HIV Infection Presenting with Stroke and Epilepsia Partialis Continua

Till date there are only few case reports of HIV infection in children, presenting with stroke and none with epilepsia partialis continua (EPC) as their first manifestation [1,2]. EPC as a neurological manifestation of HIV has been described previously, only rarely in children [3,4]. Here we report a child presenting with stroke and EPC, later diagnosed to be infected with HIV.

A 6-year old boy, developmentally normal, admitted with illness beginning with fever and abnormal jerky movements of right upper limb lasting initially for few seconds, progressing over next few days to involve right lower limb followed by involvement of right half of face. Seizures, at presentation, persisted during most times with amplitude decreasing during sleep. Child on examination had cervical lymph nodes palpable, liver 2 cm and spleen 2 cm palpable below costal margins. Patient had reduced level of alertness without meningeal signs and normal fundus. Child had right upper motor neuron facial nerve palsy, weak gag, aphasia and right hemiplegia. Reflexes were exaggerated in all four limbs with ankle clonus present. Lab investigation revealed prothrombin time 11 seconds, partial thromboplastin time 22 seconds, total serum protein 9.4 g%, albumin to globulin ratio 2.3/7.1, HIV ELISA positive, anti nuclear antibody negative, toxoplasma IgG 1:1600 and IgM <1:800, VDRL non reactive, anticardiolipin antibodies and factor V Leiden mutation absent. Contrast MRI of the brain (34th day of illness) revealed infarct involving left basal ganglia and left insular region (middle cerebral artery territory) and frontal cortex (anterior cerebral artery territory) with MRA showing no evidence of any vascular malformation. Multivoxel spectroscopy was suggestive of bilateral basal ganglia infarct. Later patient's sib and both parents were tested and found positive for HIV. Cerebrospinal fluid (CSF) examination (1 month of illness) results showed leucocytes absent, proteins 38.2 mg/dL, sugar 39 mg/dL, Gram stain negative, culture sterile, cryptococcus (India ink staining), Japanese encephalitis (PCR and serology), Herpes simplex virus 1 and 2 (PCR & serology), tuberculosis (adenosine deaminase, culture and PCR) were found to be negative. Electroencephalogram (EEG) was suggestive of diffuse encephalopathy left more than right with focal epileptiform discharges. Cardiac echocardiography was normal.

Cerebrovascular complications are associated with perinatal HIV infection, albeit as a rare presentation. Mechanisms underlying the increased risk for ischemic stroke in HIV infected include opportunistic infections, meningitis and vasculitis, primary HIV vasculopathy, altered coagulation, and cardioembolic events [5]. In absence of vasculitis, HIV-related vasculopathy may cause stroke, which was the most likely etiology in our case.

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Rickettsial Diseases

We read with interest the article on Rickettsial diseases by Rathi, *et al.* [1]. The author mentions that no case of Ricketssia has been reported from Madhya Pradesh. We herein report a child with features suggestive of rickettsial infection, whom we recently managed.

A 1-year 6 month old child from Shajapur, MP, presented to us with a history of fever for 20 days. On day 3, she had developed a generalized rash starting from the

trunk which spread to the palms and soles over the next few days. On day 5, she developed congestion of conjunctiva and of the oral cavity and on day 7 she had left focal seizures. She was admitted at a local hospital where her investigations revealed a Hb of 7.0 g/dL, a normal leukocyte count, and platelet count of 80,000/cc. Her CSF examination was not done. She received antibiotics and anti convulsants for a week. However since her fever did not resolve she was referred to us. On examination she was irritable, had a generalized maculopapular, petechial rash all over the body, including palms and soles. She had edema over the dorsum of hands and feet but no organomegaly, lymphadenopathy or meningeal signs. Her total leukocyte count was 29,800 per cu mm with polymorphs of 84%, platelet count was 4.5 lakhs, ESR was 60, and CRP was elevated. Weil Felix test was positive in a titre of 1: 160 for Proteus Ag Ox 19 and 1: 80 for Proteus Ag Ox K. She was started on oral doxycycline. She became afebrile in 48 hours.

We used the clinical scoring system described by Rathi, *et al*. [1] and she had a score of 17 which is considered to have a specificity of 100%. We could not do the Elisa for IgM antibodies to Rickettsia because of financial constraints.

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Visceral Leishmaniasis (Kala-azar) without Splenomegaly

We are reporting an unusual case of visceral leishmaniasis (VL) in a 7-year-old male, presenting without splenomegaly

A 7-year-old male child presented with fever often associated with rigor and chills and loss of weight and appetite for last one month. There were no other significant localizing symptoms. A general physical examination revealed significant pallor. Systemic examination, including absence of organomegaly on abdominal evaluation, was non-contributory. Hematological parameters revealed pancytopenia with hemoglobin 4 g/dL, total leukocyte count of 2100 (N-22%,L-74%,E-2%,M-2%,B-0%), ESR in first hr 86mm, platelet count of 38000 and peripheral smear showing a leucoerythroblastic picture. Hypregama-globulinemia with albumin globulin ratio of 0.3 was seen. Liver function test on admission was normal. Peripheral smear for microfilaria was negative. Human immunodeficiency virus (HIV), Hepatitis B (HBsAg), hepatitis C (HCV) and dengue serology were negative. Chest *X*-ray was normal and Mantoux test was negative. Urine culture showed no growth. Ultrasound of whole abdomen was a normal. RK-39 was positive. Bone marrow examination was done, which revealed Leishmania Donovani (LD) bodies.

A final diagnosis of kala- azar was made. Absence of splenomegaly was outstanding finding in our patient. Patient was started on amphotericin B and other supportive therapy including blood and platelet transfusions. The patient improved and was discharged after giving full course of amphotericin B.

VL comprises a broad range of manifestations of infection. Infection remains asymptomatic or subclinical in many cases or can follow an acute or chronic course. The clinical symptoms are characterized by prolonged and irregular fever often associated with rigor and chills, splenomegaly, lymphadenopathy, hepatomegaly, pancytopenia, progressive anemia, weight loss and hypergamma-globulinemia (mainly IgG from polyclonal B cell activation) with hypoalbuminemia [1]. A presumptive provisional clinical diagnosis is made on the basis of presenting clinical features and history of living in an area endemic for VL. Leishmanial infection does not lead to clinical disease in all cases; asymptomatic and subclinical forms are frequent which has been demonstrated in various epidemiological surveys [2,3].

In endemic areas; infected subjects may or may not develop classic signs and symptoms. Capacity to produce IL-2 and interferon-gamma (IFN-γ) is associated with asymptomatic or subclinical self-healing infection. In contrast, individuals whose lymphocytes do not proliferate and, thus, do not produce IFN-γ when stimulated by *Leishmania* antigen, will develop acute VL that progresses to classical disease [4]. The subclinical form of VL shows nonspecific clinical manifestation, characterized by, fever, hepatomegaly, and hypergamma-globulinemia, increased ESR, without splenomegaly and leucopenia, leading to difficulties in diagnosis [5]. The