



Guillain–Barré syndrome after cardiac surgery: diagnostic dilemma

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Abstract

Guillain–Barré Syndrome after cardiac surgery is very uncommon. Mechanism remains elusive although immunological reaction post surgery has been postulated. This disease can potentially increase the morbidity of the postoperative patients and generally cannot be explained by the cardiac disease or interventions. It is very much essential to diagnose the condition as appropriate management can substantially and profoundly change the course of treatment.

Keywords Guillain–Barre syndrome · Cardiac surgery · Thyroiditis

Introduction

Guillain–Barre syndrome (GBS) is a rare autoimmune disorder of rapid onset muscle weakness with an incidence of 1–2 per 100,000 per year [1]. GBS after cardiac surgery is reported in a very few anecdotal case reports [2]. This disease can potentially increase the morbidity of the postoperative patients which cannot be explained by the cardiac disease or interventions.

Case report

A 32-year-old male patient presented with extreme shortness of breath on moderate activities. Echocardiogram revealed ruptured sinus of valsalva (RSOV) aneurysm into right ventricle with severe aortic regurgitation. He was recently diagnosed with autoimmune thyroiditis and viral respiratory infection. In emergency room, patient was immediately supported with invasive mechanical ventilation and was haemodynamically optimised. Ruptured sinus of valsalva and aortic valve was repaired under hypothermic cardiopulmonary

bypass. Postoperatively patient was extubated on postoperative day (POD) 1 in a haemodynamically stable condition. On POD 2, poor breathing efforts of the patient led to noninvasive ventilation and then to endotracheal reintubation and mechanical ventilation. Patient was alert and obeying verbal commands. Neurological evaluation demonstrated normal higher motor functions but proximal muscle weakness and absent deep tendon reflexes. MRI of brain and spine and metabolic parameters were normal and acetylcholine receptor antibody (AchRAb) was negative. Nerve conduction velocity (NCV) study revealed marked reduction in MNCV (motor nerve conduction velocity) in both upper and lower limbs. F waves were not recordable. This suggested motor demyelinating polyneuropathy commonly by Guillain–Barre Syndrome. Patient had hoarseness and bilateral vocal cord paralysis was confirmed on laryngoscopic examination. So patient was tracheostomised to prevent aspiration. Plasma exchange was administered as 50 mL/kg on five separate occasions on alternate days. After receiving five cycles of plasmapheresis, patient gradually showed improvement in muscle power and vocal cord movements. Few days after the therapy, patient was successfully weaned off tracheostomy. Patient was discharged on POD 28 in a clinically stable condition.

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Discussion

Risk of developing GBS during the 6-week period after surgery is 13.1 times higher than the general population [1]. Recent retrospective review of patients with postsurgical

GBS (GBS within 8 weeks of a surgical procedure) showed strong and independent association of comorbid autoimmune disease and malignancy with the development of post-surgical GBS [3]. Autoimmune conditions (as in the present case) were present in about 10% of the postsurgical GBS patients in this series. Patients with postsurgical GBS had a significant mortality rate of 16%.

Although GBS has been reported following various other surgeries, it is rarely observed after cardiac surgery [4, 5]. GB syndrome along with bilateral vocal cord paralysis as in this case is very unusual. Recovery from GBS may take from few weeks to few years. Intravenous immunoglobulin (IVIG) or plasma exchange therapy is the standard treatment, but 30% of GBS may still have a residual weakness after 3 years. Although the optimum number of plasma exchange and the volume of plasma to be removed have not been established, widely used protocol is to do total plasma exchange of 200–250 mL/kg over 7–10 days [6]. Plasma exchange, over a 10-day-period, significantly reduces autoantibodies and immune complexes and hastens the recovery time by 50%. Intravenous immunoglobulin treatment is easier to carry out and potentially safer than plasmapheresis. However no differences were observed in the improvement in weakness after 4 weeks, residual disability, duration of mechanical ventilation or mortality between IVIg and plasmapheresis. Corticosteroid therapy has been found to be ineffective in treating GBS [6]. The present case dramatically recovered and responded very well to plasmapheresis.

Many postulated mechanisms of GBS after cardiac operation have been suggested including genetic susceptibility, missed viral infection, anaesthetic drugs, surgical stress, and an antibacterial peptide, which is generated following surgery [4]. GBS is also hypothesised to be a systemic immune response generated by surgical stress and targeting myelin of peripheral nerves. Its quite possible in this case that preceding viral infection might have triggered the immune response which got exaggerated immediately after the surgical stress. About 0.2% of patients affected by autoimmune thyroiditis exhibit myasthenia gravis (MG) that is much higher than the general incidence of MG (0.01%) [5]. However, negative AchR antibody ruled out the possibility of myasthenia gravis in our case. In intensive care unit (ICU) patients, most common causes for weakness of limb or respiratory muscles are critical illness polyneuropathy (CIP) and critical

illness myopathy. These are generally complications of sepsis and multiple organ failure or after the use of large doses of steroids or neuromuscular blocking agents. These conditions can mimic GBS clinically but can be differentiated by precipitating events, electrophysiological features, and the morphology of peripheral nerves. After excluding any organic pathology on MRI brain and spine, nerve conduction velocity (NCV) study depicted the classical pattern of motor demyelinating neuropathy confirming the diagnosis of GB syndrome and rules out CIP.

Certain patients are prone to develop GBS. Unfortunately, it is extremely difficult to recognise such patients preoperatively. Comorbid autoimmune conditions have been found to be an important risk factor. The GBS can be considered as another neurological complication very rarely encountered after cardiac surgery. It is very much essential to diagnose the condition as appropriate management can substantially and profoundly change the course of treatment.

Compliance with ethical standards

Conflict of interest The authors have declared that no conflict of interest exists.

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