DISSEMINATED GONOCOCCAL INFECTION DUE TO PENICILLINASE-PRODUCING STRAIN OF NEISSERIA GONORRHOEAE IN A PREGNANT WOMAN — A CASE REPORT

T Thirumoorthy
C T Lee
T Tan

Middle Road Hospital
250 Middle Road
Singapore 0718

T Thirumoorthy, MRCP (UK)
Senior Registrar

C T Lee, M Med (Int Med), MRCP (Ireland)
Registrar

T Tan, FRACP
Medical Director

SYNOPSIS

A penicillinase-producing strain of Neisseria gonorrhoeae was isolated from the cervix and pharynx of a pregnant woman presenting with dermatitis arthritis syndrome. This disseminated gonococcal infection (DGI) was cured with intramuscular Ceftriaxone 500 mg daily for ten days. A brief review of disseminated gonococcal infection is attempted.

INTRODUCTION

Gonorrhoea remains a major problem in Singapore. The incidence of gonorrhoea in Singapore in 1983 was 468 per 100,000 population. In the same year 1983, a total of 5,528 cases were seen in Middle Road Hospital. Out of these, 1,793 cases, (32.4%) were penicillinase-producing strains (1). The complications of gonorrhoea seen in Middle Road Hospital include pelvic inflammatory disease and Bartholin's abscess in the females and epididymorchitis, periurethral abscess and median raphe abscess in the males. It is estimated that almost 1% of untreated gonococcal infections developed dissemination. However this is the first patient with disseminated gonococcal infection (DGI) reported from Middle Road Hospital. Cases due to penicillinase-producing orgaisms are even rarer. Disseminated gonococcal infection (DGI) is usually due to non-penicillinase producing organisms. A survey of the literature revealed seven cases of DGI due to penicillinase producing organisms (2,3,4,5). We report yet another case, which represents the first reported case of DGI seen in Singapore.

CASE REPORT

The patient was a 30-year old Chinese female housewife. She complained of recurrent fever, skin rash and joint pains of 5 weeks' duration. She was well till the 21st of February 1984 when she started having fever, vomitting and pain and swelling over the ankles, hands and right shoulder followed by painful red spots on the finger of her right hand. She was treated by a general practitioner with erythromycin, diazepam and paracetamol for 2 days after which all her symptoms cleared.

She had recurrence of the fever and joint pains and swelling on the 6th of March 1984 with more red spots over the fingers and the lower legs. She was treated by the same general practitioner with the same medication. She again recovered with no trace of the skin lesions after 3 days.

She had her third recurrence on the 27th of March 1984. This time the joint pains were more severe and there were more red spots. She was referred by her Obstetrician to Middle Road Hospital as a dermatological problem. She did not have any other symptoms. In particular, she did not have any dysuria, frequency or vaginal discharge. She had regular sex with her husband throughout pregnancy but she denied any extramarital sex or oro-genital sex. There were no past history of other sexually transmitted diseases. There were no similar rash in her previous pregnancies. She has 3 other children. Her husband is a businessman who travels overseas frequently. He was seen in MRH in April 1984 and was free from any sexually transmitted disease.

Physical examination on admission revealed that she was frebrile. There were scattered small purpuric lesions over the fingers of the right hand, the right lower limb especially around the knees and the ankles. There were also some erythematous vasculitic papules over the right foot. Both ankles were red, swollen and tender with minimal effusion and some limitation of movements, the right ankle being more severe than the left. The knees showed evidence of arthritis. She was pregnant at 34 weeks. The throat was normal and vaginal examination revealed mild cervicitis with yellowish vaginal discharge.

The following investigations were unremarkable: haemoglobin, total white cell count and differential count, Rheumatoid Factor, L.E. cells, Antinuclear factor, urine analysis, urine culture, urea/electrolytes, liver function test, throat swab culture and sensitivity, uric acid, VDRL and FTA/ABS. Urethral smear and culture for gonococcus (GC), vaginal smear and culture for Trichomonas and monilia, rectal smear and culture for GC, were negative. Cervical smear for GC revealed extra-cellular gm-ve diplococci while the cervical culture for GC grew Penicillinase-producing Neisseria gonorrhoeae (PPNG). The throat culture on Thayer-Martin media also grew PPNG.

