GUILLAIN-BARRÉ SYNDROME IN A PATIENT SUFFERING ACUTE MYOCARDIAL INFARCTION

Mirella Sharma[1], Petar Kes[1], Vanja Bašić-Kes[3], Vjeran Nikolić-Heitzler[2], Vida Demarin[3] and Slava Podobnik-Šarkanji[3]

[1]Department of Nephrology and Dialysis, [2]Department of Cardiology, [3]University Department of Neurology, Sestre milosrdnice University Hospital, Zagreb, Croatia

SUMMARY – We report on a patient with acute neurologic disorder, i.e. Guillain-Barré syndrome, successfully treated by plasmapheresis. The patient suffered a myocardial infarction, which has neither been defined nor described in the available literature as a causative event for acute polyradiculoneuropathy, but merely a random observation. The course of the disease in the patient was complicated by pulmonary infection which we presumed to be a precipitating factor for the occurrence of Guillain-Barré syndrome.

Key words: *Myocardial infarction – complications; Polyradiculoneuropathy – etiology; Polyradiculoneuropathy – therapy; Guillain-Barré syndrome; Case report*

Introduction

Guillain-Barré syndrome is an acute and usually progressive form of inflammatory polyradiculopathy, and is considered to be the most common acquired demyelinating neuropathy. The reported incidence rate worldwide varies from 0.4 to 1.7 cases *per* 100,000 persons *per* year. A mild respiratory or gastrointestinal infection precedes the neuropathic symptoms by 1 to 3 weeks (sometimes longer) in about 60% of patients. Recently, it has been appreciated from serologic studies that *Campylobacter jejuni* is the most frequent identifiable preceding infection. Other, less common antecedent events or associated diseases include surgical procedure, exposure to thrombolytic agents, viral exanthemas, and other viral diseases; cytomegalovirus (CMV), Epstein-Barr virus (EBV), and human immunodeficiency virus (HIV)[1]. Inflammation results in both motor and sensory dysfunction, and therefore presents itself with ascending muscular weakness, paresthesias, and loss of reflexes.

Correspondence to: *Mirella Sharma, M.D.,* Department of Nephrology and Dialysis, Sestre milosrdnice University Hospital, Vinogradska c. 29, HR-10000 Zagreb, Croatia

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Diagnosis is based on clinical syndrome. In cerebrospinal fluid (CSF), proteins are increased but not cells[2,3]. Electromyography and nerve conduction velocity tests help confirm the diagnosis. Two thirds of patients have slow nerve conduction velocities and evidence of segmental demyelination (F-wave latency prolonged) at the time of onset.

Current therapeutic modalities include plasmapheresis and immunosuppressive drugs.

Plasmapheresis is a process of extracorporeal separation of cellular elements from plasma. It has a theoretical therapeutic advantage for most of the conditions associated with abnormal circulating plasma components (antibodies or antigens, immune complexes, inflammatory mediators, and other protein bound toxic substances)[1].

Case Report

A 46-year-old patient was admitted to the Coronary Care Unit for severe chest pain lasting for two days before admission. The pain was localized retrosternally, accompanied by diaphoresis and dyspnea. Chest x-ray showed signs of left-sided heart failure, and ECG showed ST-segment elevation in all precordial leads (V_1-V_6) with already

formed Q wave. High levels of cardiac serum markers were evident (cardiac troponin T (cTnT) - 1.63 U/L, serum creatine kinase (CK) - 163 U/L, already falling).

The ECHO performed on the same day corroborated the diagnosis of subacute myocardial infarction of the anteroseptal and partly lateral region of the left ventricle (LV). A fresh mural thrombus (2.1x2.6 cm) was also present in the septoapical part of the LV. Ejection fraction (EF LV) was approximated to 40%. Therapy with HMW heparin, nitrate, acetylsalicylic acid, diuretic, and angiotensin convertase enzyme (ACE) inhibitor was initiated.

The history revealed the patient to be diabetic (type 2a), for the last three years on insulin therapy, but with already presents chronic complications of the disease (diabetic retinopathy, polyneuropathy and nephropathy). He had also suffered from high blood pressure for the last five years, but was not taking the prescribed medication regularly.

Unfortunately, the course of disease was complicated by epistenocardial pericarditis and pneumonia of the right lower lobe. Therapy with cefuroxime was instituted.

On day 6 of hospitalization, the patient complained of paresthesia and weakness of both legs and arms, and on day 9 paresis of the lower extremities, especially of the left leg, developed. Deep tendon reflexes were weakened; dysarthria and paresis of the right facial nerve (central type) were also noted on examination. The radiogram of the whole spine showed nothing but early degenerative changes. Given the fact that there was a mural thrombus in the LV, embolization had to be suspected and CT scan of the brain was done. Although two lacunar ischemic lesions were detected in the left cortical region of the frontal and temporoparietal region, the clinical presentation (progressive paraparesis) was not suggestive of an ischemic stroke. The neurologist, suspecting Guillain-Barré syndrome, suggested lumbar puncture, however, the cerebrospinal fluid (CSF) was clear and biochemical markers were within the normal limits (protein 0.31 g/L; cell count 0/3 μL). Electromyoneurography (EMNG) performed later showed the already known severe polyneuropathic changes that were consistent with the patient's diabetic history.

