

MISUSE OF 'AFRICAN MEDICINE'

With the long-hoped-for expanded access to antiretrovirals just around the corner in South Africa, this case report from a recent issue of *The Lancet* is worth taking note of.

In May 2003 a 6-year-old Rwandan girl presented to the Great Ormond Street Hospital for Children in London. She had a 2-day history of abdominal pain, vomiting and itchy eyes, followed by confusion and drowsiness. On admission, her Glasgow Coma Score was 7/15. On examination, bilateral proptosis was found, but no focal signs or meningism. A CT scan of the brain showed cerebral oedema and bilateral exophthalmos. Hepatic dysfunction, as seen in Reye's syndrome, was noted. Serum urate, amylase and creatine kinase were all elevated, as was her white cell count. She also had increased prothrombin time, activated partial thromboplastin time, fibrinogen and D-dimers. The authors considered viral or bacterial infections, congenital metabolic defects, and acute leukaemia, but none was supported by subsequent testing. All cultures were sterile and no viral nucleic acid was detected by polymerase chain reaction. A bone marrow aspirate showed increased granulocytopenia, but no blasts. Abdominal ultrasound showed hepatosplenomegaly and a prominent pancreas.

The child was treated with broad-spectrum antibiotics and aciclovir. She remained comatose, with no cough and gag reflexes or response to pain. However, blood ammonia levels fell to normal within 24 hours.

After the child had been in hospital for 2 days, the mother told the authors that over the past 3 months her daughter

had suffered an intermittent cough and cervical lymphadenopathy. A Rwandan friend in London had given her some 'African' medicines, thought to have come from Rwanda, and told the mother to give these to her daughter. The medicines turned out to be antiretroviral nucleoside reverse transcriptase inhibitors. The mother had unknowingly given her daughter 250 mg (340 mg/m²) of didanosine and 60 mg (3 mg/kg) of stavudine daily for 3 months. Recommended doses are 240 mg/m² and 2 mg/kg, respectively. Sixty hours after admission the child's level of consciousness started to improve and she was extubated after 4 days. By day 5 she had made a complete neurological recovery and the proptosis had resolved. The patient and her mother were found to be HIV-1 positive.

This turned out to be a case of mitochondrial toxicity, which can cause myopathy, neuropathy, hepatitis, pancreatitis and lactic acidosis. The child also had a leukaemoid reaction, hyperuricaemia, lactic acidosis and proptosis; an unusual constellation of secondary responses to mitochondrial injury. Reye's syndrome may reflect mitochondrial dysfunction, and this case adds an interesting contemporary differential for this syndrome. It is also a timely reminder of the dangers of the misuse of antiretroviral medication, which is potentially fatal.

Wittenstein B, *et al. Lancet* 2004; **363**: 860.

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SINGLE SUTURE

DON'T SLEEP AND DRIVE

Sleepiness and just a small amount of alcohol create a deadly cocktail for drivers. A test of 12 healthy men who took part in a simulated driving exercise confirmed that the combination of sleep deprivation and apparently 'safe' amounts of alcohol was sufficient to make lane drifting worse. It was also reflected in the electroencephalogram. Subjectively, however, the sleepy drivers were unaware of becoming more sleepy with alcohol.

(Horne JA, *et al. Occupational and Environmental Medicine* 2003; **60**: 689-692.)