Clinical Image

Unusual Thrombus Revealed by an Ischemic Stroke in Essential Thrombocythemia

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CLINICAL IMAGE

A non-smoked 56 year-old man was admitted to our stroke unit for a sensorimotor deficit of right upper limb. He reported a 10-years history of essential thrombocythemia (ET) with JAK2V617F mutation, without thrombotics complications, and treated with hydroxyurea (75 mg per day).

He was treated for hypertension by lercanidipin (20 mg per day), supraventricular extrasystoles (SVE) by nebivolol (5 mg per day), associated with aspirin (75 mg per day) and atorvastatin (40 mg per day).

On admission, the NIH Stroke score was three.

Body temperature was 37°C. Serum platelet and leukocyte counts were 390x10⁹/L (normal: 150-500x10⁹/L) and 14.1x10⁹/L (normal: 4.10-10.50x10⁹/L), respectively. C-reactive protein (CRP) level was 6.7 mg/l (normal<4.0).

The magnetic resonance imaging (MRI) of the brain showed a small cerebral infarction on the left rolandic area on diffusion weighted (DWI) sequences (Figure 1A) without abnormalities on brain fluid attenuated inversion recovery (FLAIR) sequences. There was no intracranial arterial occlusion on magnetic resonance angiography.

Two-dimensional echography of cervical arteries showed somenon-obstructiveatheromatous lesion of carotid bifurcations. Larger examination revealed 3 cm bellow the left bifurcation, a 6x16 mm free floating thrombus attached to the wall of the left common carotid (Figure 1B). The lesion was confirmed by scanner angiography showing an endovascular defect.

Then surgical thrombectomy was performed. The histopathological examination confirmed a thrombus with abundant white cells (Figure 1C).

After surgical thrombectomy, leukocyte decreased to 10x10⁹/L and CRP to 4.6 mg/L. Then the patient was discharged without complications with null NIHS score. However, because SVE, transoesophageal echocardiography was performed, excluding left atrium or left atrial appendage thrombi.

Therefore, the treatment was strengthened by adding anti-platelet clopidogrel (75 mg per day).

DISCUSSION

To our knowledge, it is the first description of a rich leukocyte’s thrombus, responsible of a cerebral thrombo-embolism in ET.

Ischemic stroke can complicate a known ET or reveal it. Despite the best effective therapy combining hydroxyurea and antiplatelet therapy, our patient developed a rare thrombotic complication. Moreover, even if the number of platelets is less than 600x10⁹/L, the involvement of ET cannot be ruled out [1].

Hypertension and JAK2V617F mutation are predictor factor of vascular thrombosis risk [2]. At the same time, strongly association between level of leucocytes and risk of thrombosis exist [3]. Although interaction between leukocytes and arteriosclerosis are not totally understood [4], atherosclerotic history of this properly treated patient underlines the high risk of endothelial dysfunction, increasing the risk of thrombosis in the presence of leukocytosis [5].

Our observation emphasizes the need for monitoring also white cell count in such patient.
Figure 1 1A: DWI sequences of MRI: small cerebral infarction on the left rolandic area (black arrow).
B: Longitudinal two-dimensional echography of the left common cervical artery: free floating thrombus attached to the posterior wall.
C: Hematoxylin-eosin coloration of thrombus: abundant leukocytes in our case report (left panel) as compared to a common thrombus (right panel).

REFERENCES


