Case Report

A Case of Guillain–Barre Syndrome After Anterolateral Myocardial Infarction and Percutaneous Coronary Intervention for it

Farzad Emami[1], MD; Mojtaba Khazaei[1], MD; Hossein Foroughimoghaddam[2], MD; Behshad Naghshtabrizi[2], MD; Nima Naghshtabrizi[3*], MD

ABSTRACT

A 44-year-old woman presenting with a typical history of acute coronary syndrome and anterolateral myocardial infarction in electrocardiography underwent primary percutaneous coronary intervention (PCI). After a few days, she complained of motor and sensory loss in her lower extremities. Given her symptoms, signs, and laboratory exams, Guillain–Barre syndrome (GBS) was diagnosed. Her management was done successfully with therapeutic plasma exchange. Acute myocardial infarction and subsequent PCI might be one of the most uncommon reasons for GBS. ***(Iranian Heart Journal 2022; 23(3): 120-125)***

KEYWORDS: Guillain-Barre syndrome, Myocardial infarction, Percutaneous coronary intervention, Plasma exchange

[1] Hamedan University of Medical Science, Hamadan, IR Iran.
[2] Department of Cardiology, Hamadan University of Medical Sciences, Hamadan, IR Iran.
[3] Department of Cardiology, Tehran University of Medical Sciences, Tehran, IR Iran.

*** Corresponding Author:** Nima Naghshtabrizi, MD; Department of Cardiology, Tehran University of Medical Sciences, Tehran, IR Iran.
Email: nimanaghsh@gmail.com **Tel:** +989185079330

Received: November 20, 2020 **Accepted:** February 3, 2021

Guillain–Barre syndrome (GBS) is an acute polyneuropathy with a variable degree of weakness that reaches its maximal severity within 4 weeks. The disease is mostly preceded by an infection, and it generally runs a monophasic course.[1] The reported incidence rate of GBS from meta-analyses shows an increasing pattern from 0.62 to 2.66 per 100 000 person-years among all age groups.[2] There is an association with the Epstein–Barr virus, measles, *Campylobacter jejuni* (causing diarrhea), human immunodeficiency virus (HIV), *Cytomegalovirus*, and post-vaccinal and postsurgical events.[3] The current hypothesis is in favor of immunological reactions due to hypersensitivity, perhaps as a result of unidentified allergens directed against the myelin sheath of peripheral nerves, leading to acute post-infective polyradiculoneuropathy.[3] Severe forms show axonal degeneration.[3]

We describe a case of GBS following myocardial infarction (MI) and percutaneous coronary intervention (PCI), treated with therapeutic plasma exchange (TPE) successfully.

Case Report

A 44-year-old female patient presented with a complaint of the acute onset of retrosternal chest pain with radiation to the left arm, associated with nausea and vomiting. Acute anterolateral ST-segment-elevation MI was evident on electrocardiography. Her only coronary risk factor was diabetes mellitus.

On initial examination, her blood pressure was 140/100 mm Hg, and her pulse rate was

120 bpm. A physical examination showed a regular heart rhythm with no murmurs, clear respiratory sounds in both lungs, and no edema in the lower extremities. Laboratory tests were almost normal except for leukocytosis, anemia, hyperglycemia, and hyperuricemia (white blood cells: 12100/mm^3, hemoglobin: 12.1 g/dL, blood glucose: 385 mg/dL, and uric acid: 7.7 mg/dL). Transthoracic echocardiography revealed hypokinesia in the anterior wall and all apical segments. Global left ventricular ejection fraction was about 35% with no valvular disease and no myocardial rupture.

