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Septal perforation secondary to *Mycobacterium kansasii* infection

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Abstract

We report the first recorded case of a septal perforation caused by *Mycobacterium kansasii*. This atypical mycobacterium is finding increasing prevalence with the increasing incidence and longevity of human immunodeficiency (HIV) infections. Cases of chest infection, sinusitis, septic arthritis, osteomyelitis, pericarditis, brain abscess, cutaneous and oral lesions have all now been reported. This discovery represents a rare but important differential in the aetiology of septal perforation.

Key words: Nasal Septum; Fistula; Mycobacterium Kansasii

Case report

A 63-year-old English lady presented to the chest physicians with increasing shortness of breath on exertion, yellow sputum and occasional spots of blood from the nose. She had no past medical history of chest disease, nasal trauma, septal surgery or inhaled irritants. She was an ex-smoker (10 pack years). On respiratory examination she was noted to have bilateral widespread crepitations and a peak expiratory flow rate (PFER) of 250 L/min. ENT examination revealed a smooth anterior septal perforation approximately 1 cm diameter.

A chest radiograph showed shadowing in both lung fields, which on computed tomography (CT) was reported as fibrosis on the left and a cavitating lesion on the right with collapse/consolidation of the right middle lobe. This was suggestive of granulomatous disease. Full blood count showed lymphopenia, however, she had normal electrolytes and liver function tests. Anti-nuclear antibodies were raised and angiotensin-converting enzyme was normal. Over the following two months she underwent a bronchoscopy and biopsy of the septal perforation.

At operation the septal perforation was photographed (Figure 1) and biopsies taken from three separate sites around the margin. These were sent for histology and microbiology. Histologically there were fragments of partly ulcerated nasal mucosa, lined by a stratified squamous epithelium, with multiple granulomas present (Figure 2). The results of the biopsy culture matched those of the bronchial washings i.e. *Mycobacterium kansasii*.

She was started on a treatment of Rifinah® 300 two tablets o.d., pyrazinamide 2 g o.d. and pyridoxine 10 mg o.d. This was then converted once sensitivities were known to rifampicin 600 mg o.d. and ethambutol 800 mg o.d. for a total of nine months.

Initial liver function tests after commencing anti-mycobacterial therapy were abnormal. Fortnightly blood tests returned to normal after rationalizing therapy to rifampicin and ethambutol. After completion of therapy repeat sputum cultures demonstrated eradication of the disease. She continues to have an asymptomatic septal perforation for which she has declined treatment. A subsequent human immunodeficiency virus (HIV) test performed was negative.

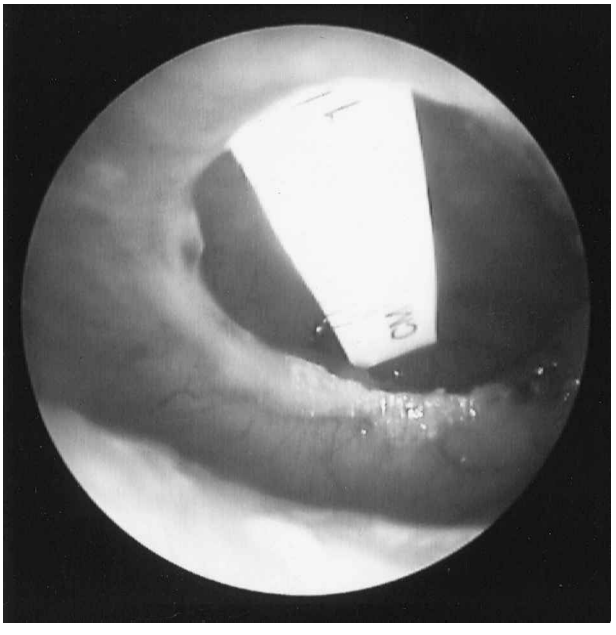

Fig. 1
Photograph of septal perforation at operation.

Discussion

Septal perforation is a not uncommon condition usually caused by trauma (Table I).[1] To determine the cause of the perforation a full history and physical examination are required. As well as routine blood tests fluorescent treponemal antibody absorption test (FTA-ABS),

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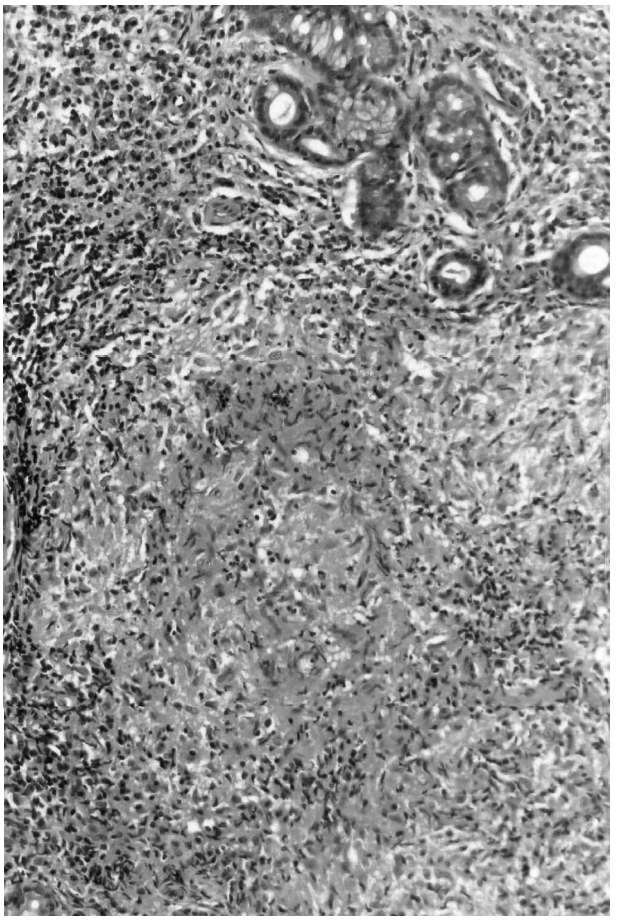

FIG. 2
Photograph of septal biopsy histology (H & E; ×100)

cANCA and ACE are performed to test for syphilis, Wegener's granulomatosis and sarcoid respectively. Unless supportive tests of cANCA or ACE are abnormal or malignancy is suspected biopsies of the perforation for microbiology and histology are not required.[2] Chest radiographs and CT scans of the paranasal sinuses should also be included.

