Clinical Image

Bilateral, Simultaneous, and Extensive Infarction of Basal Ganglia-A Clinical Image

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CLINICAL IMAGE

A 28-year-old single female was brought to our emergency department after developing progressive impairment in consciousness over few hours. Her past medical and surgical histories were unremarkable and there was no family history of note. Her sister denied any history of head trauma or drug abuse. Her Glasgow coma scale was 4/15 and there were no fever, neck stiffness, or local/lateralizing signs. The patient’s brain CT scan/MRI films are shown in figure 1. Blood tests, including thyroid stimulating hormone, rheumatoid factor, anti-nuclear factor, anti-phospholipid antibodies, lupus anti-coagulant, and prothrombin and thromboplastin times were within their normal reference range. Blood and urine toxicology screens were negative. 12-lead ECG and trans-thoracic echocardiographic examinations were normal. Carotid and lower limbs Doppler studies revealed normal findings. Cerebrospinal fluid examination was unremarkable. Brain MRV (magnetic resonance venography) showed patent cerebral venous sinuses and deep venous system. Because of the lack of expertise in our hospital, conventional cerebral angiography was note done. The human basal ganglia (corpus striatum) receive its blood supply mainly from the medial and lateral lenticulostriate branches of M1 and M2 segments of the middle cerebral artery and a contribution also comes from the recurrent artery of Huebner (which stems from the A2 segment of the anterior cerebral artery). However, the anterior choroidal and anterior communicating arteries share a minor role in this blood supply. The middle cerebral artery sometimes gives off direct small perforators to the basal ganglia, but these blood vessels contribute very little to the overall blood supply [1,2].

Accordingly, acute and extensive infarction of both basal ganglia principally requires simultaneous occlusion of deep perforating lenticulostriate branches of both middle cerebral arteries and the arteries of Huebner. According to Russmann and coworkers, [3] an embolic etiology is the commonest cause behind such extensive and bilateral infarctions. Other potential causes are cerebral vasculitis, pan-cerebral hypoperfusion, poisoning/intoxication (e.g., with cyanide or Carbone monoxide), drug abuse (e.g., cocaine), head trauma, and supratentorial neurosurgical procedures [1]. We were unable to uncover the etiology behind the patient’s presentation. Conventional angiography, transthoracic echocardiography, and genetic testing are needed to reach this goal.

REFERENCES


Figure 1

A. Urgent non-contrast CT brain scan of the patient at the time of emergency department admission. There are bilateral hypodense lesions within both basal ganglia. Panels B, C, and D are axial T1-weighted, coronal T2-weighted FLAIR, and axial T2-weighted MRI films (respectively) which were done after 6 hours of admission. Note that the ischemic infarction has involved the putamen, globus pallidus, caudate nucleus, and anterior limb of the internal capsule on both sides. The overlying cortex and adjacent thalami and spared.