After the administration of oral aspirin (325 mg), clopidogrel (600 mg), atorvastatin (40 mg), and intravenous heparin (total dose: 8000 U), she underwent emergency coronary angiography (CAG), and then primary PCI. CAG showed total occlusion in the mid-part of the left anterior descending artery (Fig. 1) and another significant ostioproximal stenosis in a well-developed obtuse marginal (OM3) with good run off (Fig. 2). A 6-Fr guiding catheter was advanced through the right femoral artery to the left coronary artery ostium. A 0.014-inch guidewire was successfully crossed distally to the culprit lesion, and initial recanalization was performed. After predilation with a 2×10-mm balloon, a 2.75×25-mm drug-eluting stent (DES) was deployed at the lesion. Afterward, post-dilatation with a 3×12-mm noncompliant balloon was performed, with no residual stenosis (Fig. 3).

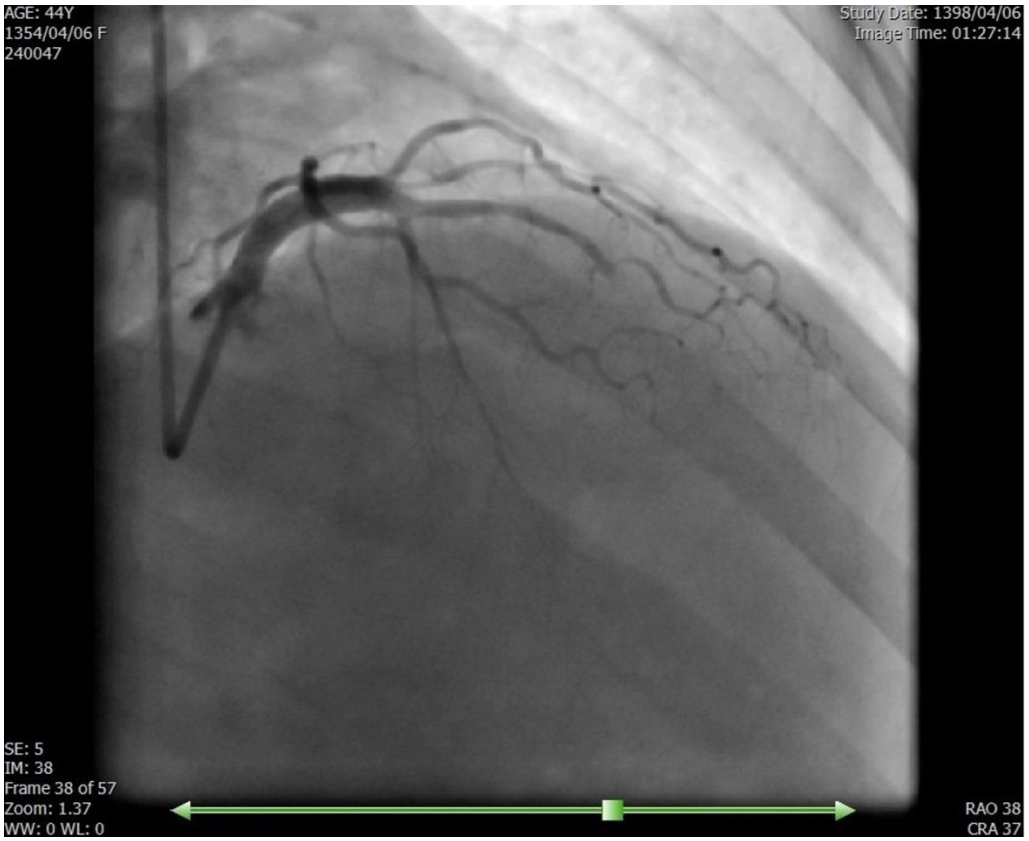

Figure 1: The coronary angiogram shows total occlusion in the mid-part of the left anterior descending artery.

