

Guillain–Barré Syndrome after Coronary Artery Bypass Graft Surgery: a Case Report

Manouchehr Hekmat¹, Hamid Ghaderi¹, Mahnoosh Foroughi², and S. Adeleh Mirjafari³

¹ Department of Cardiovascular Surgery, Modarres Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

² Department of Cardiovascular Surgery, Cardiovascular Research Center, Shahid Modarres Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

³ Brain and Spinal Injury Research Center (BASIR), Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran

Received: 17 Jun. 2013; Accepted: 25 Dec. 2014

Abstract- Guillain-Barre syndrome is a neurologic disorder that may appear after infection or major surgery. Guillain-Barre syndrome following cardiac surgery is rare and only based on case reports, and we review all of the published cases. A 52-year-old man after 5 months suffering from chest pain was referred to our hospital and underwent coronary artery bypass graft for 3 vessel disease. The patient was discharged without complication on the 5th postoperative day. He presented Guillain- Barre syndrome after 12 months. He has not completely recovered weakness of upper extremities grade 4/5 with atrophy of both upper extremities remains after 18 months. This disorder is similar to classic GBS. It is important to be alert to de novo autoimmune neurological disorders after cardiac surgery. These disorders are similar to classic autoimmune disease and treated with standard therapies.

© 2015 Tehran University of Medical Sciences. All rights reserved.

Acta Med Iran 2016;54(1):76-78.

Keywords: Guillain–Barré syndrome; Cardiac surgery; Coronary artery bypass graft

Introduction

Guillain- Barre syndrome (GBS) is a disorder that human's immune system attacks part of the peripheral nervous system. The overall incidence of GBS is 1 to 2 per 100,000 per year (1,2). The disorder usually occurs after respiratory or gastrointestinal viral infection. Also, it can be triggered by pregnancy, surgery or vaccination (2,3). GBS is a post infectious disorder in which the most common infectious agent is *Campylobacter jejuni*. Other agents include cytomegalovirus, mycoplasma pneumonia, Epstein-Barr virus, and influenza virus (4)

Only 66% of patients with GBS have a respiratory tract infection or gastrointestinal illness before the onset of GBS (5), and there have been reported of GBS following surgeries without any history of infectious etiology (2).

Evidence of GBS following major surgery is not common and about 5% of the GBS cases are following surgery (6). The simplest hypothesis for the association between major surgery and GBS would be a non-specific mechanism, major surgery triggers an immune

reaction targeted to peripheral nerves as part of a systemic response to the surgical stress (5).

GBS after cardiac surgery is rare and only five cases have been reported, and to the best of our knowledge, our reported case is the 6th case that can be documented.

Case Report

A 52-year-old man presented with chest pain for 5 months before admission. His functional class was 3; his Ejection fraction was 55% with diastolic dysfunction in echocardiography; and 3 vessel disease was demonstrated on angiography, requiring coronary bypass surgery.

His past history was negative for diabetes mellitus, hypertension, dyslipidemia, hyperthyroidism or asthma. He was not a smoker but used opiate agents (orally).

He did not give any history of recent trauma, he didn't have past history of myocardial infarction but had a CCU admission one month before hospitalization for CABG and was receiving atorvastatin 20 mg, aspirin 100 mg, Nitrocontin® 6.4 mg, and Metoral® 50 mg, all

Corresponding Author: H. Ghaderi

Department of Cardiovascular Surgery, Shahid Modarres Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran
Tel: +98 21 22083106, Fax: +98 21 22074101, E-mail address: hghaderi@razi.tums.ac.ir

once daily since surgery.

Routine hematological investigations revealed Hct of 34.3 %, total leukocyte count of 12,200 cells/mm³ and platelet count was 89000; routine blood biochemistry was normal (fasting blood sugar 90 mg/dL, serum sodium 138 mEq/L, potassium 4.6 mEq/L, calcium 9 mEq/L, phosphor 3.6 mEq/L, blood urea nitrogen 30 mg/dL, serum creatinine 1.3 mg/dL, total protein 7.7 g/dL, AST 31 U/dL, ALT 37 U/dL, alkaline phosphatase 165 U/dL). Lipid profile revealed normal lipidemia (total cholesterol 119 mg/dL, triglycerides 88 mg/dL, HDL 37 mg/dL, LDL 79 mg/dL.)

