Disseminated Infection due to *Mycobacterium celatum* In patient with AIDS

SIR---AIDS predisposes to mycobacterial disease but infection with mycobacteria other than *Mycobacterium tuberculosis* (MOTT) occurs late in the course of HIV infection, usually after the AIDS-defining disease has been diagnosed. *M avium* complex (MAC accounts for most non-tuberculous disseminated infections and the prevalence of other MOTT in patients with AIDS is much lower, the most usual ones being *M kansasii*, *M xenopi*, *M gordonae*, *M malmoense*, and *M haemophilum*. Lately, two new species have emerged as opportunistic pathogens in AIDS patients: *M* genavense, first isolated in 1990 from a Swiss patient, has now been reported from other European countries, the United States, and Australia; and *M celatum*, described in 1993,[1] seems biochemically indistinguishable from *M avium*, but shows a mycolic acid pattern closely related to that of *M xenopi*. We describe a case of disseminated infection due to M celatum in a patient with AIDS.

A 28-year-old woman, partner of an AIDS patient, was found to be HIV-positive in May 1992. She was repeatedly admitted to hospital with *Pneumocystis carinii* pneumonia and cytomegalovirus retinitis. She presented in November, 1993, with fever, malaise, and severe anaemia (haemoglobin 5.9 g/L); ultrasound examination revealed splenomegaly. Cultures of blood for mycobacteria were positive and a regimen of isoniazid, rifampicin, and ethambutol had a good effect on her symptoms. The patient is still alive and follow-up blood cultures, except one, have been negative.

The strain was isolated from blood on Bactec 13A (Becton-Dickinson, Towson, USA), inoculated following lysis centrifugation. On conventional biochemical and cultural testing (table) of the isolate, the data being fed into a computer program for the identification of mycobacteria,[2] *M avium-intrace11u1are* seemed likely. The Bactec NAP test (Becton-Dickinson) showed no inhibition by NAP.

Hybridisation tests with DNA probes specific for *M avium* or *M intracellulare* (AccuProbe; Gen-Probe, San Diego) were negative. Mycolic acids analysis by high-performance liquid chromatography[3] suggested *M xenopi* but closer comparison of the eluate profiles did reveal minor differences.

Table: **Biochemical and culture tests on isolate**

Biochemical		Cultural	
Niacin	-	Growth at 25/45°C	+/+
Nitrate reductase	-	MacConkey	-
Thermostabie catalase	+	Tolerance to:	
β-glucosidase	-	*p*-nitrobenzoate	+
Tween 80 hydrolysis (10 days)	-	Na Cl (5%)	-
Tellurite reduction	+	Thiophene-2-carboxylic hydrazide	+
Arylsulphatase (3 days)	+	Thiacetazone	+
Urease	-	Hydroxylamine	-
Catalase	-	Oleate	-
Acid phosphatase	+	*p*-aminosalicylate	+
Photochromogenicity	-	Toluidine-blue	+
Scotochromogenicity	-	Growth rate	Slow
		Colonial morphology	Smooth

The susceptibility pattern of our *M celatum* strain was determined by the macrodilution method developed for MAC.' The close similarity of growth kinetics between *M* celatum and MAC easily allowed us to do the susceptibility tests validated for MAC. The minimal inhibitory concentrations (μg/mL) able to inhibit the growth of 99% of mycobacteria were: amikacin 0-5, azithromycin 4, ciprofloxacin 0-5, clarithromycin 0-25, clofazimine 0-12, ethambutol 1, kanamycin 4, ofloxacin 1, rifabutin 0-25, rifampicin 256, sparfloxacin 0-25, and streptomycin 0-5.

16S rRNA gene fragment: sequencing,[5] kindly performed by Dr E Böttger (Hannover), definitively allowed the attribution of our strain to the recently described new species *M celatum*.

Unlike *M genavense*, *M haemophilum*, and *M malmoense*, *M celatum* is not a "fastidious" mycobacterium and it grows well on conventional media; the delay in its recognition is probably due to its similarity with MAC in conventional tests for the identification of mycobacteria. DNA probes and chromatography cast doubts on the attribution of this strain to MAC, and genetic sequencing allowed the recognition of the new species *M celatum*.

No other isolation of *M* celatum from the blood of an AIDS patient has been reported. The repeated isolation from blood stresses the clinical significance of this novel organism to the list of mycobacteria which cause disseminated infection in AIDS.

Our susceptibility data are not so alarming as suggested in the only other report.' Our *M* celatum strain had a susceptibility pattern halfway between that of MAC and *M* tuberculosis. The full susceptibility to quinolones and the strong resistance to rifampicin seem the salient features. The clinical improvement observed after chemotherapy supports our susceptibility data.

M celatum should be suspected when a mycobacterium behaving in conventional tests as MAC fails hybridisation with probes specific for this complex. Genetic sequencing and mycolic acid analysis will confirm the identification.

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