness prior to the onset of neurologic symptoms with *Campylobacter jejuni* being the most commonly isolated pathogen^{3,4}.

Gastrointestinal and upper respiratory tract symptoms can be observed with *Campylobacter jejuni* infections. *Campylobacter jejuni* infections can also have a subclinical course, resulting in patients with no reported infectious symptoms prior to development of GBS. Patients who develop GBS following an antecedent *Campylobacter jejuni* infection often have a more severe course, with rapid progression and a prolonged, incomplete recovery as we believe in our case.

A strong clinical association has been noted between *Campylobacter jejuni* infections and the pure motor and axonal forms of GBS. The virulence of *Campylobacter jejuni* is thought to result from the presence of specific antigens in its capsule that are shared with nerves. Immune responses directed against capsular lipopolysaccharides produce antibodies that cross-react with myelin to cause demyelination⁵. We didn't measured patient's serum autoantibodies because of the rapid progression of the condition and the strongly positive laboratory diagnostic tests.

In Summary we presented a patient with acute fulminant neuropathy which showed characteristic features of GBS, strongly suggested by the rapid progression of symptoms over hours and supported by nerve conduction studies as well as CSF analysis who had maculopapular skin rash before developing neurological symptoms. We also stress the importance of careful and thoroughly evaluating patient with skin rash before general anesthesia which might render serious medical problems.

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