Guillain–Barré syndrome after coronary artery bypass surgery

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Abstract

A case of the Guillain–Barré syndrome occurring after otherwise uneventful cardiac surgery using cardiopulmonary bypass is presented. Though the Guillain–Barré syndrome has been reported after surgical procedures, there are very few case reports after cardiopulmonary bypass surgery in the literature. The exact pathophysiological cause of the syndrome is still unknown. However, the most widely accepted hypothesis is that the syndrome is the result of an immune-mediated process. Cardiac surgery may be a trigger for immune-mediated response.

Keywords: Guillain–Barré syndrome • Off-pump surgery

INTRODUCTION

The Guillain–Barré syndrome (GBS) is a frequent cause of neuromuscular paralysis occurring at all ages, and the incidence of GBS is reported to be 1.2–2.3 per 100 000 per year [1]. GBS is a postinfectious disorder in which the most frequently identified infectious agent is Campylobacter jejuni. Others include cytomegalovirus, mycoplasma pneumonia, Epstein–Barr virus and influenza virus. In addition, many reports have documented the occurrence of GBS shortly after vaccinations, operations or stressful events, but the causality and pathophysiology are still debated [1]. Although the GBS is a descriptive clinical entity with relatively widely accepted diagnostic criteria, and the topographic layout, pathological features, pathophysiology and prognostic features of the disease are well understood, the immunopathogenesis of the disease remains uncertain [1, 2]. However, the infectious agents that trigger GBS have epitopes that mimic the epitopes on peripheral nerves [1].

The clinical course of GBS includes rapidly progressive weakness, which is the core clinical feature [2]. Although maximal weakness is reached within 4 weeks, most patients complain of weakness within 2–3 weeks. Thereafter, a plateau phase occurs that may range from days to several weeks or months. This phase is followed by a usually much slower and variable recovery phase. About one-third of GBS patients remain able to walk (‘mild patients’); ~25% of the GBS patients who are unable to walk (‘severe patients’) need artificial ventilation [2]. The cause is predominantly due to weakness of the respiratory muscles. Despite standard treatment with intravenous immunoglobulin (IVIG) or plasma exchange treatment, ~20% of severely affected patients remain unable to walk after 6 months.

Neurological complications are common after cardiopulmonary bypass surgery secondary to embolic phenomena, cerebral hypoperfusion, hypotension or technical problems in establishing cardiopulmonary bypass. In this article, we present the case of the GBS occurring in a 67-year old male patient after coronary artery bypass surgery.

CASE REPORT

A 67-year old male patient was admitted to the cardiology department with a complaint of epigastric pain. The patient was admitted to our cardiovascular surgery department after coronary angiography by means of critical stenosis of the left anterior descending and diagonal arteries. The patient underwent off-pump cardiac surgery without any blood transfusion. Though perioperative and intraoperative periods remained uneventful, the patient noticed weakness and paresthesia of his legs, which progressed rapidly on the postoperative second day. Subsequently, he was unable to stand on the same day. There was no history of toxin exposure, fever or other neurological diseases. Physical examination revealed slight hypotonia, areflexia and loss of strength in all muscle groups in the legs. After neurology consultation, the possible diagnosis of Guillain–Barré was considered and confirmed by electromyography. The patient showed uneventful improvement after 5 days of plasmapheresis. Then the patient was discharged without sequelia on the 10th postoperative day.

DISCUSSION

The pathological mechanism of the GBS is unknown. The dominant view of this topic is that the syndrome is the result of immune-mediated response [1], because the clinical picture of the patient improves with the treatment of plasmapheresis. In a
recent study, Gensicke et al. [3] performed a retrospective analysis of 63 patients with the GBS from January 2005 to December 2010. They observed that 6 of 63 (9.5%) GBS cases have a history of surgery within 6 weeks prior to GBS. The relative risk of developing GBS during the 6-week period after surgery is 13.1 times higher than the normal incidence in the study population. They demonstrated that the incidence of GBS may increase after surgery.

The American Society for Apheresis completed an evidence-based review of 16 neurological indications of apheresis therapy and 10 were ranked as category I or II [1]. Category I denotes that the therapy is indicated as first-line treatment and category II implies second-line treatment. GBS and chronic inflammatory demyelinating polyneuropathy were designated category I. However, although apheresis therapy is designated category I for GBS, the alternative treatment option is IVIG, because the treatment of GBS is centered on therapeutic plasma exchange or IVIG. In our case, we did not prefer the IVIG treatment because the patient did not need to be intubated. Hence, we think that it is not cost effective for the mild form of GBS due to its price.

Although surgery may increase the incidence of GBS, the pathological process is still unclear. The GBS may be another neurological complication that may be very rarely encountered after cardiac surgery [4, 5].

Conflict of interest: none declared.

REFERENCES


eComment. Immune-mediated neurological disorder after cardiac surgery

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In this interesting article by Cingoz et al. [1], the authors describe the prompt diagnosis and successful management of a 67-year-old male patient suffering from Guillain-Barré syndrome on the second day after coronary artery bypass grafting (CABG) surgery. We would like to add a brief comment about the manifestation of Guillain-Barré syndrome and myasthenia gravis after cardiac surgery. Autoimmunity and autoimmune neurological diseases can occur de novo after surgical procedures such as cardiac surgery. Little is known about the mechanisms by which autoimmune diseases develop after cardiac surgery, however, genetic factors, molecular imitation, and exposure to new antigens might be involved in the pathophysiology of these immune-mediated diseases.

Myasthenia gravis is an autoimmune disorder and is generally caused by auto-antibodies to the nicotinic acetylcholine receptor located in the postsynaptic area. By damaging the thymic remnants during sternotomy, some authors [2, 3] speculated that the myoid cells would release acetylcholine receptor in the bloodstream, inducing specific auto-antibodies, and this could precipitate post-cardiac surgery myasthenia gravis. Resatoglu et al. [2] presented the case of a 64-year-old man who developed severe myasthenic symptoms eight weeks after on-pump myocardial revascularization. Antevil et al. [3] reported a case of post-pericardiomyotomy myasthenia gravis in a 68-year-old male patient, without prior history of autoimmune disorders, four weeks after mitral valve replacement. Plasma exchange was successful in these two cases.

There are only three reported cases of Guillain-Barré syndrome following coronary artery bypass surgery. Renlund et al. [4] reported the first case of this immune-mediated ascending polyradiculopathy after CABG surgery in a 65-year-old man with no recent history of infection. An additional two cases of Guillain-Barré syndrome post-cardiopulmonary bypass surgery have been published by Hogan et al. [5]. These three patients were treated with plasmapheresis. Of particular concern is the finding that these patients were all operated on using cardiopulmonary bypass; the patient presented by Cingoz et al. is the first case of Guillain-Barré syndrome occurring after off-pump CABG. The simplest hypothesis for the association between major surgery and Guillain-Barré syndrome would be a non-specific mechanism, whereby major surgery generates an immune reaction targeted to myelin in the peripheral nerves as part of a systemic response to surgical stress, and could not be explained solely by the immune response induced by cardiopulmonary bypass.

Regardless of the pathogenic mechanisms, it is important to remain alert to the existence of de novo autoimmune neurological disorders after cardiac surgery; these disorders are similar to classic autoimmune diseases and are therefore treated with standard therapies and yield similar prognosis.

Conflict of interest: none declared

References