His cardiac enzymes were in normal range (CPK 46 mg/dL, CK-MB 13 mg/dL, and LDH 370 mg/dL).

Blood group was B negative. HBS Antigen, HCV antibody and HIV antibody were negative. Chest X ray was normal.

The patient underwent cardiac surgery CABG (LIMA to LAD and SVG to Diagonal 1 and PDA). His perioperative and intraoperative periods remained uneventful. The patient was discharged without complication on the 5th postoperative day.

After 12 months he had complained of paresthesia and weakness of both upper and lower limbs.

The symptoms started 20 days prior to admission and progressed for 10 days. On examination, he had proximal muscle weakness (grade 4/5) of both the upper extremities and weakness of distal muscle (grade 3/5). In addition, he had grade 4/5 weakness of lower extremities. He had an absence of DTR reflexes and both plantar reflexes. He had feet drop, but his sensory examination was normal. A clinical diagnosis of GBS was made. Lumbar puncture was performed and CSF examination was done (pressure normal, appearance:

clear, protein 84 mg/dL, WBC cells 0 cells/mm³, RBC cells 0 cells/mm³, glucose 62 mg/dL). His serum protein was 75 mg/dL, WBC cells 8500 cells/mm³, glucose 90 mg/dL. Urine protein electrophoresis was negative, serum protein electrophoresis showed decreased albumin and increased alpha 2 and beta protein. In EMG-NCV studies, CMAP (Compound Muscle Action Potential) amplitude was reduced. Denervation in all four limbs and decreased recruitment and polyphasic Action Potential with increased duration was found. After treatment, he was discharged from neurology ward to continue his treatment as out-patient.

After 18 months follow-up, upper limbs weakness were grade 4/5, and he had atrophy of both hands, lower limbs recovered, and his forces were 5/5.

Discussion

In patients with GBS, about 5 % history of surgery are reported (2). In a recent retrospective study in University Hospital Basel and University Children's Hospital Basel from January 2005 to December 2010, among 63 cases of GBS, 6 (9.5%) cases had a history of surgery (2).

Although GBS has been reported after surgery of head, chest, abdomen and limb (3) but it is rare syndrome after cardiopulmonary bypass and CABG operation. There are only five cases of GBS after coronary artery bypass surgery. The risk for GBS after surgery is about 4.1 % cases per 100000 operations (2). Summary of published GBS after coronary artery bypass graft surgery Case reports concluded on Table 1.

Table 1. Summary of published reports Guillain-Barré syndrome after coronary artery bypass graft surgery

First author/reference	Publication year	Number of case	Patient's age	Patient gender	Kind of operation	Start time of GBS	Treatment	Outcome
Renlud (7)	1987	1	65	Male	On pump CABG	--	plasmapheresis	Rapid improvement
Hogan (8)	1992	2	--	--	On pump CABG	--	plasmapheresis	Rapid improvement
Punith (6)	2011	1	65	Male	On pump CABG	12 days	Intravenous immunoglobulin for 5 day	Completely recovery within 12 weeks
Cingoz (9)	2012	1	67	Male	Off pump CABG	3 days	plasmapheresis	Discharged without sequel on the 10 th post operative day
Hekmat	present	1	52	Male	On pump CABG	12 months	Intravenous immunoglobulin	Incompletely recovery

In 1987 the first GBS after CABG was reported by Renlund *et al.*, The patient was a 65 years man, he recovered after plasmapheresis (7). Hogan *et al.*, in 1992 reported two cases of GBS after CABG (8). The 4th case reported by Punith *et al.*, from India. A 65 years old man developed GBS 12 days after CABG; he was also treated by plasmapheresis (6). Cingoza *et al.*, reported 5th case from Turkey, A 67-year-male patient underwent off-pump CABG and after the second day presented signs and symptoms of GBS (9). He recovered after plasmapheresis and discharged after 10 days. The current patient is the sixth case of GBS after CABG and the first one reported from Iran.

