Acute myocardial infarction revealing a polycythemia vera

Infarctus du myocarde révélant une polyglobulie primitive

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Abstract. The occurrence of myocardial infarction in myeloproliferative disease is mostly attributed to coronary thrombosis due to hyperviscosity and thrombocytosis. We report a 55-year-old man case with polycythemia vera none diagnosed before and revealing with ST-segment elevation myocardial infarction; this is a relative rare association. The clinical status, comorbidities and patient outcome were studied. This case illustrates the importance of early diagnosis of polycythemia vera and research almost systemic thrombotic complications.

Key words: myocardial infarction, polycythemia vera, diagnosis, therapy

Résumé. Nous rapportons ici une observation originale d’infarctus antérosepto-apicale révélant une polyglobulie primitive ; c’est une association relativement rare dans la littérature. Il s’agit d’un homme âgé de 55 ans hospitalisé au service de cardiologie pour un syndrome coronarien aigu qui va permettre de découvrir fortuitement, suite à un hémogramme, une polyglobulie primitive. L’état clinique, les comorbidités et le devenir du patient ont été étudiés. Ce cas illustre l’importance du diagnostic précoce de maladie de Vaquez et la recherche quasi systémique des complications thrombotiques.

Mots clés : infarctus du myocarde, polyglobulie primitive, diagnostic, traitement

Polycythemia vera is a chronic myeloproliferative disorder, involving a multipotent hematopoietic progenitor cell, which causes in general an increased production of red cells, granulocytes and platelets, but most significantly in erythrocytes. The occurrence of myocardial infarction in myeloproliferative disease is mostly attributed to coronary thrombosis due to hyperviscosity and thrombocytosis. Patients with polycythemia vera (PV) or essential thrombocythemia (ET) are at increased risk of arterial and venous thromboembolic events. Arterial ischemic complications occur in 24 to 43% of these patients, particularly those with cardiovascular risk factors (especially cigarette smoking) [1].

Case report

A 55-years old male patient, former smoking 20 pack-years, was admitted at the cardiology department for acute coronary syndrome with ST- segment elevation. There was no known hypertension, dyslipidaemia, diabetes, or family history of cardiovascular disease. On admission, the patient was conscious with good mucocutaneous color, good hydration status. Blood pressure and pulse rate were measured as 120/80 mmHg and 78/min, respectively. Cardiovascular examination discovered a status of I NYHA class without chest pain. Electrocardiogram showed ST-segment elevation in leads V1-V5 with T wave inversion, Doppler echocardiography revealed hypokinesia of the septolateral wall. The brain scan was without alteration.

Treatment was initiated by administration of a beta-blocker, aspirin, statin, low- molecular- weight heparin and an angiotensin-converting enzyme inhibitor (ACE). After stabilization of clinical, hemodynamic and electrical status, the patient was transferred to another hospital for angiography which findings a stenosis and heterogeneous appearance of the anterior descending coronary artery, a non-dilated left ventricle with apical akinesia.
The complete blood count (CBC) analysis was as follows: a hemoglobin (Hb) at 205 g/L (N: 115 to 160), hematocrit (Ht) 0.65 (N: 0.37-0.47), red blood cells: 7.27 T/L (N: 4.5-5.1), mean corpuscular volume (MCV) 90 fl (N: 84-96), white blood cells: 8.44 G/L (N: 4-10) with 3.71 G/L neutrophils, 0.59 G/L eosinophils, 2.78 G/L lymphocytes and 1.53 G/L monocytes; platelets: 689 G/L (N: 150-400). The arterial blood gas test showed normal oxygen saturation $\text{SaO}_2$ of 97.4%. Serum concentrations of glucose, lipid, calcium, urea, creatinine, and electrolytes, and hepatic functions were within normal limits.

The bone marrow examination showed an increase in all cell lineages, consistent with a myeloproliferative disorder, cytogenetic analysis was normal. According to these results the diagnosis of polycythemia vera was confirmed.

The final diagnosis was a polycythemia vera complicated by acute myocardial infarction. Myelosuppressive therapy was introduced a basis of 15 mg/kg per 24 of hydroxyurea, evolution of the disease was favorable after a month of treatment and hematological parameters were reduced to normal levels over a period of 5 months (figure 1).

**Discussion**

Polycythemia vera is a disease in the elderly, often discovered between 50 and 70 years and more rarely, on the occasion of a cardiovascular complication. There is often a gradual installation mode and location of subcortical lesions. The risk of thrombosis is correlated with higher hematocrit and probably with the quantitative and qualitative abnormalities of platelets associated with it.

The diagnosis of polycythemia vera was placed under the revised criteria of the World Health Organization [2]: two major criteria (hemoglobin $>185$ g/L and no secondary cause) associated with two minor criteria with thrombocytosis $>400$ G/L and diffuse myelofibrosis with erythroid and megakaryocytic proliferation in the bone marrow biopsy.

In our case, it was a patient with no family history and without associated conditions (diabetes, hypertension or dyslipidemia) who experienced an episode of acute coronary syndrome. After a thorough exploration biological and given the absence of risk factors and angiographic appearance of the remaining normal coronary arteries, the diagnosis of acute myocardial infarction secondary to polycythemia vera was used.

The pathophysiology of thromboembolic events in polycythemia vera has not been elucidated, but many factors are involved: increases in hematocrit and blood hyperviscosity, stimulation of platelet aggregation and thrombogenesis, the presence of leukocytosis, rigidity of the membrane and intimal proliferation [3-5].

The prevention of vascular risk and foremost is the main objective thrombotic treatment of polycythemia. The hydroxyurea is the perfect option in treating patients with polycythemia vera with an increased risk of thrombosis [6], however, the effectiveness of a treatment strategy antiplatelet and antithrombotic was recently reported in a similar case [7].

The systematic control of vascular risk factors associated (smoking, hypertension, high cholesterol, overweight) is a common sense measures. Generally the treatment of acute coronary syndromes secondary to chronic myelo-proliferative disorders require special attention in maintaining the delicate balance between the risk of hemorrhage and thrombosis tendency [8].

In light of these data, we conclude that CBC is a haematological screening sufficient for the vast majority of patients hospitalized for a first ischemic stroke to avoid missing a polycythemia.

**Conflict of interest:** none.

**References**


