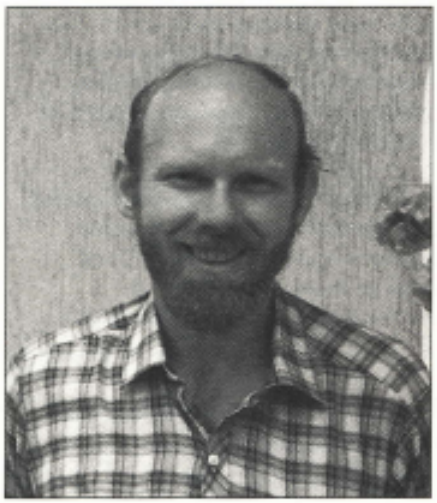
A Patient with AIDS — Chris de Muelenaere



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Curriculum vitae

Chris de Muelenaere was born in Belgium and came to South Africa with his parents as a child. He matriculated from the Afrikaans Boys' High in Pretoria and qualified MBChB (UP) in 1968, MPraxMed (UP) in 1975, and LFAP (SA) in 1976. The de Muelenaere family has a strong medical tradition. Chris has been practising in Pretoria as a private practitioner since 1970, and has a special interest in the ethical-moral aspects of the practice of medicine, and in matters affecting the sanctity of life. His wife, Diedre, studied Human Sciences and is a high school teacher.

Summary

A history of an Aids patient is given, highlighting the circumstances why the correct diagnosis was missed and reporting on how he experienced the disease and how he was cared for by his GP and his family.

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KEYWORDS:
Aids; Case Report; Domiciliary Case; Diagnosis.

MISSED DIAGNOSIS OF AIDS BECAUSE OF LACK OF AWARENESS

AIDS is a newly recognised disease, with protean manifestations only after a long incubation of the Human Immunodeficiency Virus (HIV). When initially investigated, the indications of a possible Acquired Immune Deficiency Syndrome were found to be chronic diarrhoea with cachexia ("slim disease") and/or repeated attacks of lower respiratory tract infections with *Pneumocystis Carinii* and/or rare tumours like Kaposi sarcoma.

Karstaedt[1] at Baragwanath hospital found that the major clinical presentation of AIDS in HIV infected adult blacks were mainly tuberculosis, acute pneumonia, herpes zoster and "slim disease" in that order of importance. Only 1 of 181 cases had documented *Pneumocystis Carinii* pneumonia.

Patient report

Background History

The patient, a married white male, had been a patient of the author for many years. He had been a security policeman in the counterinsurgency unit, a very active and self-reliant man who once walked on his own from the Caprivi to Moçambique through the bush. He lived in the Caprivi for two years and was dark suntanned.

Previous operations include a spinal fusion in the lumbar area in 1981, and a quadruple coronary artery bypass operation in 1983. For both operations he received blood transfusions, which in those days were not tested for the presence of HIV. He recovered fully from each of those operations.

He had been a heavy smoker (40 - 50 cigarettes/day) but stopped when he needed the bypass operation. He had at times indulged in heavy drinking but was not an alcoholic. There was never any indication of homosexuality and he never, in 17 years, presented to the author with a venereal disease.

Presentation

The patient had repeated attacks of lower respiratory tract infections (bronchitis or pneumonia) in October 1986, December 1987, January 1988 and February 1988. Various pathogens were cultured (see Table 1). Because of the repeated attacks, and one incidence of plate atelectasis of the lung he was referred to a thoracic surgeon who found no trace of malignancy.

During the February 1988 pneumonia episode he developed a severe Herpes Zoster attack of T5 dermatome. He also developed porphyria cutanea tarda on the hands in that period.

Another lower respiratory tract

. . . A Patient with AIDS

Table 1. Sputum cultures

January 1988	± B Haemolytic strep no *Mycobacterium tuberculosis*
February 1988	1 + *Klebsiella pneumoniae* 1 + *Haemophilus hemolyticus*
January 1989	1 + B haemolytic strep 2 + *Haemophilus hemolyticus* no *Mycobacterium tuberculosis*
February 1989	± *Escherichia coli*

Note: Haemophylus hemolyticus "rarely, if ever, causes clinical disease"[2]

infection occurred in April 1988 and a pharyngitis in September 1988. On 24.10.88 he was hit on the left parietal area when playing softball (the ball is *not* soft!) suffering a concussion, headache, scotomas, dizziness and nausea. Then it was noted that he had lost 5 kg in body mass over the previous eight months. He mentioned loose stools. He was treated conservatively under observation and was much better after two days.

