Intra-arterial pulmonary thrombolysis at the postoperative period of brain aneurysm clamping. Case report

Trombólise intra-arterial pulmonar no pós-operatório de clipagem de aneurisma cerebral. Relato de caso

INTRODUCTION

Pulmonary thromboembolism (PT) is a leading cause of morbimortality in patients submitted to neurological procedures. The purpose of this study was to present a case of intra-arterial pulmonary thrombolysis in recent neurosurgery postoperative period. Male patient, undergoing neurosurgery, presented as a complication on the seventh day of postoperative massive pulmonary embolism with hemodynamic instability and intra-arterial pulmonary thrombolysis with alteplase was indicated. Evolution was satisfactory without bleeding complications and patient was discharged. Pulmonary thromboembolism is a high morbidity and mortality condition at neurosurgical postoperative period and thrombolysis should be an alternative therapy in cases refractory to clinical treatment.

Keywords: Intracranial aneurysm/surgery; Intracranial aneurysm/complications; Pulmonary embolism/etiology; Postoperative period; Thrombolytic therapy; Case reports

CASE REPORT

Male, 36 year old patient with a diagnosis of anterior communicating artery aneurysm, with indication for surgical treatment. As previous event he presented with ischemic stroke in the occipital region.

He was admitted at the intensive care unit (ICU) at immediate postoperative period of anterior communicating artery aneurysm clamping without intercurrence, and was discharged from the ICU on the second postoperative day.

On the seventh postoperative day he presented with precordial burning pain, together with tachycardia, cold sudoreis and hemodynamic instability, and was transfer to the ICU requiring administration of vasoactive drugs and oxygen.

The electrocardiogram disclosed dispersed alteration of ventricular repolarization. Transthoracic echocardiogram showed dilation of the right chamber.
Intra-arterial pulmonary thrombolysis and brain aneurysm clamping

and dysfunction of the right ventricle without other significant alterations. A computed tomography angiography of the chest was requested showing filling defects in both pulmonary arteries and their segmentary branches, related to an extensive bilateral pulmonary thromboembolism (Figures 1 and 2).

Thereupon invasive hemodynamic monitoring was chosen with a Swan-Ganz catheter, initially presenting with a cardiac index of 2 L/min and mean pulmonary artery pressure of 37 mmHg. Patient was hypotensive, receiving noradrenaline and dobutamine, without clinical improvement. During hematological investigation a protein C deficiency and resistance to activated protein C were detected.

Because of the condition’s severity, intravenous heparin was introduced in a 60 UI/kg dose and, later, intra-pulmonary arterial thrombolysis with maneuvers of mechanical fibrinolysis and injection of 20mg of alteplase (rt-PA) in bolus. This was followed by 30 mg of the same substance intravenously, with improvement of the clinical, hemodynamic and angiographic parameters which allowed for reduction of vasoactive drugs.

Intravenous heparin was maintained in continuous infusion for 72 hours with control of activated partial thromboplastin time (APTT) between 1.5 – 2.0 times the normal values. Low molecular weight heparin (enoxaparin) 1 mg/kg subcutaneous every 12 hours was introduced, as from the fourth day of thrombolysis.

Patient remained stable, presenting as only post-thrombolysis intercurrence a discreet bleeding at the site of the vascular puncture. He did not present neurological deficits and was discharged from the ICU and later from the hospital.

**DISCUSSION**

Pulmonary thromboembolism is a severe condition affecting about 2.5% of hospitalized patients, with a mortality of over 30% in cases of massive PT3,4. Included among predisposing factors are extensive surgical procedures, prolonged immobilization, stroke, chronic venous insufficiency of the lower limbs, in addition to disorders of the coagulation system such as antithrombin III, protein S and protein C3,5, deficiency. In such cases diagnosis is mostly achieved after the thrombotic event. In this case the patient presented more than one predisposing factor for the condition, that is to say, the protein C deficiency and neurosurgical procedure.

After diagnosis, risk stratification was required and patients who present hemodynamic instability, respi-
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**REFERENCES**


