

Cerebral infarction in pediatric acquired immunodeficiency syndrome

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Abstract. Cerebral infarction is an uncommon complication of AIDS in pediatric patients. We have seen three HIV-infected children who developed acute neurological deficits due to stroke. Cerebral infarction must be considered in the work-up of a child with AIDS who presents with focal neurological deficit, seizure or mental status change.

Stroke is a complication of HIV infection that occurs in approximately 1% of affected children [1]. At autopsy, evidence of cerebral infarction was documented in 10–30% of children with HIV infection [2]. We have seen three children with focal infarction who are HIV positive.

Case reports

Case 1

An 8-year-old HIV-positive boy presented with sudden onset of right hemiparesis and a right facial palsy.

Past medical history included multiple hospitalizations for recurrent oral and esophageal candidiasis. Laboratory work-up revealed spinal fluid which was negative for organisms or infection. Platelets, prothrombin, and partial thromboplastin time were normal. On physical examination, the patient was afebrile and alert with a right facial palsy and a right hemiparesis. There were brisk deep tendon reflexes and an up-going right toe.

Initial non-enhanced axial computed tomography (CT) of the head revealed evolving low-attenuation lesions of the left basal

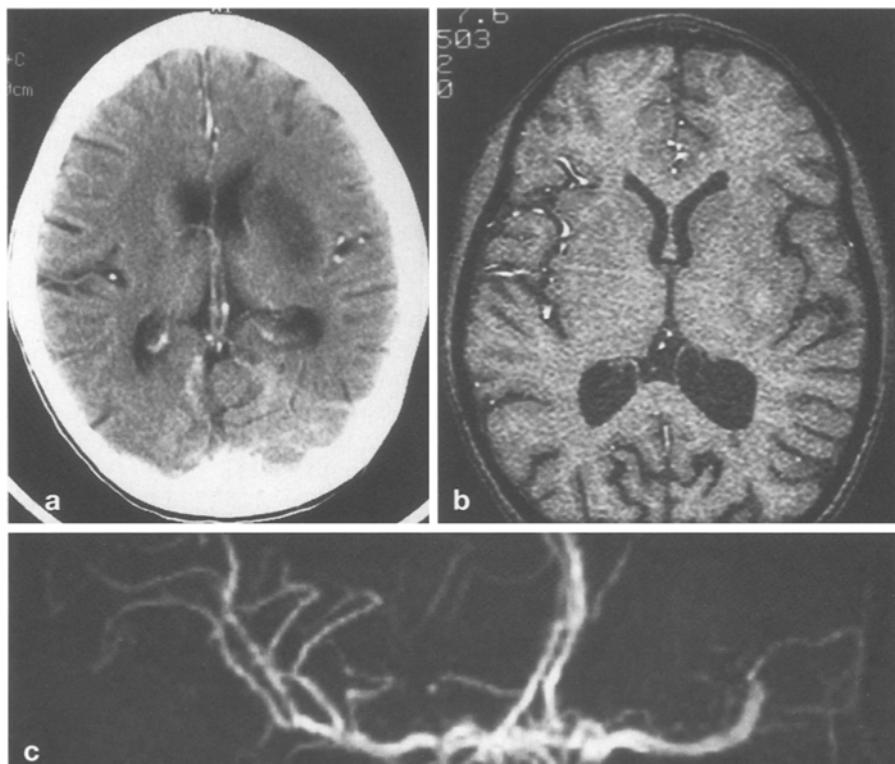


Fig. 1a-c. Case 1. **a** CT shows low-attenuation lesions in the left basal ganglia. **b, c** MRA demonstrates absence of signal within the branches of the left middle cerebral artery on both the source image and the coronal reconstructed image

ganglia (Fig. 1a). A magnetic resonance angiogram (MRA) of the head revealed absence of signal within the sylvian branches of the left middle cerebral artery on both the source images (Fig. 1b) and the coronal reconstructed images (Fig. 1c). These findings correlated with the CT findings demonstrating an evolving left middle cerebral artery stroke.

Case 2

A 14-year-old HIV-positive girl presented with progressive neurological signs and ataxia.

Laboratory and clinical work-up revealed no evidence of acute infection. A magnetic resonance imaging (MRI) scan demonstrated a left cerebellar hemisphere high T2-weighted signal lesion in the distribution of the left posterior inferior cerebellar artery. These findings are consistent with a left cerebellar infarction (Fig. 2).

