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Pulmonary artery dissection: From diagnosis to management

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Pulmonary artery dissection (PAD) is a rare and often lethal event. It affects the main pulmonary artery in 80% of cases. It occurs mainly in patients with underlying chronic pulmonary hypertension leading to pulmonary artery aneurysm. It is often associated to congenital cardiac anomalies and often a patent ductus arteriosus is present. However, other causes include connective tissue disorders, right heart endocarditis, amyloidosis, trauma, and severe atherosclerosis. Usually, patients present with chest pain, dyspnoea and cyanosis, progressing rapidly to hemodynamic deterioration leading to sudden death due to rupture of the aneurysm. However, in rare cases, PAD may present with subtle non-specific symptoms.

PAD is usually diagnosed during post-mortem examination and only very few cases have been previously treated surgically and reported in the literature. It is worth considering this rare condition during the differential diagnosis workup. An enlargement of the PA on the chest x-ray should raise the suspicion of a pulmonary artery aneurysm and call for further imaging studies. Echocardiography should represent the first line of investigation followed by computed CT-scan. This one would accurately evaluate the pulmonary artery anatomy with the aneurysm extension, the intimal flap, the presence of intraluminal thrombi and the presence of haemopericardium.

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Restoration of sinus rhythm by catheter ablation: Time course of prothrombotic and endothelial function responses

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A trial fibrillation (AF) is a known prothrombotic state, which is associated with endothelial dysfunction, increased platelet reactivity and hypercoagulation. However, little is known whether restoration of sinus rhythm by radiofrequency (RF) ablation acutely improves inflammation, myocardial injury and prothrombotic tendencies.

Ninety consecutive patients undergoing elective RF catheter ablation for AF were recruited prospectively. Clinical, echocardiographic and procedural details were recorded. High-sensitivity CRP (hs-CRP), white cell count (WCC), neutrophil count, Troponin-T, creatine kinase (CK) and creatine kinase-MB (CKMB), fibrinogen and D-Dimer were measured at baseline, 1, 2, 3, 7 days and 1 month post ablation. AF recurrence was documented on physician review at 3 days, 1, 3 and 6 months post procedure.

The cohort comprised 53.3% paroxysmal, 34.4% persistent and 12.2% long-standing persistent AF patients. Hs-CRP peaked and was significantly elevated at days 2 (36.89 ± 34.87 vs. 2.57 ± 2.16 mg/L, p<0.05) and 3 (44.29 ± 37.37 vs. 2.57 ± 2.16 mg/L, p<0.05) post ablation compared to baseline. Troponin-T (1.61 ± 1.07 vs. 0.05 ± 0.08 µg/L, p<0.05) and CKMB (10.65 ± 5.10 vs. 3.21 ± 1.20 µg/L) peaked at day 1 post procedure. Fibrinogen (4.71 ± 1.42 vs. 3.11 ± 0.61 g/L, p<0.05) and D-Dimer (0.58 ± 0.46 vs. 0.30 ± 0.18 FEU, p<0.05) levels were significantly elevated at 1 week post procedure. Hs-CRP elevation correlated with Troponin-T (rs=0.35, p<0.02) and fibrinogen (rs=0.59, p<0.01) elevation. Increased hs-CRP, Troponin-T and CKMB elevation post ablation was significantly associated with AF recurrence within 3 days post procedure and increased fibrinogen elevation with AF recurrence at 3 days and 1 month.

Patients undergoing RF ablation for AF exhibit an inflammatory response and myocardial injury within the first few days post ablation. Increased inflammatory response is linked to immediate AF recurrence. Prothrombotic markers are elevated one week post ablation, may be driven by inflammation and explain the increased risk of early thrombotic events post AF ablation. Targeting the inflammatory response during this peri-procedural time frame could aid in maintenance of sinus rhythm post AF ablation.

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