In spite of the aforementioned data, relying mainly upon clinical presentation, we decided to perform plasmapheresis. Twelve sessions were done on a daily basis. Motor improvement was evident after the first few plasma exchanges. The whole process had to be quite attentive due to the possibility of hemodynamic compromise in the already cardially unstable patient.

During the hospital stay, the patient had no anginal pain or rhythm disturbances, and blood pressure and glycemia were well regulated; diuresis was maintained appropriate by diuretics, so pulmonary congestion subsided, and so did the inflammation of the lung.

Repeat EMNG of the lower extremities was performed on day 11 of the disease. Slow nerve conduction and prolonged F waves were evident, thus confirming the diagnosis of Guillain-Barré syndrome. After 30 days of hospitalization, the patient was able to walk alone and was referred to a rehabilitation institution.

Discussion

Guillain-Barré syndrome is an acute inflammatory demyelinating polyneuropathy characterized by muscular weakness and mild distal sensory loss, which in about 2/3 of cases begins 5 days to 3 weeks after a banal infection, surgery, or an immunization.

Focal areas of segmental demyelination with perivascular and endoneurial infiltration of lymphocytes and monocytes are found along peripheral nerves, their roots and cranial nerves. In severe lesions, axonal degeneration may also be found[1,2].

Weakness and paresthesias usually begin in the legs and progress to the arms. Deep tendon reflexes are lost. Affection of cranial nerves may result in weakness of facial and oropharyngeal muscles, and 5%-10% of patients with severe disease have to be intubated due to respiratory failure. Autonomic dysfunction can occur (blood pressure and heart rate fluctuations) as well as cardiac arrhythmias[1].

The most important laboratory aids are electrodiagnostic studies and CSF examination. The CSF is usually under normal pressure and is acellular (or contains only a few lymphocytes). Usually, the protein content is normal during the first few days of symptoms, but then it begins to rise, reaching a peak in 4 to 6 weeks. In few patients (10 percent or less), the CSF protein values are normal throughout the disease.

Nerve conduction studies are a dependable and an early diagnostic indicator of Guillain-Barré syndrome, and in cases with a typical clinical presentation the CSF analysis can be omitted. While a limited electrodiagnostic examination may be normal early in the course of disease, a more thorough study which includes the measurement of late responses, almost invariably shows abnormalities in an affected limb within days of the first symptom. Also early on, if there are features that indicate widespread axonal damage, a poor prognosis for complete motor recovery is

very likely. The most frequent early findings are reduction of the amplitudes of muscle action potentials, and slowed conduction velocity or conduction block in motor nerves. Prolonged distal latencies (reflecting distal conduction block) and prolonged or absent F responses (affection of the proximal parts of nerves) are other important diagnostic findings, all reflecting demyelination[1].

Patients with Guillain-Barré syndrome need constant monitoring and support of vital functions. It is thought that corticosteroids in acute stage worsen the outcome and should not be used[5]. Plasmapheresis proved to be efficient in several multicenter studies. It is particularly effective for patients who receive the treatment within 7 days of onset[4], and is the treatment of choice in acutely ill patients. Usually 1-1.5 plasma volumes should be exchanged within 12-24 hours of the decision to perform total plasma exchange (TPE). This should be followed by daily plasmapheresis for the first 5 days, then five additional one-plasma volume exchange every other day. Isonatric 5% albumin is the recommended replacement solution[5-7]. In this case, TPE sessions were done consecutively for 12 days, and the plasma exchange regimen involved removal of 3500 mL of plasma. The replacement fluid was 5% albumin. One might argue the regimen used, but due to the severity of the disease, already compromised status of the patient, and a very good response to plasmapheresis, we decided as previously stated.

Plasmapheresis is relatively safe; it shortens the course of the disease and hospitalization, and reduces mortality and incidence of permanent paralysis. Rare complications of plasmapheresis include hypotension, hypoprothrombinemia with bleeding, and cardiac arrhythmias. Hepatitis and AIDS present a risk only when plasma is used as the replacement fluid[8].

To our knowledge, only two cases of Guillain-Barré syndrome after myocardial infarction have been described, with no other obvious cause for the condition[9]. Our patient had pneumonia which might have precipitated the autoimmune response and caused the acute neurologic disorder. Therefore, we conclude that although there is a chance association between myocardial infarction and Guillain-Barré syndrome, the patients who complain of weakness after myocardial infarction should undergo careful neurologic examination.

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Sažetak

GUILLAIN-BARRÉOV SINDROM U BOLESNIKA S AKUTNIM INFARKTOM MIOKARDA

M. Sharma, P. Kes, V. Bašić-Kes, V. Nikolić-Heitzler, V. Demarin i S. Podobnik-Šarkanji

Prikazan je bolesnik s akutno nastalim neurološkim poremećajem, Guillain-Barréovim sindromom, koji je uspješno liječen postupkom plazmafereze. Bolesnik je hospitaliziran zbog subakutne slike srčanog infarkta koji prema našim saznanjima i na temelju dostupne literature nije definiran kao uzrok nastanka ove akutne poliradikuloneuropatije, već je opisan kao slučajnost. U našega je bolesnika klinička slika bila komplicirana respiracijskim infektom. Smatramo da je to bio čimbenik koji je ubrzao nastanak Guillain-Barréova sindroma.

Ključne riječi: *Infarkt miokarda – komplikacije; Poliradikuloneuropatija – etiologija; Poliradikuloneuropatija – terapija; Guillain-Barréov sindrom; Prikaz slučaja*