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Topic 21 – Heart failure, cardiomyopathy – B

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0036

Norepinephrine induced apical ballooning syndrome in resuscitation department

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Introduction: Apical ballooning syndrome or Takotsubo cardiomyopathy is an acute syndrome characterized by cardiac failure from disturbances in the contractility of the left ventricle. It is presumably caused by sympathetic over stimulation.

Case presentation: We describe the case of apical ballooning syndrome in a 17-year-old male after receiving accidently 2 mg of norepinephrine.

The patient was admitted to the reanimation unit for severe sepsis with disseminated intravascular coagulation and acute renal failure. The LVEF was controlled at the admission and was normal. After accidental flash of norepinephrine, the patient developed pulmonary edema. The electrocardiogram showed depression of ST segment in apical leads. The transthoracic echocardiogram revealed a large akinesia of the apex which seemed ballooned. The left ejection fraction was about 45%.

During 48 hours, the patient needed vasoactive drug infusions of dobutamine and non-invasive ventilation. There was pseudonormalization of ventricular repolarization on ECG. The evolution was good after 2 days with imporvement of LVFE at chocardiogram. A cardiac MRI performed 7 days later found a normal left ventricular function withaout any late enhancement.

Conclusion: This case illustrates that infection is a condition that could make the myocardium vulnerable to cathecolamine.

0390

Cardiac involvement in systemic lupus erythematosus: about 80 cases

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Introduction: Systemic lupus erythematosus (SLE) is an autoimmune disease with a large clinical polymorphism. Many factors: genetic, endocrine, immunological and environment contribute to disease onset. SLE is characterized by the involvement of multiple viscera, particularly the kidney, the heart and the skin. Cardiac manifestations are relatively common and can involve life-threatening.

Materials and methods: This is a retrospective descriptive study out of ten years, on the records of patients admitted in our department of internal medicine for LES (final diagnosis according to the criteria of the American Rheumatology Association: ACR). We studied the case with cardiac events.

Results: In a series of 80 cases of SLE patients, cardiac involvement was retained in 70% of patients. It was dominated by pericarditis in 52% of cases and it was symptomatic in 30% of them. Ultrasound noted endocarditis in 44% of cases with lesions of the mitral valve in 74% of cases. Myocarditis with left ventricular failure was observed in six patients. These events had occurred at the beginning of the disease in 60% of patients. Indeed, patients had dyspnea in 30% of cases, chest pain in 25% of cases. At ECG, a microvoltage was noted in 40% of cases (pericarditis, myocarditis and endocarditis. The treatment was the strengthening of corticosteroids.

Conclusion: Cardiac manifestations of SLE are severe and asymptomatic in most cases. Their research must be systematic in order to treat earlier. Ultrasound appears to be the first-line examination in the detection of cardiac involvement in SLE. The prognosis remains severe coronary insufficiency and rhythm disorders.

0391

Cardiac involvement in ankylosing spondylitis

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Background: Cardiac involvement in ankylosing spondylitis is common. We have tried through this study to analyze the cardiovascular events among 50 patients with ankylosing spondylitis.

Methods: A retrospective study including 50 patients with ankylosing spondylitis. All patients underwent a complete physical examination with a heart and lung auscultation and an electro-cardiogram (ECG). Transthoracic ultrasound was performed whenever there was an abnormal physical examination and/ or ECG.

Results: The study included 47 men and 3 women, the sex ratio is 15,6. The average age of onset was 26 ± 7 years. The mode of onset is axial in 95% of cases (low back pain and/or buttock). The extra-articular manifestations are present in 54% of cases. Cardiac involvement is present in 9 cases (18% of cases). The reason for consultation is dyspnea in 2 patients. In other cases, cardiac involvement was discovered incidentally. Aortic regurgitation was noted in 4 patients. Mitral insufficiency was found in 3 cases. Two patients have predominantly septal hypertrophic cardiomyopathy and one patient presented an array of pulmonary insufficiency. The average time of onset of cardiac involvement was 8 ± 5 years. All patients were put under special medical treatment of their heart, with good clinical outcome.

Conclusion: Cardiac involvement in ankylosing spondyllitis is seen more frequently in men, especially in the old cases. A close relationship between time to onset of aortic insufficiency and duration of disease progression was found. It would be responsible for one third of deaths of patients. It was significantly more frequent in HLA B27 positive patients (especially complete atrioventricular block and aortic insufficiency).

0249

Iron overload does not potentiate doxorubicin induced cardiotoxicity in vivo in mice and in vitro in cardiomyocytes cell cultures

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Background: Doxorubicin (DOX), an anticancer anthracycline, is known to induce serious cardiotoxicity, which is believed to be mediated by oxidative stress and complex interactions with iron. However, the relations between iron metabolism and DOX-induced cardiotoxicity remain a matter of controversy.

Methods: Firstly, we used an in vivo murine model of iron overloading (IO) where male C57BL/6 mice received during 3 weeks (D0-D20) a daily dextraniron injection (15 mg/kg/day.) and then (D21) a single dose of 6 mg/kg DOX. We evaluated cardiac function with echocardiography, myocardial gene's expression, nitro-oxidative stress levels and iron status. Secondly, the anti-pro-liferative activity of DOX, in combination with dextraniron, was evaluated in vitro in cultures of cancerous cells (EMT-6) or cardiomyocytes (H9c2).

Results: At D30, there was a significant decrease in left-ventricular ejection fraction (LVEF) in all groups of DOX-treated mice. In IO mice treated by DOX, the LVEF fall was not majored and there was no increase in atrial natriuretic peptide mRNA cardiac gene-expression. IO alone resulted in cardiac hypertrophy and up-regulation of b-myosin heavy-chain expression. In myocardial tissue, electron spin resonance spectroscopy revealed an increase in intro-oxidative stress in IO groups. While 1 μ M of DOX induced a significant reduction of EMT-6 or H9c2 cells proliferation, dextran-iron (125-1000 μ g/mL) alone did not modify cell viability and did not impair DOX cytotoxicity.

Conclusions: IO did not result in a significant increase in DOX cardiotoxicity neither in mice, nor in cardiomyocytes, and did not impair DOX capacity to inhibit cancerous cells proliferation.