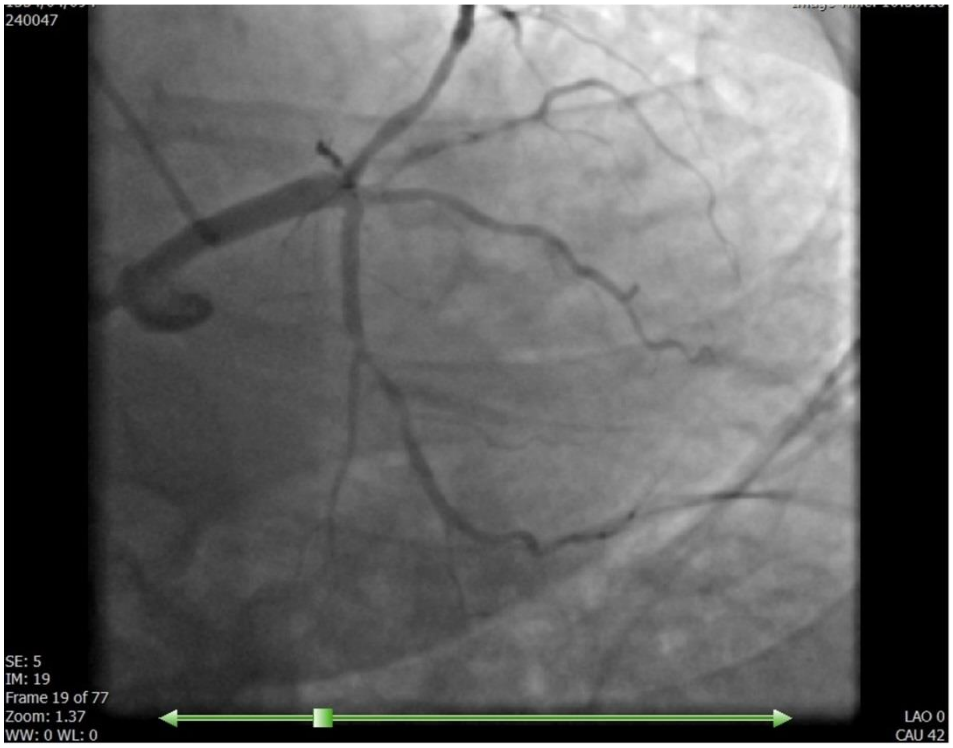

Figure 2: The coronary angiogram shows significant ostioproximal stenosis in a well-developed obtuse marginal (3) with good runoff.

