Severe ischemic stroke in a young man with active EBV infection

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Background

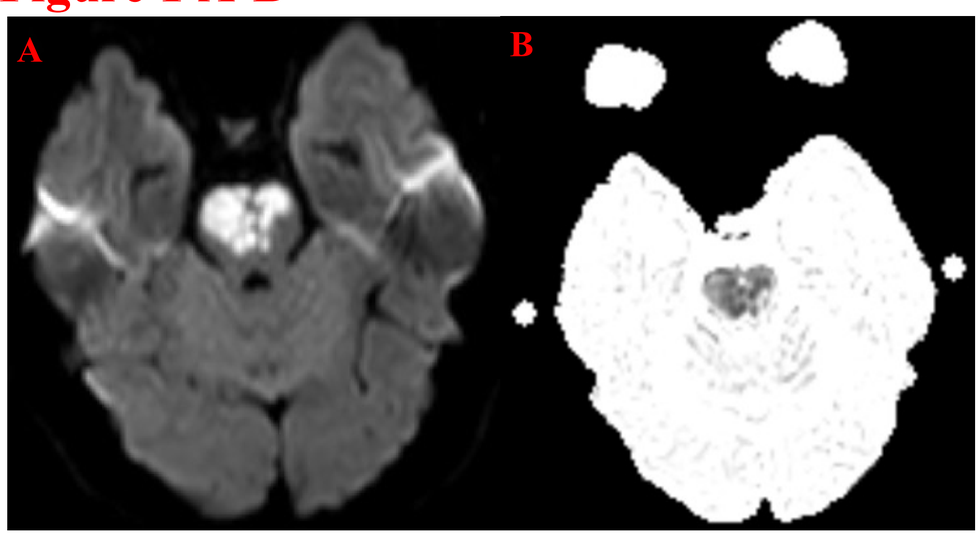
Epstein-Barr virus (EBV) infection has been associated with many neurologic manifestations, including encephalitis, transverse myelitis, and Guillain-Barre syndrome.[1] Cerebral infarction associated with EBV infection has been reported only in two cases [2,3], thus confirming that ischemic stroke is an extremely rare, but possible, complication of infectious mononucleosis.

Case report

A 21- year-old boy was admitted to our hospital because of acute left hemiplegia, anarthria, and confusion. Mild bilateral cervical and inguinal lymphadenopathy had been recognized for two weeks prior to hospitalization. On admission the neurologic examination revealed anarthria, confusion, left brachiocrural hemiplegia and facial palsy in the right side. Deep tendon reflexes were slightly exaggerated with positive Babinski's sign on the left side. Pharyngitis, skin rash, lymphadenopathy and hepatosplenomegaly were absent. Blood examination reported leukocytosis (17.03 x 10e3/uL, with segmental neutrophil 60.5% and lymphocyte 30.1%). Biochemistry, fasting blood sugars, lipid profile, inflammatory markers, thrombophilic and autoimmunity tests were within normal ranges. Cytomegalovirus, Toxoplasma, Rubella, Herpes and Human Immunodeficiency Virus I/II IgM antibodies were negative. Anti-EBV IgM titer was 160 UI/mL. EBV-DNA detected by quantitative-PCR was 9200 copies/mL in serum (reference: <100 copies/mL), thus confirming active EBV infection. CSF was negative for acute infections and revealed normal glycorrhachia and proteinorrachia, no cells and negative oligoclonal bands. Brain MRI showed a DWI-positive pontine lesion, with lower signal intensity on apparent coefficient map (Figure 1A-B), consistent with acute infarction. MR angiography characterized a distal stenosis of the basilar artery (Figure 1C). Color-coded carotid, transthoracic and transesophageal echocardiography were unremarkable. Electrocardiogram and 24-hour Holter did not demonstrate any arrhythmia. Patient was diagnosed with ischemic stroke and active EBV infection. He started on a course of i.v. methylprednisolone, acyclovir and antiplatelet therapy (aspirin 300 mg/die).

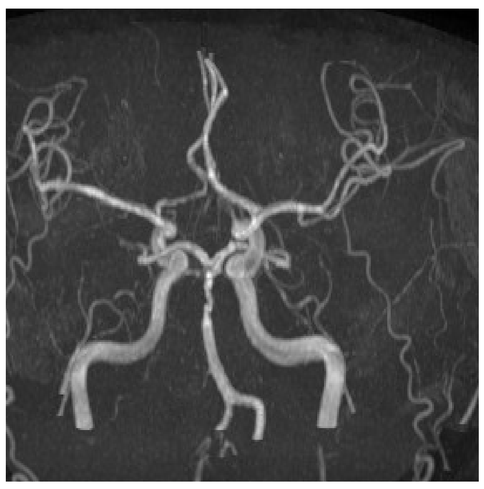
His status improved during the following days and neuroimaging follow-up was negative for new ischemic lesions.

Figure 1 A-B



(A)Axial DWI MRI image showing a pontine hyperintense lesion.
(B)MRI apparent coefficient map demonstrating lower signal intensity of the pontine lesion.

Figure 2



MR angiography showing a distal stenosis of the basilar artery.

Conclusion

Systemic infections increase the risk of stroke by changes in lipid metabolism, fibrinogen increase, platelet activation/aggregation, hypercoagulation, alteration in endothelial function.[4,5] Post-infectious inflammatory mechanisms have been proposed to explain the pathogenesis of cerebral infarction in patients with viral infections. It has been assumed that vasculitis may occur following either direct infection to vessels or post-infectious immune-mediated responses. EBV infection has to be taken into account in young patients with ischemic stroke of unknown etiology.

References

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