The hypodense artery sign in acute cerebral infarction

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A 77-year-old man was admitted with severe stenosis of both left anterior descending and circumflex coronary arteries and mitral valve regurgitation. He underwent mitral valve replacement and coronary artery bypass grafting (CABG). During the second post-operative day, sedation having been stopped, the patient presented a left hemiplegia and homolateral central facial paralysis.

Head CT performed in emergency showed a large hypodense area in the right cerebral hemisphere (arrowheads on Fig. A) corresponding to an infarction in the right middle cerebral artery territory as well as a bifurcated longitudinal hypodensity (arrows on Fig. A). CT angiography (Fig. B) demonstrated a hypodense filling defect located in the right segment of the middle cerebral artery (arrow) with a -56 HU negative density value suggesting its fatty embolic nature.

Thrombolytic treatment is not effective in this pathology and due to the patient’s age decompression craniotomy was not indicated. He died one month later due to general deterioration.

Comment

Causes of cerebrovascular infarction after cardiac surgery are many. Most frequently, emboli originate in the heart, particularly in the setting of atrial fibrillation. Persistent atrial fibrillation is the most significant risk factor for stroke after mitral valve replacement. Emboli can also propagate from the carotid arteries as a complication of stenotic or complicated atherosclerotic plaques.

Cerebral fat embolism is a rare cause of thromboembolic stroke. Fat emboli that usually originate from long bone fractures are most commonly described in the trauma and orthopedic literature, but are also described after cardiac surgery with sternotomy and paradoxically brain embolization in patients with venous-arterial shunt in the lung and patients with patent foramen ovale.

In other cases, mitral valve replacement procedure may dislodge a macroscopic portion of fat from the cardiac or surrounding tissue which subsequently embolizes in the cerebral artery, or the fat embolus may originate from a fatty atherosclerotic plaque within the carotid artery or aortic arch.

In our case, the diagnosis of fat embolism was suggested on the basis of the negative attenuation values of the arterial cerebral defect in the context of cardiac surgery.

A hypodense middle cerebral artery sign associated with acute stroke from a fat embolus has been described in 3 cases in the literature, all of them with a poor vital prognosis.

Reference


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