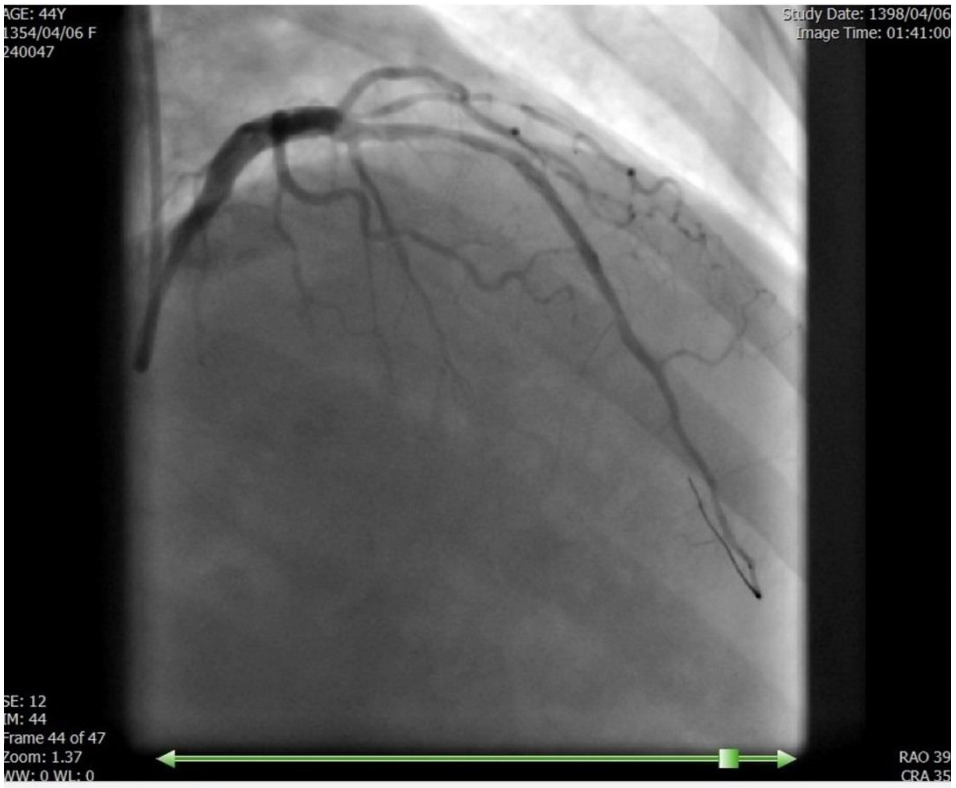

Figure 3: The coronary angiogram shows a successful primary percutaneous coronary intervention on the left anterior descending, with no residual stenosis.

After being transferred to the cardiac care unit, the patient continued to receive dual antiplatelet therapy with aspirin (160 mg) and clopidogrel (150 mg) daily, plus atorvastatin (80 mg daily), nitrate, carvedilol, captopril, and heparin.

Three days after primary PCI (Day 4), the patient complained of retrosternal chest pain. Consequently, she underwent CAG again. CAG via the right radial artery approach revealed a patent stent in the left anterior descending artery with insignificant stenosis at the mid-part and significant ostioproximal stenosis in a well-developed OM3. After predilation with a 2×13-mm balloon, a 2.5×20-mm DES was deployed in the OM3; then, it was postdilated with a 2.75×18-mm noncompliant balloon (successful PCI on the OM3) (Fig. 4).

The day after the second PCI, the patient developed an intermittent fever. She complained of weakness in the lower extremities. She also noticed numbness and loss of superficial sensation such as touch, pain, and temperature below the level of the umbilicus; therefore, a neurological consult was done. The patient was transferred to the neurology ward with a primary diagnosis of acute inflammatory demyelinating polyradiculoneuropathy, one of the variants of GBS. A nerve conduction test was done, and the result was suggestive of acute inflammatory demyelinating polyradiculoneuropathy. She was diagnosed with GBS based on the Brighton case definitions, including the bilateral and flaccid weakness of the limbs, decreased deep tendon reflexes in the weak limbs, a monophasic course, time between the onset-nadir of 12 hours to 28 days, a cerebrospinal fluid (CSF) cell count below 50/μL, and the absence of an alternative diagnosis for weakness.[4]

