Streptococcus group A pneumonia in an intravenous drug misuser (IVDM)

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ABSTRACT: Streptococcus pyogenes appears to have become an uncommon cause of pneumonia. In view of the recent increase in S. pyogenes infections this situation is likely to change. An intravenous drug user presented with acute onset of fever and chills. At presentation pleuritic chest pain was a prominent symptom, and later he developed pulmonary abscesses and an empyema. The patient had a good response to benzyl penicillin, and his pulmonary lesions resolved completely. Although his clinical picture was characteristic of S. pyogenes pneumonia, it could easily be mistaken for Staphylococcus aureus septicaemia.


Staphylococci are the commonest cause of sepsis in intravenous drug misusers (IVDMs) [1] and this can obscure the frequency of group A streptococcal (Streptococcus pyogenes) sepsis. S. pyogenes is found to be the commonest isolate after Staphylococcus aureus [2]. Streptococcal pneumonia, with its marked propensity to abscess formation, may pass unrecognized owing to its current infrequent occurrence [3]. However, as there appears to be a resurgence of serious invasive streptococcal sepsis [4], streptococcal pneumonia may also return.

Case report

A twenty six year old male intravenous drug addict who had been well, apart from documented hepatitis B 2 years before, was admitted after a three to four week binge of parenteral drug abuse. This followed release from prison after a 14 month term. He reported that he had been seronegative for human immunodeficiency virus 1 (HIV1), but declined testing while an in-patient.

Two days prior to admission the patient became nau-seated and developed right pleuritic chest pain and severe chills. On admission he was in considerable pain, pyrexial, tachycardic and tachypnoeic. There were raised tender red lesions on his right forearm and left wrist and forearm, which he said were not near to injection sites. There was also an area of superficial ulceration at an injection site, on the patients left knee. His throat was not inflamed. There was tenderness over the right chest wall. No cardiac murmurs were heard. Chest X-ray showed shadowing in the right middle lobe and a probable abscess in the left lower lobe. The patient was empirically treated with intravenous erythromycin one gram six hourly and flucloxacillin one gram every four hours, in the expectation of staphylococcal sepsis. He had a creatinine of 57 μM·L⁻¹, haemoglobin of 124 g·L⁻¹ and a white count of 14x10⁹·L⁻¹, with 80% neutrophils. Hepatitis surface antigen was negative and liver enzymes were normal.

Eight hours after admission the patient became hypotensive, blood pressure (BP) 60/40 mmHg and his temperature fell to 35°C. His pulse was inappropriately slow at 60 bpm, but an electrocardiogram (ECG) showed sinus rhythm. He responded to intravenous fluids, and was clinically much improved the next morning. The erythematous lesions on his arms had almost completely resolved.

Blood cultures grew a non-typable group A strepto-coccus. Benzyl penicillin three grams six hourly was substituted for the erythromycin and the flucloxacillin was discontinued on the seventh day. A low grade pyrexia persisted over the next seven days. During this time the patient had a normal liver ultrasound and liver function tests, excluding a liver abscess and chronic hepatitis. Echocardiogram revealed no vegetation and normal valves. At this time he had developed signs of a right pleural effusion, and repeat chest X-ray documented a loculated effusion at the right base and an abscess in the left lower lobe. "Thin" sterile pus was obtained on ultrasound guided aspiration.

By day 20 the patient's chest X-ray had improved considerably, and his C reactive protein had fallen from 139 mg·L⁻¹ to 21 mg·L⁻¹ (normal <6 mg·L⁻¹). Eighteen days after admission teichoic acid antibodies were negative, suggesting that staphylococci had not played a part in his illness. The benzyl penicillin was discontinued on
day 20, and he was discharged the following day having made a good recovery.

Prospects of the patient not returning to intravenous drug misuse were considered to be slim. He has defaulted from follow-up and has no general practitioner. We understand from contacts that he has developed no serious problems five months after discharge.