On 16.1.1989 he complained of dizziness and itch and deafness of the right ear. On examination he was mildly febrile 37,1°C, had BP 100/70 and had prostatitis and peripheral neuritis. He was treated with neurotropic vitamins and co-trimoxazole. Two weeks later he collapsed at home and was unconscious for 30 minutes, after which he felt limp, unsteady and felt as if in a trance. On examination he had basal pneumonia, prostatitis and a BP of 140/90. Treatment with an NSAID and tetracycline/bromhexidine was given, with marked improvement three days later.

Another two weeks later, on 17.2.1989, he presented with chronic headache, loss of balance, loss of ability to judge distances, disturbed sleep, nausea and weakness. His right ear was fine except for a "peculiar" feeling in the ear. On examination he again had basal crepitations, but had an upper normal temperature of 36,9°C. He was hospitalised and

AIDS is still incurable

treated with cephalosporins and after eight days was discharged on digoxin 0,0625 mg daily because of mild cardiac failure.

Two days after discharge, a home visit was requested because he had fallen on his back, with severe pain. He was treated at home with bed rest, NSAID and analgesics. He had a compression fracture of L1.

Because of his steady deterioration, he was referred to specialists. One ENT surgeon for his vertigo and then two physicians and two neurologists. Brain scan showed mild frontal lobe atrophy and early blunting of the ventricles. None of the specialists made a definite diagnosis in spite of extensive blood tests.

In February 1989 it was also noticed that his dark suntan had become darker in spite of being confined indoors most of the time. A provisional diagnosis of Addison's disease was made. The serum cortisol was normal (405 n mol/*l*), but the serum ACTH was low (7,5 ng/*l* - normal 10-110 ng/*l*) and an empiric test dose of prednisone 5 mg daily produced marked improvement of his complexion and a mild improvement of his dementia.

Do not rely on *Pneumocystis Carinii* isolation as an indication of AIDS

He had high levels of IgA, IgG and IgE.

On 7.3.89 he was readmitted because of further deterioration in his general condition. He again developed lower respiratory tract infection and his dementia (memory loss, confusion, disorientation and incontinence) and muscular incoordination worsened. A blood test for the Human Immunodeficiency Virus (HIV) was ordered as a last resort. It was positive for antibodies and confirmed positive for antigen. The case was reported to the National Institute for Virology (NIV) as a case of cerebral AIDS. The NIV refused to accept the diagnosis without proof of antigen in the cerebrospinal fluid. This was confirmed. Treatment with zidovudine was started.

. . . A Patient with AIDS

On 24.5.1989 the patient developed severe pneumonia and entered a state of stupor. The author (doctor in charge) ordered no resuscitation but intensive treatment in the medical ward was continued. The patient

Quality living was the aim

recovered from his pneumonia and made a slow recovery mentally and physically. He was discharged from hospital on 6.6.89, able to walk with a cane.

Confidentiality

The ward nursing staff, lab staff and physiotherapists were immediately warned of the danger of contagion and to take special precautions when caring for the patient.

The patient's wife was immediately informed. She was tested for the presence of HIV antibodies. The test was negative, in spite of having consorted with him during the quiescent stage. She looked after him at home until his death on 4.12.1991. At the time of writing she was still free of HIV.

The patient, after recovering his mental faculties, asked for the diagnosis. The author informed him that the cause was a virus in the brain; the source of which was unknown, but possibly due to blood transfusions. He never enquired as to the precise nature of the virus. The information was not volunteered as it was considered by both family and doctor that the exact diagnosis would have caused the patient too much anguish. Had he asked, he would have been told.

The patient's siblings and children were notified by the wife.

All the specialists consulted were informed of the final diagnosis, and all expressed surprise.