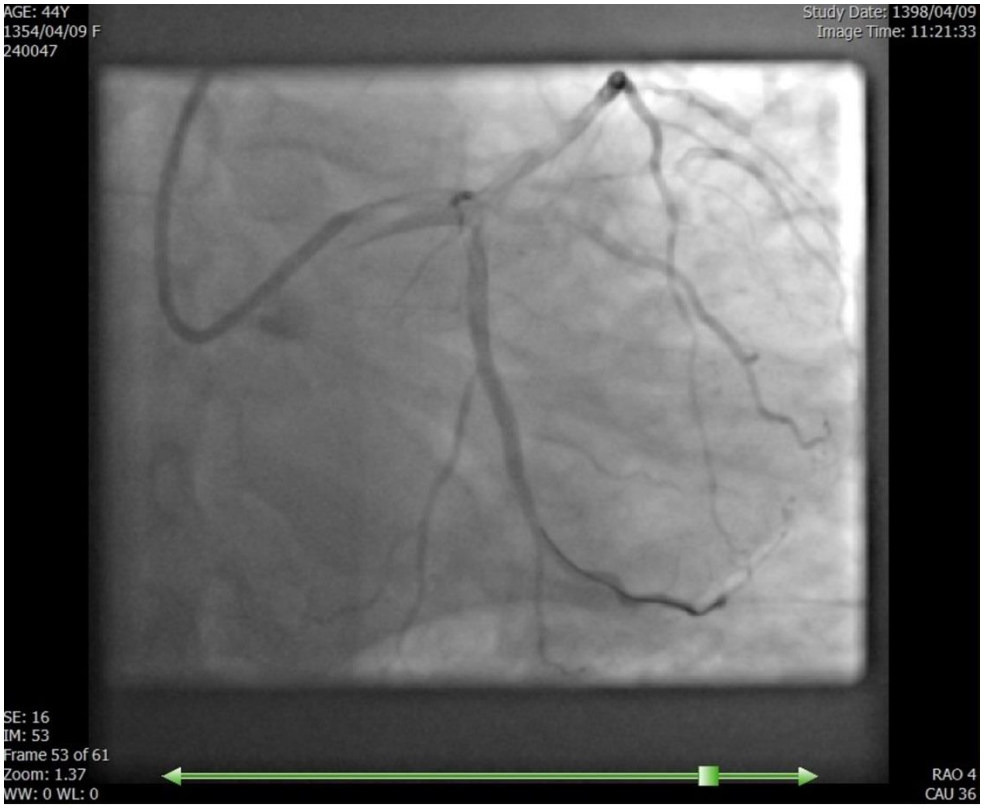

Figure 4: The coronary angiogram shows a successful percutaneous coronary intervention on the obtuse marginal (3), with no residual stenosis.

GBS is included in Category I indication for TPE as per the American Society for Apheresis (ASFA).[5] Thus, 1 volume of TPE was performed with the removal of 40 mL of plasma/kg as per patient weight, height, hematocrit, and total blood volume using a Spectra Optia apheresis machine (TERUMO BCT) in combination with the replacement of 20 mL/kg of fresh frozen plasma and 15 mL/kg of normal saline. Continuous monitoring of vitals (eg, pulse, blood pressure, and respiratory rate) was carried out during the procedure to prevent any adverse events related to the procedure. Two cycles of TPE were performed on alternate days within 4 days, with the removal of 80 mL/kg of plasma (about 2700 mL in each cycle in this patient with a total body weight of 67.5 kg). A few days after the initiation of this therapy, neurological improvement was detected. The patient's muscle power improved from grade II to grade IV (grading of muscle power according to the Medical Research Council Scale)[3] after the completion of 2 cycles of TPE. TPE was discontinued because of improvement in the patient's symptoms and the self-limited nature of her disease.

DISCUSSION and CONCLUSION

A typical case of GBS begins with symmetric leg weakness and distal paresthesia. In more severe cases, these symptoms worsen and spread as the face, respiratory muscles, and oropharyngeal muscles become involved. About one-third of patients need mechanical ventilation at some point due to the involvement of respiratory muscles. In the worst cases, patients have quadriplegia and a marked sensory deficit and may remain ventilator-dependent for months. Demyelination is usually associated with some evidence of associated inflammation. Pathogenic evidence suggests that GBS is the consequence of a misdirected humoral immune response to a preceding infection, most likely *Campylobacter jejuni*, the Epstein–Barr virus, *Varicella zoster* virus, Lyme disease, *Mycoplasma*, and HIV. The most significant laboratory aids are electrodiagnostic studies and CSF examinations. CSF is usually under normal pressure and is acellular. Nerve conduction studies are a dependable and early diagnostic indicator of GBS in cases with a typical clinical presentation.

Patients with GBS need constant monitoring and support of vital functions. TPE is particularly effective for patients who receive the treatment within 7 days of the onset and is the treatment of choice in acutely ill patients. One to 1.5 plasma volumes should be exchanged within 12 to 24 hours of the decision to perform total plasma exchange. TPE involves the removal of injurious macromolecules from the plasma of patients with various medical conditions. The ASFA states that TPE is indicated for many autoimmune diseases, and its benefit occurs through the removal of all inflammatory mediators and toxic proteins, including autoantibodies, complement components, and cytokines.[5] TPE is relatively safe. It shortens the course of hospitalization and reduces the mortality and incidence of permanent paralysis.[6] Minor complications like hypotension and allergic reactions due to fresh frozen plasma have been observed in the procedure, but they can be managed easily. In our case, the patient developed GBS after PCI, which could be lethal if not treated promptly. Thus, patients with GBS after PCI can be managed by TPE.

