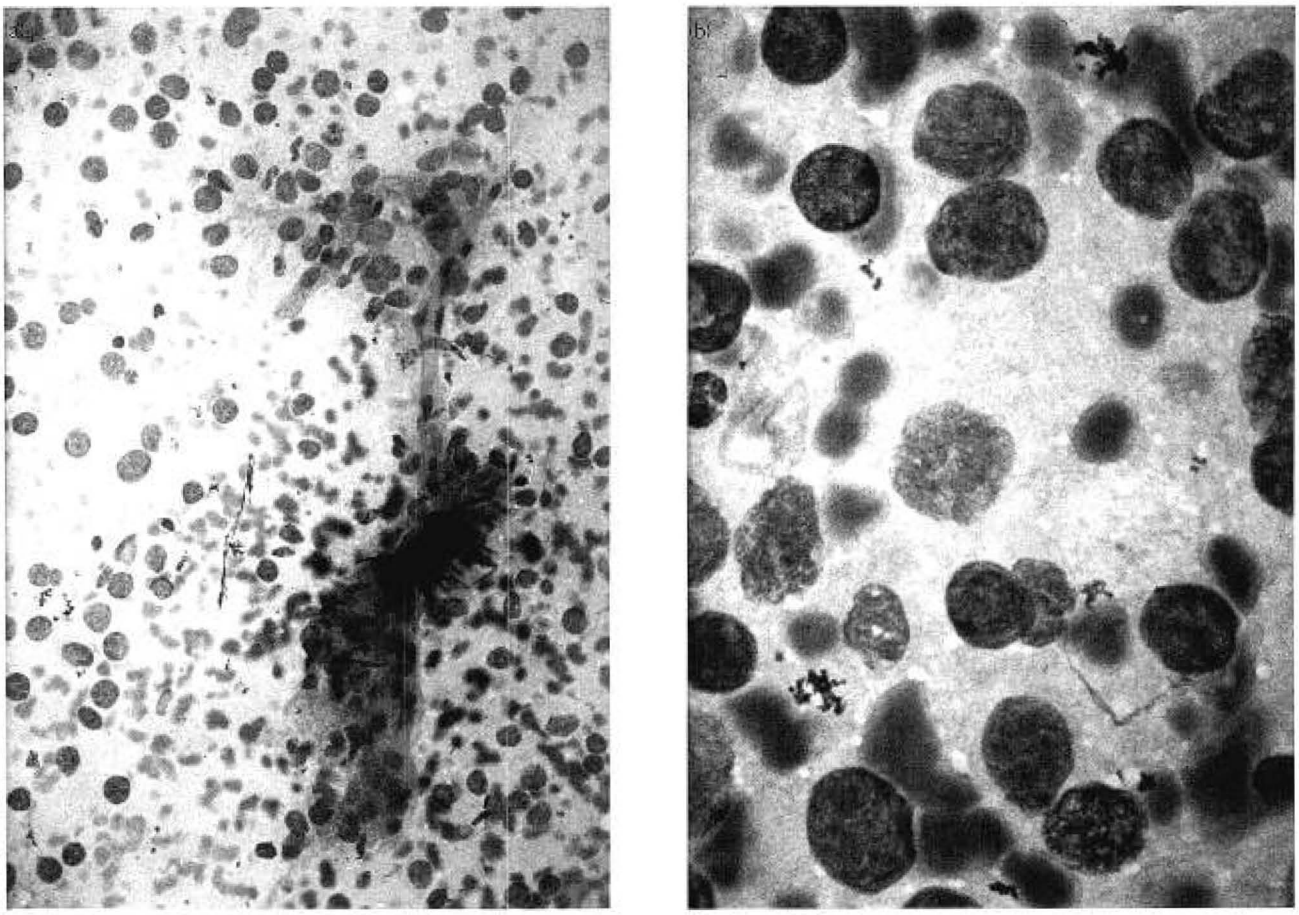

Fig. 1. Cytopathological features of adrenal carcinoma. (a) Malignant cells arranged in follicular or trabecular appearance along fibrous and vascular structures (May–Grünwald–Giemsa stain, × 100). (b) Multinucleated cells with giant nuclei, prominent nucleoli and abundant, vacuolated, clear and badly delimited cytoplasm (May–Grünwald–Giemsa stain, × 400).

HIV-related ocular microangiopathic syndrome and neuropsychological functioning

Wilkie *et al.* [1] recently reported that cognitive alterations occur in HIV-1-infected individuals before the manifestation of AIDS, and appear to be independent of the clinical status and degree of immunosuppression as measured by CD4 cell count and immunoglobulin A (IgA) level. The association between HIV-1 serostatus and cognitive impairment has not been completely explained by known potentially confounding factors, such as age, education and psychopathological status [1].