Lessons to be learned

1. In hindsight the diagnosis should have been suspected and confirmed much earlier. The patient's lifestyle did not fit the homosexual stereotype, but he had spent a lot of time in Central Africa and had twice had blood transfusions. The warning bell should have sounded at the first repeat of the lower respiratory tract infection, in this case especially January or February 1988.

2. The HIV can attack any part of the body and should be considered another "great mimic". Tests for the great mimic syphilis were negative.

3. The HIV is not very contagious, and as long as standard elementary

Impatient with his own weakness

precautions are followed, it is safe to treat and nurse the patients. The essential precaution is to avoid contact with body fluids or excreta.

4. Do not rely on *Pneumocystis Carinii* isolation as an indicator of AIDS. Note that the pathogens cultured in the sputum on various occasions (Table 1) are known to be fairly unusual except in immunocompromised patients, ie they are opportunistic pathogens.

HOME MANAGEMENT

A white male of 53 was diagnosed as suffering from advanced AIDS - cerebral dementia, vertigo and ataxia, repeated lower respiratory tract infections, and attacks of diarrhoea. Both HIV antigen and antibodies were present in the blood and in the cerebrospinal fluid. The patient was also suffering from Addison's disease - whether due to the HIV or not, is not known. He also had porphyria cutanea tarda.

AIDS is still incurable. The primary aim of management was to help the patient along as much as possible, and to delay the inevitable decline and death as long as possible. Quality living was the aim and achieved by palliative and symptomatic treatment at home. In consultation with the patient and the family, it was decided not to resort to active resuscitation or intensive care ward admission, but only to hospitalise in an ordinary ward for amelioration of any complications of the disease. The mainstay of medical treatment was:

1. Zidovudine 1 000 mg per day divided in 5 doses;
2. Prednisone 5 mg alternate days to keep his Addison's disease in check;
3. Autrin caps daily to prevent deterioration of his chronic anaemia.

Clinical Course of the Disease

The patient was discharged from hospital on 6.6.89, weighing 70 kg with an indwelling catheter but able

. . . A Patient with AIDS

to walk and with lucid mental faculties. He had normochromic normocytic anaemia, low serum calcium of 2,04 m mol/*l* and an *Escherichia coli* cystitis.

On discharge, apart from the routine medication described above, he was given calcium supplements, Cinnarizine 75 mg bd for the vertigo was only needed for a few days. Chlorpromazine HCl 110 mg tds was given for the porphyria and Etilefrine HCl for low blood pressure.

The patient regained bladder control on 1.7.89 and the catheter could be removed.

By September 1989 he could walk more than one km; and by December 1989 his weight had improved to 86 kg (normal for 1,85 m).

He sustained repeated attacks of urinary tract infections, lower respiratory tract infections (Table 2) and diarrhoea from October 1989;

Table 2: Microbiological culture of sputum

7.5.1990	B haemolytic streptococcus non-A
21.9.1990	*Proteus mirabilis* *Pseudomonas aeruginosa*
26.10.1990	*Branhamella catarrhalis*
26.11.1992	*Pseudomonas aeruginosa*

At no stage was tuberculosis or *Pneumocystis Carinii* found.

Table 3: Haemotological profile

Date	Haemoglobin g/d*l*	Haematocrit	ESR mm/hr
1.7.1989	11,7	0,35	54
26.8.1989	11,6	0,34	53
2.4.1990	13,6	0,393	50
25.10.1990	10,6	0,305	61
23.4.1991	9,7	0,28	
5.8.1991	9,5	0,281	65

Please remember that the steady decline in haemoglobin and haematocrit took place in spite of the constant Autrin capsule daily. Autrin contains Vit B12, folic acid, ferrous fumarate and ascorbic acid.

and decline in motor activity and general health started at the end of 1989. The laboratory profile of his deterioration is presented in Table 3 (haemotology), Table 4 (liver function tests) and Table 5 (immunoglobulins). Laboratory tests were requested as required. He suffered a relapse of Herpes zoster in T4. He developed chronic headaches for which he required Syndol all the time (at first 100 tablets per month, later increasing to 200 per month).