A few studies have revealed some kinds of polyneuropathies after cardiac interventions. A previous study showed that sepsis, catecholamine support, and increased urea levels were associated with the development of polyneuropathy.[7] In another investigation, polyneuropathy was the most common peripheral nervous system (PNS) concern in heart transplant recipients, occurring in 13% of the studied patients.[8] Davidov et al[9]

showed that the development of polyneuropathy in the postoperative period in patients undergoing cardiac surgeries was mostly associated with sepsis and multiple organ failure, rather than the procedure itself. In classifying PNS diseases after cardiac surgeries, 4 distinct categories are most common. First, benign and transient postoperative complications may occur, including brachial plexopathy and peroneal neuropathy. Second, PNS diseases associated with an underlying disease prompting heart transplantation become apparent months to years after the transplantation. This is manifested by the large number of patients with polyneuropathy who suffer underlying amyloidosis. Some patients also have a high preponderance of underlying diabetes mellitus and symptomatic diabetic neuropathy. Third, PNS complaints are associated with necessary medications. The side effects of aggressive treatment for dyslipidemia with statins are notable among myopathies. Finally, the sophisticated, life-long care provided to patients has increased the post-intervention survival time, with many patients reaching old age. Therefore, PNS diseases such as degenerative disk disease and herpes zoster have been seen in the general aging population.[8] As was mentioned above, none of these categories includes GBS. This classification was about PNS diseases after cardiac surgeries, and it is even more uncommon after PCI. In this case report, we described a case of GBS following MI and PCI, which, albeit very rare, can lead to a lethal outcome if not treated promptly. Patients who complain of weakness in their limbs after MI should undergo careful neurological examinations for the early diagnosis of GBS. GBS after PCI is a rare condition and associated with high mortality if not treated promptly. The TPE procedure can be performed in these patients under continuous cardiac monitoring. TPE should be started as early as possible after the diagnosis of GBS for early recovery and better outcomes.

REFERENCES

1. van Doorn PA. Diagnosis, treatment and prognosis of Guillain-Barré syndrome (GBS). La Presse Médicale. 2013; 42(6):e193-e201.

2. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population Incidence of Guillain-Barré Syndrome: A Systematic Review and Meta-Analysis. Neuroepidemiology. 2011; 36(2):123-33.

3. Kundu AK. 6th ed. Kolkata: Kaustuv Paul for KSP Udyog; 2003. Bedside Clinics in Medicine; p. 160.

4. Fokke C, van den Berg B, Drenthen J, Walgaard C, van Doorn PA, Jacobs BC. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. Brain. 2014; 137(1):33-43.

5. Szczepiorkowski ZM, Winters JL, Bandarenko N, Kim HC, Linenberger ML, Marques MB, et al. Guidelines on the use of therapeutic apheresis in clinical practice—evidence-based approach from the Apheresis Applications Committee of the American Society for Apheresis. J Clin Apher. 2010; 25(3):83-177.

6. Kes P, Pasini J. Therapeutic plasma exchange in the critically ill patients. Acta Clin Croat. 1999;38(4):259-74.

7. Thiele R, Jakob H, Hund E, Tantzky S, Keller S, Kamler M, et al. Sepsis and catecholamine support are the major risk factors for critical illness polyneuropathy after open heart surgery. The Thoracic and cardiovascular surgeon. 2000;48(03):145-50.

8. Mateen FJ, van de Beek D, Kremers WK, Daly RC, Edwards BS, McGregor CG, et al. Neuromuscular diseases after cardiac transplantation. The Journal of heart and lung transplantation. 2009;28(3):226-30.

9. Davidov N, Vinogradov O, Gorokhovatskiy Y, Kuznetsov A. Polyneuropathy in Patients Undergoing Cardiac Surgery. J Anesth Crit Care Open Access. 2017;9(2):00338.