The fact that not all HIV-infected patients develop cognitive impairment or progress to a dementia syndrome suggests that factors other than HIV-1 are responsible for this condition. Dunbar *et al.* [2] showed that neuropsychological changes are not exclusively associated with progression from AIDS-related complex (ARC) to AIDS. The aetiology of cognitive symptoms may be multifactorial; pathological findings in computed cranial tomography or magnetic resonance imaging do not necessarily relate to neurocognitive decline [3], psychogenic versus somatogenic reasons are to differentiate.

In our study of 237 seropositive subjects we found that HIV-1-seropositives showed reduced cognitive functioning compared with HIV-negative controls, and detected significant correlations between psychopathological impairment and neuropsychological functioning [4]. To look for other influences on or possible explanations for a decrease in the memory functions of HIV-1-infected patients, a subgroup of 37 seropositive subjects underwent ophthalmological and neuropsychological examination. We examined these patients to evaluate a possible association between HIV-1-related ocular microangiopathic syndrome and cognitive functioning [5]. Ocular microangiopathic syndrome is common in patients with AIDS or at an

advanced stage of HIV infection [6]. Symptoms of this microvascular syndrome can include retinal cotton-wool spots, haemorrhages and Roth's spots [7].

There was a strong correlation between HIV-related ocular microangiopathic syndrome, measured by counting the number of cotton-wool spots, and a decrease in cognitive functioning, determined by neuropsychological examination including five standardized tests (Auditory Verbal Learning Test, Benton Test, Vocabulary, Stroop Colour Word Test, Trail-Making Test part B), in AIDS patients. The multiple correlation between the number of cotton-wool spots and the five neuropsychological tests was $r = 0.70$ ($P < 0.001$). Ocular microangiopathic syndrome was also strongly associated with cerebral blood flow as measured by hexamethyl-propyleneamine oxime single-photon emission computed tomography (HMPAO-SPECT) of the brain [8].

These correlations between ocular microangiopathic syndrome, HMPAO-SPECT and cognitive performance do not prove a causal relationship, but indicate that there may be a close association between cerebral blood flow and functional cerebral impairment. HIV-related microangiopathic syndrome may thus be involved in the aetiology of cognitive alterations in HIV-infected individuals.

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Decay of HIV-1 infectivity in whole blood, plasma and peripheral blood mononuclear cells

Accurate quantitation of the level of HIV-1 *in vivo* is important in understanding AIDS pathogenesis and viral transmission, as well as in monitoring the efficacy of antiviral agents given to patients. We have previously defined the infectious levels of HIV-1 in plasma and peripheral blood mononuclear cells (PBMC) from infected subjects [1]. We have also determined the infectious titers in sequential blood samples from patients with primary HIV-1 infection [2]. These studies were performed in real time (i.e., immediately after any transport and processing delays) with freshly obtained blood samples, and it is unclear whether the time de-

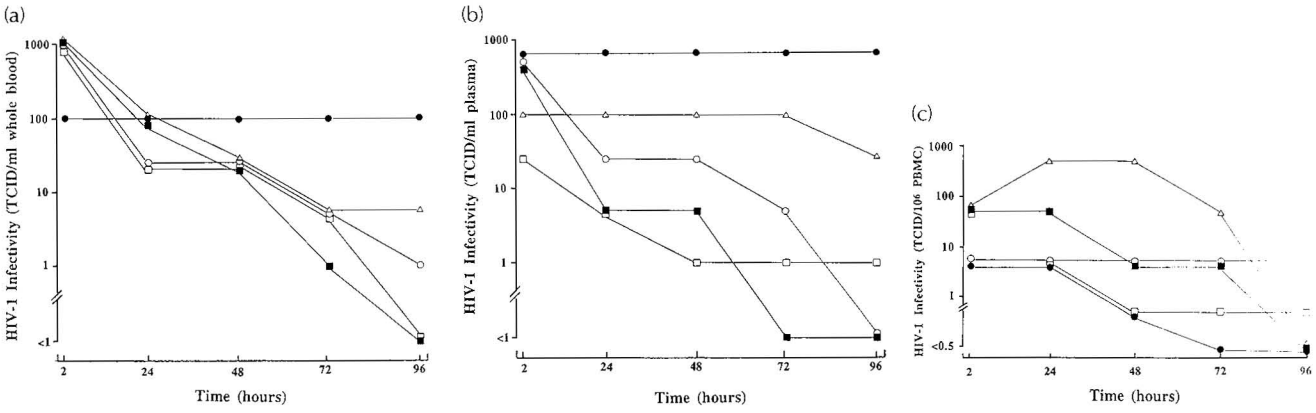

Fig. 1. Decay of HIV-1 infectivity for whole blood (a), plasma (b) and peripheral blood mononuclear cells (PBMC); (c) from five patients with AIDS. ○, patient 1; ●, patient 2, □, patient 3; ■, patient 4; △, patient 5. TCID, tissue culture infective dose.