

Psychiatric aspects of the acquired immunodeficiency syndrome

A Case Report

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Summary

Psychiatric manifestations of the acquired immune deficiency syndrome are being increasingly recognised. We report on a case who presented with psychiatric symptoms. The psychological reactions of the patient as well of the medical and nursing care-givers are briefly discussed.

Introduction

The acquired immune deficiency syndrome (AIDS) has emerged in Africa, United States and Europe as the most serious public health problem in this quarter of the twentieth century. Given the high mobility of people across international borders, recent warnings of an imminent worldwide epidemic are not misplaced.

The human immunodeficiency virus (HIV) is well known for its predilection for the T-cells of the immunological system, but its affinity for the central nervous system (CNS) is only being recently recognised. Psychiatric symptomatology as a presenting complaint is now being increasingly reported. About 30%-40% of AIDS patients present CNS dysfunction.¹ The psychiatric sequelae of AIDS may range from an adjustment disorder with anxiety or depression to a psychotic reaction and dementia.²

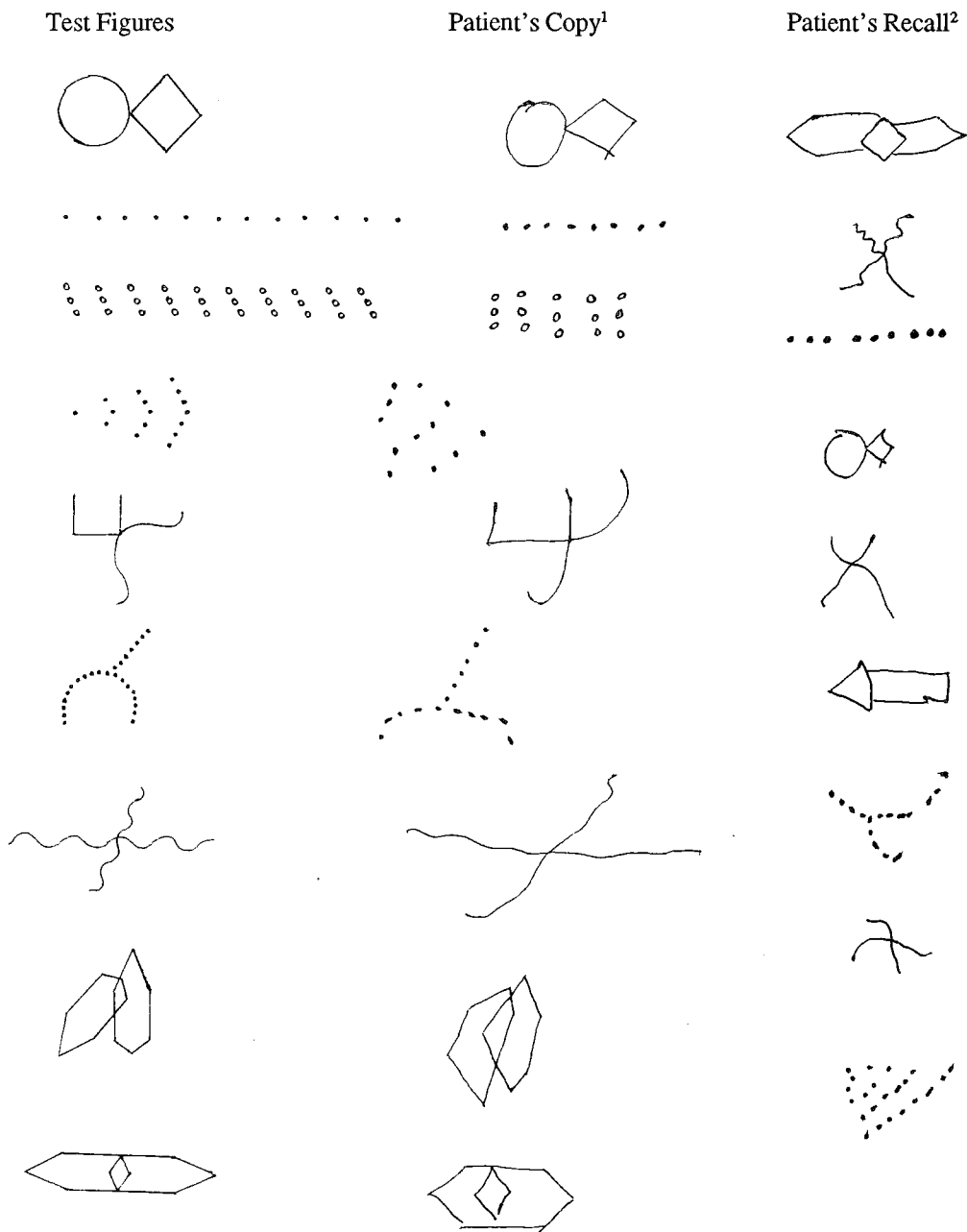
The purpose of our report is to alert doctors to the possibility of AIDS when a person in a high risk group for HIV infection (homosexuals, bisexuals, intravenous drug abusers and haemophiliacs receiving transfusions) presents with psychiatric symptomatology especially when the clinical picture is atypical.

