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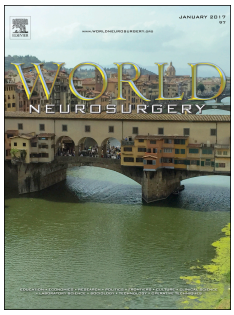

Plateletpheresis: non-operative management of symptomatic carotid thrombosis in a patient with reactive thrombocytosis

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Plateletpheresis: non-operative management of symptomatic carotid thrombosis in a patient with reactive thrombocytosis.

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Introduction:

Extracranial carotid artery intraluminal thrombus is an important cause of acute ischemic stroke or transient ischemic attack (TIA). It is a rare condition and the actual incidence remains unknown. The most common pathology associated with an intraluminal carotid thrombus is underlying atherosclerosis.[1] In cases where an underlying plaque is not identified, the condition may be associated with a hypercoagulable state, use of CNS stimulant drugs, iron deficiency anemia, or thrombocytosis.[2] Few case reports describe the association between reactive thrombocytosis (RT) and stroke. Thrombocytosis can occur as a response to a variety of stimuli such as iron deficiency, infections, trauma, malignancies, inflammation, hemorrhage and burns. Currently there are no clear recommendations for treatment of ischemic stroke associated with thrombocytosis. Plateletpheresis has been suggested as an accepted temporizing measure to reduce platelet count in patients with thrombocytosis associated active thrombosis; however, there is no reported case of its use in acute ischemic stroke. [3]

Case Report:

A 55-year-old female with past medical history of menorrhagia due to uterine fibroids who presented to the Emergency Department (ED) with acute onset left-sided weakness and left facial droop 4.5 hours prior to the arrival. By the time of her evaluation in ED the neurologic deficits had almost completely resolved except for left hemisensory deficit, making her National Institute of Health Stroke Scale (NIHSS) 1. The initial non-contrast computed tomography scan (CT) was non-revealing. Laboratory results revealed mild hypochromic anemia and a platelet count of 1014 x 10^3/mL. She was not a candidate for thrombolytic therapy due to the time window and her mild non-disabling residual deficit. Thirteen hours after admission she had acute worsening of the neurological symptoms. Her NIHSS was now 18 (LOC 1 gaze, 2 visual fields 2 facial palsy 2 left arm 3 left leg 3 sensory 2 dysarthria, 1 extinction 2). A CT angiogram showed an acute ischemic infarction involving the right middle cerebral artery territory with non-occlusive intraluminal filling defect within the right carotid bulb (Figure 1a and 1b). The patient was immediately transferred to the Neuro Intensive Care Unit and initiated on antiplatelet therapy with aspirin 325 mg and a heparin intravenous infusion. An MRI Brain without contrast showed acute infarction involving the right middle cerebral artery (MCA) territory (Figure 2).

Peripheral blood smear showed microcytic hypochromic red blood cells, occasionally pencil shaped and increase in platelet number with variation in size. Janus kinase 2 (JAK2) mutation was negative. She was also ruled out for other hypercoagulable states. Anemia work-up confirmed severe iron deficiency anemia (iron level 11, percent saturation 2.5% and ferritin 3) and mild Vitamin B12 deficiency (305 pg/mL). After ruling out a myeloproliferative neoplasm, the cause of her elevated platelet count was determined to be due to reactive thrombocytosis secondary to iron deficiency anemia.