Having made the diagnosis, the cause should be treated (e.g. anti-mycobacterial therapy in the case of *Mycobacterium kansasii*). Local symptoms can also be treated medically through topical hydration or surgically by the insertion of a septal button or septal cartilage reconstruction. Reconstruction results are generally poor if the perforation is greater than 2 cm in diameter.[1]

TABLE I
THE CAUSES OF SEPTAL PERFORATION

Aetiology	Example
Trauma	Previous surgery e.g. septoplasty
	Cauterization
	Nose picking
Inhaled irritants	Cocaine
	Snuff
Neoplasm	
Inflammatory diseases	Sarcoidosis
	Wegner's granulomatosis
	Systemic lupus erythenatosis (SLE)
	Rheumatoid arthritis
Infection	Bacterial e.g. *Staphylococus aureus*
	Fungal e.g. *Candida* sp.
	Syphilis
	Tuberculosis

[adapted from Collman *et al* [1]]

TABLE II
THE INCIDENCE OF MYCOBACTERIUM KANSASII INFECTION

Incidence	Population
2.4 per 100 000	general population
115 per 100 000	HIV patients
647 per 100 000	AIDS patients

[data from Bloch *et al* [4]]

Mycobacterium kansasii is a Gram positive aerobic acid/alcohol-fast bacillus causing chronic inflammation and granuloma formation. It is the second most common atypical mycobacterium after *Mycobacterium avium* complex (MAC).[3] *Mycobacterium kansasii* is particularly prevalent amongst (HIV) patients. With the increasing incidence of HIV infection and increased longevity of the HIV population, due to improving retroviral therapy, there follows an increase in *Mycobacterium kansasii* infection (Table II).

The clinical and radiographic picture of *Mycobacterium kansasii* infection is much more similar to *Mycobacterium tuberculosis* (TB) than other atypical mycobacterial infections.[3] It is second only to TB in its virulence[5] with a positive culture being 10 times more likely to represent disease than simply colonization. It is transmitted via tap water and milk, however there is no evidence of human to human transmission.[3]

In a recent study of the patients with *Mycobacterium kansasii* 60–74 per cent had a pulmonary disease only, 16–22 per cent had both pulmonary and extra pulmonary disease, and four to 18 per cent had disseminated disease with no evidence of pulmonary involvement.[3] The patient is usually male presenting with symptoms of TB e.g. fever, night sweats, weight loss, fatigue, productive cough, dyspnoea, chest pain and haemoptysis, or asymptomatically with an abnormality on the chest radiograph.[6] Cavitating lesions are the most common chest radiograph abnormality, interestingly seen more frequently in the HIV-negative patients.[4]

TABLE III
AMERICAN THORACIC SOCIETY GUIDELINES FOR DIAGNOSIS OF AN ATYPICAL MYCOBACTERIAL RESPIRATORY INFECTION

These criteria apply to symptomatic patients with nodular or cavity infiltrate on chest radiograph or a high resolution chest computed tomography scan that shows multifocal bronchiectasis or multiple small nodules

A. At least 3 sputum/bronchial wash results available from the previous 12 months;
1. 3 positive cultures with negative acid fast bacillus (AFB) smear results, or
2. 2 positive cultures and 1 positive AFB smear
B. If only one bronchial wash is available:
Positive culture with heavy positive (2+, 3+, 4+) AFB smear or heavily positive (2+, 3+, 4+) growth on media
C. If sputum/bronchial wash evaluations are nondiagnostic or another disease can not be excluded:
1. Transbronchial or open lung biopsy yielding an atypical microbacterium
2. Biopsy showing compatible histopathologic features (granulomatous inflammation or AFB) and one or more sputums or bronchial washing positive for an atypical mycobacterium

Reproduced from Griffith DE.[5] Mycobacteria as pathogens of respiratory infection. *Infect Dis Clin N Am* 1998;**12**:593–611

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- **This is a case report of a septal perforation caused by *Mycobacterium kansasii***
- **This is the first such report of a perforation caused by this organism**

Mycobacterium kansasii has also been reported causing septic arthritis and osteomyelitis, pericarditis, brain abscess, paranasal sinus infection, aphthous-like ulceration of the oral cavity or cutaneous and nasal lesion.[7] Diagnosis is by culture of the sputum, bone marrow, skin etc. although a DNA probe is being developed.[3] Table III shows the American Thoracic Society (ATS) guidelines for diagnosis of atypical mycobacterial respiratory infection.

The current ATS recommended treatment for *Mycobacterium kansasii* susceptible to rifampicin is rifampicin, isoniazid, pyridoxine and ethambutol for 18 months. Resistant isolates are treated with isoniazid, pyridoxine ethambutol and sulfamethoxazole for 12 months with amikacin or streptomycin during the first six months. Documentation of at least 12 months of negative sputum cultures is required to confirm disease-free status.[15,16]

This discovery is the first reported case of septal perforation caused by *Mycobacterium kansasii*. This represents a rare but important differential in the aetiology of septal perforation.

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