Case Report

A 34-year old single Malaysian male, who had just returned from overseas, was noted to behave abnormally. He was talking irrelevantly, acting impulsively and threateningly towards his family

Figure 1:

Bender Visual Motor Gestalt Test – Copy and recall by patient



Note: 1. Patient's copy with test figures in front of him.
2. Patient's recall after 1 minute without the test figures.

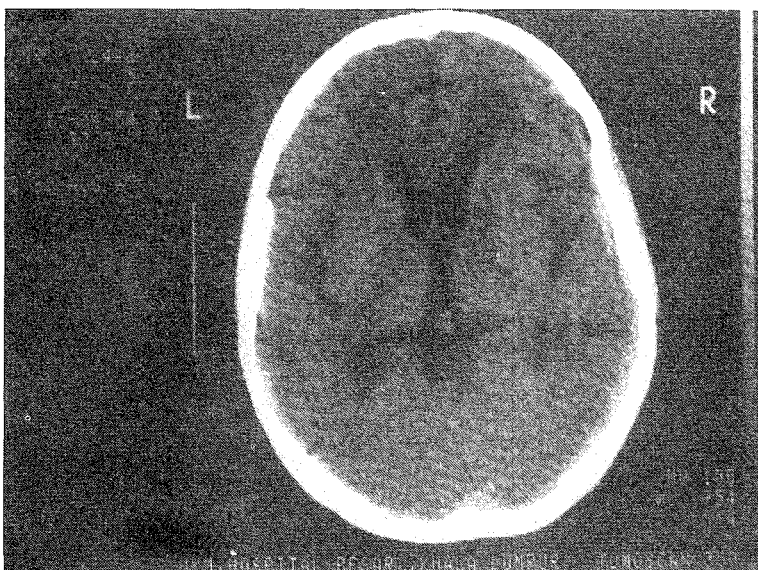


Figure 2:
Computerized tomography brain scan showing hypodense areas in frontal lobes, cortical atrophy and enlargement of ventricles.

members. He had previously been admitted to our psychiatric unit for unipolar depression in December 1984 but had been lost to followup since April 1985.

On examination he was evasive, suspicious and secretive. He had no hallucinations but had paranoid delusions of being poisoned. His memory was fluctuating between normal and patchy recollections, and concentration was poor; but he was unconcerned about his difficulties. He had considerable weight loss and scattered loss of hair. Hairy leukoplakia was present on the lateral sides of the tongue and the cheeks as well as oral candidiasis and seborrhoeic dermatitis on the eyebrows. Some ichthyosis was noted on the limbs.

Performance on the Bender visual motor Gestalt test was impaired (Fig. 1).

Further investigations revealed a lifestyle of high risk to HIV infection. An enzyme-linked immunosorbent assay (ELISA) for HIV proved to be highly positive and was confirmed by the Western Blot test in both blood and cerebrospinal fluid (CSF). Subsequent enquiries revealed two previous hospital admissions overseas for AIDS related dementia complex and AIDS with pneumocystis pneumonia in 1986.

A computerised tomography brain scan showed low attenuation of frontal lobe white matter bilaterally with presence of cerebral atrophy and secondary enlargement of the CSF spaces (Fig. 2). The T-helper (T_H)/T-Suppressor (T_S) cell ratio was reversed (1:2) and a cell-mediated immunity skin test showed the patient to be anergic, both indicating immune deficiency. Stool examination was positive for Cryptosporidia.

With a diagnosis of AIDS dementia the patient was started on Azidothymidine (AZT) or Zidovudine with an initial dose of 500 milligrammes every eight hours. He has been on AZT for the past two months with the weight increasing steadily and his memory seems to be slightly improved. Performance on the Bender Gestalt test remains impaired.

Discussion

The most common form of CNS dysfunction in AIDS is a nonfocal encephalopathy which may present with features suggestive of depression.¹ The depressive symptoms may antedate the

appearance of other signs of a dementing illness, and we believe our patient may have had an AIDS related complex when he first presented with depression in December 1984. Other common symptoms of this encephalopathy include forgetfulness, poor concentration, apathy, loss of interest and loss of libido, all of which were present in our patient in varying degrees.

Perry and Markowitz³ have described three common psychiatric sequelae of AIDS – the three Ds of depression, delirium and denial. The depression is not merely a grief reaction but a pathological process with irrational guilt, helplessness, hopelessness, worthlessness and high suicidal ideation. Denial may become so extreme that it may interfere with medical care and public health preventive steps. Our patient exhibited the defence mechanism of denial at the beginning.

Another interesting aspect to this case is the psychological reaction of the medical staff when the diagnosis became known. Anxiety was the most common reaction with accompanying symptoms of mild insomnia. Fear of having contacted the disease induced crying spells in some and a reluctance to handle one's children at home. Anger that he had exposed others to the illness by not divulging his illness at the outset and rejection were also noted. However, these reactions were transient and disappeared when information concerning AIDS – its mode of spread, groups at risk et cetera, were given together with reassurances. A number of staff underwent the ELISA test themselves (all negative). This underscores the vital importance of providing information quickly and clearly.

Clinicians would need to have a high index of suspicion in dealing with persons in high risk groups who have travelled and who present with atypical psychiatric symptoms.

References

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