HYPERTROPHIC CARDIOMYOPATHY WITH LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION. CLINICAL CASE PRESENTATION

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Background. Hypertrophic cardiomyopathy (HCM) is caused by genetic defects in sarcomeric proteins, with autosomal dominant inheritance. The estimated prevalence is 1:500 cases. It may remain undiagnosed for a long time, being often asymptomatic or presenting with non-specific clinical manifestations.
Objective(s). Clinical case presentation of a patient with obstructive HCM, positive family history, who underwent surgical treatment, outlining the importance of early diagnosis and family screening.
Materials and methods. Patient Z, 64 years old, was admitted to the cardiology department with the following symptoms: constrictive retrosternal pain, dyspnea on medium exertion, syncope. Anamnestic data were collected, clinical and paraclinical examination (laboratory tests, electrocardiogram, echocardiography and chest X-ray) was performed.
Results. Anamnesis: the patient's daughter was diagnosed with HCM at the age of 16. EKG: sinus rhythm, HR 84 b/min, signs of left ventricular (LV) hypertrophy. EchoCG: normal size heart chambers, severe LV myocardial hypertrophy with LVOT obstruction (PGmax 55mmHg), sigmoidal IVS (28/22mm) with muscle band fixated on the antero-lateral pillar, EF 65%, moderate-to-severe MV regurgitation. Laboratory biomarkers within normal limits. Morrow septal myectomy was performed with complete mitral valvuloplasty. Postoperatively, EchoCG shows regression of LV hypertrophy, without LVOT obstruction (IVS 15/21mm PG max 12.5 mm Hg), mild MV regurgitation, EF 59%.
Conclusion(s). Patients with hypertrophic cardiomyopathy require early diagnosis through careful history taking, Holter ECG, echocardiography, MRI, genetic testing. Screening of first-degree relatives is of paramount importance in order to prevent fatal arrhythmias and sudden cardiac death.
Keywords: hypertrophic cardiomyopathy, LVOT obstruction, myectomy

ETIOPATHOGENETIC ASPECTS OF SUPRAVENTRICULAR TACHYCARDIA IN YOUNG PEOPLE

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Background. Supraventricular tachycardia (SVT) is the most common arrhythmia diagnosed in young people. The prevalence of SVT in young adults is about 2,29/1000 people, with a higher incidence in women vs men. Studying the etiology of SVT is essential for optimizing diagnosis, prophylaxis and treatment.
Objective(s). Studying data from the scientific medical literature regarding the etiological and predisposing factors involved in the pathophysiology of supraventricular tachycardia in young people.
Materials and methods. A systematic review of the National Library of Medicine and the International Institutes of Health MEDLINE was performed, using the authentic databases UpToDate, Embase, and PubMed, to search for works focused on etiopathogenetic aspects of supraventricular tachycardia in young people. 32 bibliographic sources were analyzed.

Results. SVT is the result of reentry circuits, abnormal automatism or trigger-driven activity. Factors contributing to SVT in young people include: congenital heart defects (Wolff-Parkinson-White Syndrome, Ebstein's anomaly, atrial septal defects, valvulopathies, which create abnormal electrical pathways), electrolyte imbalances, genetic predisposition, female gender, stress, anxiety, alcohol, caffeine, medications, smoking, physical exertion, thyroid pathologies, cardiac (coronary artery disease, heart failure, valvular diseases, cardiomyopathy, heart surgery), pulmonary pathologies, DM, pregnancy, dehydration, recreational drugs.
Conclusion(s). SVT in young people is characterized by a wide spectrum of etiological and risk factors, which requires a multidisciplinary medical approach, including education, counseling, promotion of a healthy lifestyle, in order to increase the quality of life and decrease the risk of complications.
Keywords: supraventricular tachycardia, etiopathogenesis, young

PERIPHERAL NERVE COMPRESSION IN THE LOWER LIMBS OF ATHLETES – CLINICAL, DIAGNOSTIC, AND THERAPEUTIC ASPECTS

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Background. Peripheral nerve compression is a common yet frequently overlooked cause of lower limb pain in athletes. Without timely diagnosis, it can delay return to sport. The underlying mechanisms include repetitive microtrauma, anatomical variations, and both external and internal sources of compression.
Objective(s). To analyze peripheral compression neuropathies in athletes, diagnostic methods, and therapeutic strategies, with particular focus on enabling a swift return to athletic performance.
Materials and methods. A literature review and synthesis of scientific sources addressing the anatomy and pathophysiology of peripheral nerves, as well as the interpretation of documented symptoms of the most common peripheral neuropathies in the lower limb among athletes, were carried out through the consultation of search sources such as PubMed and MedScape.
Results. The most frequently identified neuropathies affected the common peroneal nerve (dorsiflexion weakness, dorsal foot paresthesia), deep peroneal nerve (anterior tarsal tunnel syndrome), superficial peroneal nerve (entrapment in the distal third of the leg), posterior tibial nerve (tarsal tunnel syndrome), sural nerve (posterolateral ankle pain), and plantar digital nerves (Morton's neuroma). Imaging studies revealed morphological alterations of the affected nerves, while electrodiagnostic evaluations allowed differentiation between demyelinating and axonal damage and provided insights into the severity and chronicity of the condition.
Conclusion(s). Peripheral nerve compressions are often underdiagnosed in athletes. Accurate diagnosis requires a thorough clinical evaluation supported by imaging and electrodiagnostic testing. Improved awareness and timely management can accelerate recovery and optimize the return to athletic activity.
Keywords: compression neuropathy, nerve, nerve decompression, pain