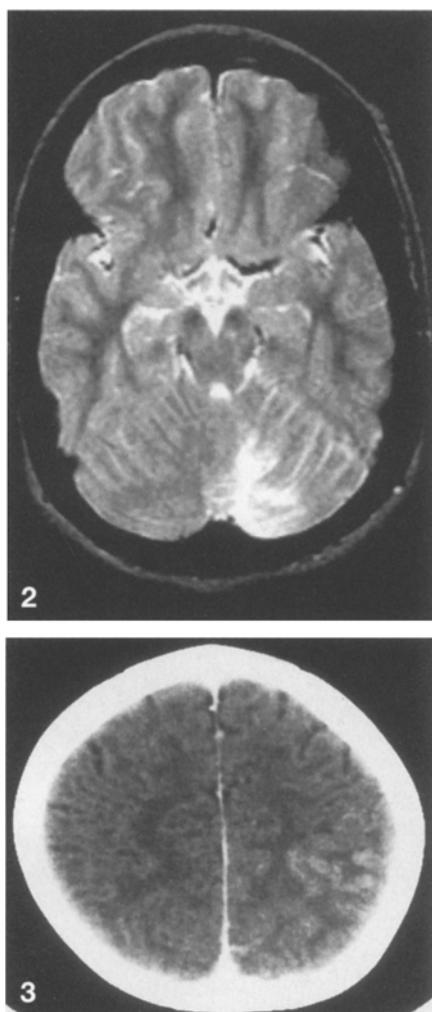


Fig. 2. Case 2. MRI shows a left cerebellar hemispherical lesion with high T2-weighted signal intensity, consistent with an infarction

Fig. 3. Post-contrast CT shows an area of patchy gyriform enhancement in the left parietal lobe

Case 3

A 2-year-old HIV-positive boy presented with a right hemiparesis and lethargy.

Past medical history included numerous hospital admissions for pneumocystis pneumonia and otitis media. Laboratory work-up revealed spinal fluid which was negative for organisms, and toxoplasmosis titers were also negative. On physical examination the patient was afebrile with a right hemiparesis and a right facial palsy.

The post-contrast CT demonstrated a vague area of patchy gyriform enhancement in the left parietal lobe (Fig. 3). A study 4 days prior to this, in retrospect, demonstrated a vague loss of gray/white discrimination in this region. These findings are most typical of an evolving cerebrovascular accident in the left parietal lobe.

Discussion

Patients with AIDS are at an increased risk of strokes [3]. The cause of these strokes is often unknown, however, vasculopathies of various etiologies have been implicated in many cases [2, 3]. Opportunistic infection may lead to vasculopathy and subsequent stroke in patients with AIDS [1]. Pathogens such as *Candida albicans* and cytomegalovirus are known to cause vascular invasion and secondary thrombosis of cerebral vessels in the immunocompromised host. However, in children, in contrast to adults, infection of the CNS with opportunistic organisms is uncommon [4].

Lymphoma is a known neoplastic cause of vasculopathy in the immunocompromised host. This tumor can be focally invasive of blood vessel walls resulting in direct vascular injury, invasion of the lumen and focal thrombosis [1].

The vasculopathy in patients with AIDS also may be due to primary infection by the human immunodeficiency virus itself. In addition, HIV may indirectly mediate vascular injury by the reaction of elastases from repeated infections. This may injure the elastic lamina of vessels [5]. These strokes at the site of vasculopathy are thought to be thrombotic rather than embolic vascular occlusions [2].

Hemorrhagic infarcts also have been reported in children with AIDS although these are reported

to occur less frequently than non-hemorrhagic infarcts. In one report of four patients with hemorrhagic infarcts, all had immune thrombocytopenia.

The three patients presented had clinical evidence of stroke with abrupt onset of neurological deficits. Imaging studies confirmed clinical suspicions revealing characteristic lesions that corresponded to the area of focal deficit. Clinically and radiologically there was no evidence of infection or lymphoma in these patients. CNS lymphoma and opportunistic infections are distinctly unusual in children with AIDS, in contrast to adults. Therefore, we believe that cerebral infarction in these patients was due to primary infection by the HIV virus itself. These children are alive and undergoing continued medical therapy for AIDS at this time. Cerebral infarction secondary to HIV vasculopathy should be a consideration in pediatric AIDS patients.

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