By June 1990 his neuromuscular condition had weakened so that he walked with difficulty and needed a wheelchair by August 1990. He became incontinent in April 1991. In July 1991 he developed chronic sores of the hand dorsi (? porphyria) and buttocks and in September 1991 severe ulcers in the mouth.

He had several attacks of diarrhoea and after having stabilised his weight for six months at 86 kg there was a rapid and inexorable decline with cachexia losing 10 kg in 2 months.

When he died in December 1991 of pneumonia he weighed only 58 kg. He was 56 years old.

Psychological stress

The patient had been a very active, strong and self-reliant man. His weakness and emaciation weighed heavily on him. At discharge and with the initial improvement he was ebullient. When the headaches started he became despondent and remained depressed until the end. At times he was very irritable and impatient with

Frequently invited to talk about his fears and hopes – but he never wanted to

his weakness and this carried over in the relationship with his wife and other caregivers. As he weakened and deteriorated he withdrew more and more. He realised fairly early on that

. . . A Patient with AIDS

Table 4: Liver function tests

Date	Total protein g/l	Albumin g/l	S-AST u/l	GGT u/l
normal	64 - 86	32 - 51	0 - 25	8 - 36
26.8.1989	102	29	32	27
2.4.1990	92	37	33	16
23.4.1991	82	26	39	56
5.8.1991	79	25	43	90

he was terminal, even though he lasted another two years. He was repeatedly assured by both his doctor (the author) and his wife that he would not be neglected or abandoned. He was invited to ask questions all of which were answered frankly and sympathetically. When the patient asked what the problem was (while still in hospital) he was told that he had a virus in the brain. He never enquired as to the precise nature or type of virus. Had he done so, he would have been told.

The author saw the patient at least once every two weeks, often more frequently and took care of him during his intermittent hospitalisations for respiratory tract infections. Frequently the patient was invited to express his feelings and to talk openly about his fears and hopes. He rarely used these opportunities and never wanted to discuss his impending death.

A pattern of social isolation developed

In the last month he sometimes asked for confirmation that the end was near.

After discharge, a younger brother who lived across the street from the patient, came every day to help wash and dress him. The brother was aware of the diagnosis. He helped until the patient regained his strength; and then kept on visiting. Under pressure of his own wife he reduced his visits and after a few months did not ever visit him again. This pattern of social isolation extended to the patient's other siblings, children and stepchildren. In the last year the only people visiting the patient were the social worker and the doctor. The social isolation deepened the depression.

The strain under which the wife lived, was occasionally unbearable

The patient's wife

The patient's wife had a tremendous burden to bear. She looked after her husband until the end: washing and cleaning up after him, giving him a soft diet when he had mouth ulcers, pushing him around in his wheelchair, etc. On two occasions the patient was admitted to hospital for a

Table 5: Immunoglobulins

Date	IgA g/l	IgM g/l	IgG g/l	IgE IU/l
normal	*1 - 3.25*	*0,5 - 3.2*	*8 - 17*	*0 - 25*
26.8.1989	9,14	5,14	31,9	503
2.4.1990	12,4	2,79	31,5	746
25.10.1990	9,28	3,23	31,2	235
23.4.1991	11,6	3,72	36,7	144

He developed hyperimmunoglobulinaemia. HIV suppresses immunity (the cellular response), but increases the total immunoglobulins. (Immunological tests for HIV infestation would not become positive if there were no antibodies - they are however ineffective).

. . . A Patient with AIDS

week to give her a rest. She was helped in the last few months by a hired nurse-aid half a day. Throughout it all she also was encouraged to speak openly and express her feelings, which she did more readily than her husband. Some family members criticised her for being hard on her husband, but that was grossly unfair. The others stayed away while she remained to bear the burden and the strain under which she lived was occasionally unbearable - she did sometimes have to resist his demands to maintain her own sanity.

> Family and doctor repeatedly assured him he would not be neglected or abandoned

In spite of her cohabitation before the terminal illness, and in spite of having to live with and care for him for more than two years afterwards, and being exposed to his various body fluids, she remained uninfected. She took only the elementary precautions, once the diagnosis was established, to avoid exposure to his body fluids (sputum, urine, stools, weeping open sores, etc) by using disposable gloves and thoroughly cleaning the linen.

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