GBS affects more males than females (10). Current patient and all of GBS cases after CABG were male.

Gensicke *et al.*, (2) analyzed 63 patients with GBS during 5 years and found that 6 of 63 (9.5%) of them have a history of surgery within 6 weeks prior to GBS. Therefore, the relative risk of developing GBS during the 6-week period after surgery is 13.1 times higher than the normal (2), the current patient developed the disease 12 months after surgery.

GBS recovery is not necessarily quick and the recovery period may be as short as a few weeks or as long as a few years. Despite standard treatment with intravenous immunoglobulin (IVIG) or plasma exchange treatment, about 30% of GBS have a residual weakness after 3 years (1,9). This patient has not completely recovered after 18 months.

The mechanism and pathogenesis of GBS after cardiac surgery is unknown. This disorder is similar to classic GBS. Most of GBS cases appear in few weeks after surgery but current patient presented after 12 months after the operation. In addition, most of the GBS after cardiac surgery recover after standard therapies but this patient didn't recover completely, and he has some degrees of weakness in follow-up.

Although surgery may increase the incidence of GBS, the pathological process is still unclear. The simplest hypothesis for the association between GBS and major surgery would be a nonspecific mechanism. Major surgery generates an immune reaction targeted to myelin in the peripheral nerves as a part of the systemic response to surgical stress, and could not be explained only by immune response induced by cardiopulmonary bypass. It is important to be alert to de novo

autoimmune neurological disorders after cardiac surgery. These disorders are similar to classic autoimmune disease and treated with standard therapies.

Acknowledgment

We sincerely acknowledge Professor Mahmood Mirhoseini MD for editing the manuscript.

References

1. Sejvar JJ, Baughman AL, Wise M, et al. Population incidence of Guillain-Barre syndrome: a systematic review and meta-analysis. *Neuroepidemiology* 2011;36(2):123-33.
2. Gensicke H, Datta AN, Dill P, et al. Increased incidence of Guillain-Barre syndrome after surgery. *Eur J Neurol* 2012;19(9):1239-44.
3. Winer JB, Hughes RA, Anderson MJ, et al. A prospective study of acute idiopathic neuropathy. II. Antecedent events. *J Neurol Neurosurg Psychiatry* 1988;51(5):613-8.
4. Gwathmey K, Balogun RA, Burns T. Neurologic indications for therapeutic plasma exchange: 2011 update. *J Clin Apher* 2012;27(3):138-45.
5. Koc M, Ozalp N, Zulfikaroglu B. Major surgery with Guillain-Barre syndrome: a case report. *J Int Med Res* 2002;30(6):601-4.
6. Punith K SU, Rudresh K, Anil Kumar T. Guillain-Barré syndrome following coronary artery bypass surgery. *Indian J Med Spec* 2011;2(2):157-9.
7. Renlund DG, Hanley DF, Traill TA. Guillain-Barre syndrome following coronary artery bypass surgery. *Am Heart J* 1987;113(3):844-5.
8. Hogan JC, Briggs TP, Oldershaw PJ. Guillain-Barre syndrome following cardiopulmonary bypass. *Int J Cardiol* 1992;35(3):427-8.
9. Cingoz F, Tavlasoglu M, Kurkluoglu M, et al. Guillain-Barre syndrome after coronary artery bypass surgery. *Interact Cardiovasc Thorac Surg* 2012;15(5):918-9.
10. Hadden RD, Cornblath DR, Hughes RA, et al. Electrophysiological classification of Guillain-Barre syndrome: clinical associations and outcome. Plasma Exchange/Sandoglobulin Guillain-Barre Syndrome Trial Group. *Ann Neurol* 1998;44(5):780-89.