ANAEMIC COR INDUCED BY THROMBOTIC MICROANGIOPATHY ASSOCIATED WITH SYSTEMIC LUPUS ERYTHEMATOSUS

Flávio Barboza^{1,*}, Fernanda da Costa Ferreira Guerra¹, Amanda Rocha dos Santos¹, Rafael Garcia de Maria¹

¹Universidade Federal do Mato Grosso, Sinop (MT), Brazil.

*Corresponding author: flavio.fbarboza@uol.com.br

BACKGROUND

Anemic *cor*, a kind of high output heart failure, is a clinical situation in which the heart cannot maintain its normal contracting function due to anemia. The patient's anemic state induces reduction of peripheral vascular resistance and increase of cardiac output. Hemodynamic modifications may happen mainly in the short-term in acute anemic states. Multiple causes of anemia may lead to anemic *cor*, such as iron, B12 deficiency and hemolytic anemias. Anemic *cor* secondary to thrombotic microangiopathy induced by systemic lupus erythematosus (SLE) is a clinical challenge and must be distinguished from other differential diagnosis, like myocardiopathy, acute pericarditis, heart attack, and antiphospholipid-associated valvopathy. Furthermore, systemic hypertension, sedentarism, obesity, dyslipidemia, and chronic corticoid use, when in concomitance with lupus, may difficult the heart failure etiology in many cases.

CASE REPORT

A 38-year-old woman diagnosed with SLE (joints and hematologic alterations, 1/640 nuclear speckled ANA, positive anti-SM) presented with important asthenia. Recent exams revealed low complement (c3 and c4), 2.6 g/dL hemoglobin (Hb), leucocytes 4,000 u/L, lymphocytes 1,500 u/L, low platelets (90,000 u/L), reticulocytosis, high levels lactate dehydrogenase, negative direct coombs, elevated unconjugated bilirubin, high ferritin, normal B12, erythrocyte sedimentation rate 160 mm/h, creatinine 2.1 mg/dL and schizocytes in peripheral blood. Reduced ADAMTS13 test. Having excluded infectious etiologies and having normal echocardiography, negative hemoculture and tomographies without abnormalities, methylprednisolone and intravenous immunoglobulin was initiated. The patient developed improvement of renal function, thrombocytopenia and mild improvement of Hb (4 g/dL). After 4 days of hospitalization, the patient suddenly evolved with hemolysis' worse and respiratory failure, needing mechanical ventilation and intensive care support. A new echocardiogram revealed global hypokinesia with preserved ejection fraction. Other exams showed normal troponin, creatine phosphokinase and MB-fraction, as well as sinus tachycardia in the electrocardiogram. Assuming a diagnostic of anemic *cor*, blood and plasma transfusion was initiated. Despite the increase in the hemolysis and worsening of renal insufficiency, platelets and hemolysis markers, the patient improvement Hb (7 g/dL) of cardiac function and output mechanical ventilation. She was transferred to another unit to start plasmapheresis and sequential rituximab.

CONCLUSION

Despite being rare, anemic *cor* is an emergency and its treatment involves correction of the anemia. In this case, the microangiopathic hemolytic anemia secondary to SLE with relative contraindication to blood transfusion and failure response to methylprednisolone and immunoglobulin led the patient to anemic *cor*. This life-threatening condition needs transfusion of blood and plasma despite the hemolysis worsening state.

KEYWORDS

Anemic *cor*, Lupus, Thrombotic microangiopathy.