AIDS BRIEFS

RISING INCIDENCE AND PREVALENCE OF ORPHANHOOD IN MANICALAND, ZIMBABWE, 1998 - 2003

Investigators set out to quantify and describe orphan incidence in Manicaland, eastern Zimbabwe, using an open cohort study. They looked at 13 740 and 10 308 children, aged 0 - 14 years, identified in household censuses in four socio-economic strata, in 1998 - 2000 and 2001 - 2003, and 10 184 children seen in both censuses, a 74% follow-up.

They found that all forms of orphanhood increased. The overall rate of losing a parent among non-orphans was 27.5 per 1 000 person-years. Paternal orphan incidence was higher than maternal orphan incidence and maternal orphans lost their fathers at a faster rate than paternal orphans lost their mothers. Paternal and maternal orphan incidence increased with age. Incidence of maternal orphanhood and double orphanhood among paternal orphans rose at 20% per annum and 71% per annum respectively between 1998 and 2003, but incidence of paternal orphanhood and double orphanhood among maternal orphans was unchanged. For 82% of children with a parent who died, the parent was HIV-positive at baseline. More new paternal and double orphans – but not new maternal orphans – than non-orphans had left their original household. Mortality was higher in orphans than in non-orphans, with the highest death rates observed among maternal orphans.

The authors concluded that orphan incidence and prevalence are high and increasing owing to HIV in eastern Zimbabwe. Orphan incidence patterns differ from orphan prevalence patterns and need to be understood if support programmes are to assist children during periods of high vulnerability.

Watts H, *et al. AIDS* 2005; **19:** 717-725.

CASE OF FANCONI'S SYNDROME CAUSED BY DDI

The side-effects of didanosine (ddI) are well known and include peripheral neuropathy, lactic acidosis and hepatitis. However, there has only been one previously published case of kidney failure associated with its use. Recently, a team in Paris reported a case of Fanconi's syndrome involving a 32-year-old man who had been diagnosed with HIV 16 years previously. He started antiretroviral therapy in 1997 with a regimen that included indinavir, which can cause kidney problems, and was diagnosed with hepatitis B in 2002, which was being treated with 10 mg adefovir daily. In the summer of 2004 he was hospitalised when he reported fatigue, dehydration, weight loss and blood in his urine. He also reported a dry mouth in spite of drinking 4 - 5 litres of water a day. When he was hospitalised his CD4 count was 87 cells/mm3 and his viral load was 13 000 copies/ml. At the time, his antiretroviral regimen included ddI.

On examination, he had high blood pressure and a racing pulse, with no clinical evidence of inflammation or infection. Laboratory analysis showed high lactate levels, blood phosphate of 0.54 mmol/l and a creatinine clearance of 66 mmol/l. Urine output on the second day after admission was 5.4 litres with glucose and blood present. The renal biopsy showed Fanconi's syndrome. Although high-dose adefovir can be toxic to the kidneys, the 10 mg dose was not thought to be responsible. Doctors concluded that the patient's Fanconi's syndrome was caused by ddI. Administration of the latter was discontinued and the adefovir continued. One month later the patient's renal function was returning to normal.

Izzedine H, *et al. AIDS* 2005; **19:** 844.

Bridget Farham