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Cardioembolic events in young patients: beyond the heart

Dear Editor,

Among cardioembolic events, nonvalvular atrial fibrillation remains the most common etiology. However other potential major cardioembolic sources include recent myocardial infarction, left ventricular aneurysm, rheumatic valve diseases, mechanical valve prosthesis, or infective endocarditis. On the other hand, unclear risk sources include mitral valve prolapse, calcified aortic stenosis, defects at the atrial septum (including the patent foramen ovale (PFO)) and right-to-left shunting at the pulmonary level due to pulmonary arteriovenous fistula.

PFO has been associated with numerous conditions such as decompression illness in divers, migraine, high-altitude pulmonary edema, obstructive sleep apnea syndrome and cerebrovascular and coronary ischemia. In patients with PFO the risk of embolization depends, mostly, on the increased right atrial pressure caused by the tricuspid valve regurgitation and the size and function of the right ventricle. This may lead to transient shunt reversal and paradoxical embolization. In fact, in the absence of demonstrable elevation of the right atrial pressure caution should be exercised to incriminate the PFO or the atrial septal defect in paradoxical embolism. Nonetheless, other factors such as atrial fibrillation, an atrial septal aneurysm, a prominent Eustachian valve, a Chiari network, an enlarged right atrium, a large shunt size or a right-to-left shunt detected by resting or Valsalva maneuver echocardiogram may explain cryptogenic vascular events in young patients[1]. Likewise, patients with recent paradoxical embolism usually have simultaneous deep vein thrombosis or pulmonary embolism. In fact, young patients with cryptogenic transient ischemic attack or stroke and PFO should be evaluated for lower extremity or pelvic venous thrombosis, which would also be an indication for anticoagulation[2]. Also, considering the high risk of intracranial bleeding with thrombolysis in pulmonary embolism, which may be partly due to hemorrhagic transformation of subclinical strokes, screening PFO with transesophageal echocardiography should be considered in acute intermediate-risk pulmonary embolism when thrombolytic treatment is discussed[3].

Also, an uncommon consequence of intermittent right-to-left shunting is the platypnea-orthodeoxia syndrome. In it, an anatomical and a functional component must coexist. The former may be an atrial septal defect or a patent foramen ovale. The latter may be a pericardial effusion, a constrictive pericarditis, pulmonary emphysema, an arteriovenous malformation, a pneumonectomy or an aortic aneurysm/elongation that produces a deformity in the atrial septum and results in a redirection of shunt flow with the assumption of an upright posture[4].

Although it is possible that closure of the defect would reduce the risk of paradoxical embolism and cryptogenic strokes, there is still no definitive evidence that closure of PFO is better than medical therapy. In fact, when cryptogenic ischemic events are treated with antiplatelet therapy or anticoagulation therapy, the recurrence rate is the same whether or not the patient has a PFO. On the contrary, PFO closure should be addressed in specific clinical situations, such as recurrent cryptogenic stroke in young patients (<55 years) with evidence of venous thrombosis or high-risk anatomic features, medication failure or contraindications to anticoagulation[1,5]. On the contrary, if the patient has severe pulmonary arterial hypertension with associated right-to-left shunt the PFO closure in contraindicated.

Best regards,

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