Discussion

Streptococcal sepsis is again being recognized as an important cause of severe systemic illness [4] in addition to pharyngitis and cellulitis. The potential of group A streptococci to cause fulminant septicemia is well documented [5, 6], although often receiving brief mention in major texts. Life-threatening illness often occurs in the absence of predisposing conditions. The current trend against antibiotic treatment for minor respiratory tract infections may be contributing to this increase [7].

IVDMs are at increased risk of sepsis, partly due to reduced host defences, unsterile injecting techniques [8], and periods of depressed consciousness which compromise pulmonary protective mechanisms and lead to aspiration [1]. HIV infection also predisposes to bacterial sepsis [9]. A study in Harlem, New York, of 85 clinically diagnosed cases of endocarditis [10] included 43 IVDMs. Of the seven cases of *S. pyogenes* infection six were drug users, all of whom had signs of pulmonary infection.

Since the documented large epidemics of the 1960's, streptococcal pneumonia has been seen only in sporadic cases, often with an underlying problem [4]. In streptococcal septicemia the respiratory tract is the most frequently involved site after skin and soft tissue sepsis [5]. Development of disease is usually presumed to be secondary to pharyngeal colonization, although many cases have negative throat swabs at the time of presentation [11]. It is likely that the pneumonia is due to haematogenous seeding of the lungs, rather than the bacteremia being secondary to transbronchially acquired infection. This case may have been due to bacteremia associated with chaotic injecting rather than pharyngeal infection. The multiple cutaneous lesions were also compatible with this. Given the high mortality of streptococcal septicemia [6], the favourable outcome of streptococcal pneumonia is striking. Thus, pneumonia may be a feature of the natural progression of streptococcal sepsis in those who are not initially overwhelmed by septicemia. This is to be distinguished from adult respiratory distress syndrome which has a poor prognosis [6]. Severe disease is not always associated with toxin producing strains [12] and may occur in the absence of bacteremia [6].

Streptococcal pneumonia is characterized by the abrupt onset of fever, chills and cough, with marked pleuritic chest pain. Pulmonary abscesses and empyema are characteristic features, in contrast with the spreading cellulitis usually associated with *S. pyogenes* infections. A history of an antecedent sore throat is often absent.

Most series suggest that in the antibiotic era streptococcal pneumonia is not a disease associated with a high mortality [11]. Benzyl penicillin was effective treatment, although in 60% of cases the fever took one week to settle. As in this case the empyema usually appears to resolve quickly with antibiotics. Extensive drainage procedures are not often required and there is little long-term morbidity. The development of abscesses may lead to hesitation in the acceptance of streptococci as the aetiological agent. With the increasing use of 4-quinolones this has important implications for the choice of empirical treatment of infections in such patients. Although ciprofloxacin is proving to be a useful antistaphylococcal agent [13], its activity against *S. pyogenes* is poor. Misdiagnosis could result in the suboptimal treatment of a serious infection whose causative organism remains very sensitive to penicillin.

We conclude that this case is a timely reminder of the underestimated role of streptococcal sepsis in IVDMs and of the potentially aggressive nature of streptococcal infection.

References


RÉSUMÉ: Streptococcus pyogenes semble être devenu une cause rare de pneumonie. Toutefois, vu l’augmentation récente des infections par S. pyogenes, cette situation est susceptible de se modifier. Un drogué par voie intraveineuse a développé de façon aiguë de la fièvre et des frissons. À l’examen, la douleur thoracique de type pleuritique était le symptôme dominant, tandis que plus tard il a développé des abcès pulmonaires et un empyème. La réponse fut bonne à la benzyl penicilline, et les lésions pulmonaires se sont résorbées complètement. Quoique ce tableau clinique soit caractéristique de la pneumonie à Streptocoque pyogenes, elle aurait pu facilement être confondue avec une septicémie à Staphylocoque doré.