The patient was treated with intramuscular ceftriaxone (Rocephin) 500 mg daily for 10 days, paracetamol and bed rest. Fever subsided on the first day of injection. There was decrease in the joint swelling and pain on the third day after the injection. Skin lesions started to fade on the 4th day. The erythrocyte sedimentation rate (ESR) did not fall with treatment. It increased from 74 to 103 despite 9 days of treatment. Repeat smears and cultures for gonorrhoea from the throat, urethral and cervix were negative on the 14th day of onset of treatment. The patient was last seen on the 27th April 1984. There were no more rash, joint pains or swelling. The ESR fell to 70 mm.

The patient had a lower segment caesarean section on the 5th of May 1984 for poor progress after induction for static weight. A baby girl of 2.3 kg was born with some features of intrauterine growth retardation. There was no ophthalmia neonatorum. The placenta was grossly normal. The baby was subsequently reported to be growing well.

DISCUSSION

Disseminated gonococcal infection (6,7) is a clinical syndrome consisting of fever, arthritis and dermatitis, with or without meningitis, endocarditis, myopericarditis, or clinical sepsis, together with the isolation of N. gonorrhoeae from the blood, synovial fluid, skin, cerebrospinal fluid or urogenital areas. The gonococcal strains associated with disseminated infection are usually of the same auxotype, that is, they require arginine, hypoxanthine and uracil (AHU) for their growth. They are usually sensitive to penicillin as they do not produce penicillinase.

Disseminated gonococcal infection occurs mainly in young sexually active females. 70% of cases occur in persons aged 20 to 30 years. 70% to 80% of documented cases occur in women. The symptoms of disseminated gonococcal infection began in the 2nd or 3rd trimester of pregnancy or menstruation in 71% of cases. During menstruation it is known that endocervical shedding of N. gonorrhoeae is maximal.

Gelfand et al (8) divided the clinical manifestations into 3 phases

(a) Haematogenous Phase
(b) Transitional Phase
(c) Joint Localisation Phase

The haematogenous phase is characterised by constitutional symptoms (for example, high fever and chills), polyarthritis with minimal effusion, tenosynovitis and gonococcal dermatitis. The blood cultures are positive in 50% of patients while joint fluid cultures are negative.

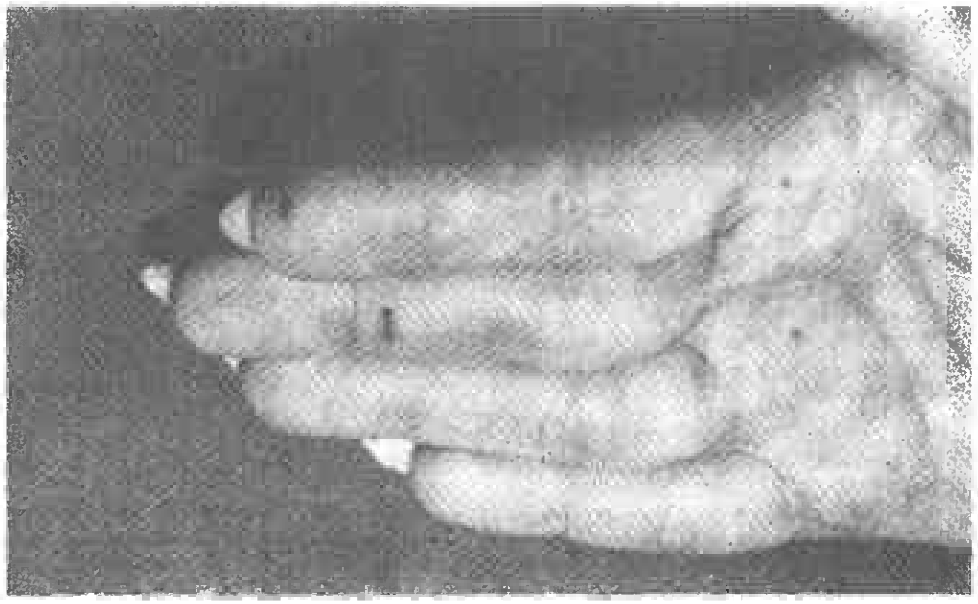
In the transitional phase, joint effusion occurs with gonococcal dermatitis. The blood cultures may or may not be positive while joint fluid cultures are positive.

Untreated patients will enter the joint localisation phase in which systemic symptoms abate and skin lesions disappear. The infection seems to settle in one or two larger joints causing purulent monoarticular or pauciarticular arthritis. In this phase, blood cultures are invariably negative and joint fluid cultures are often positive.

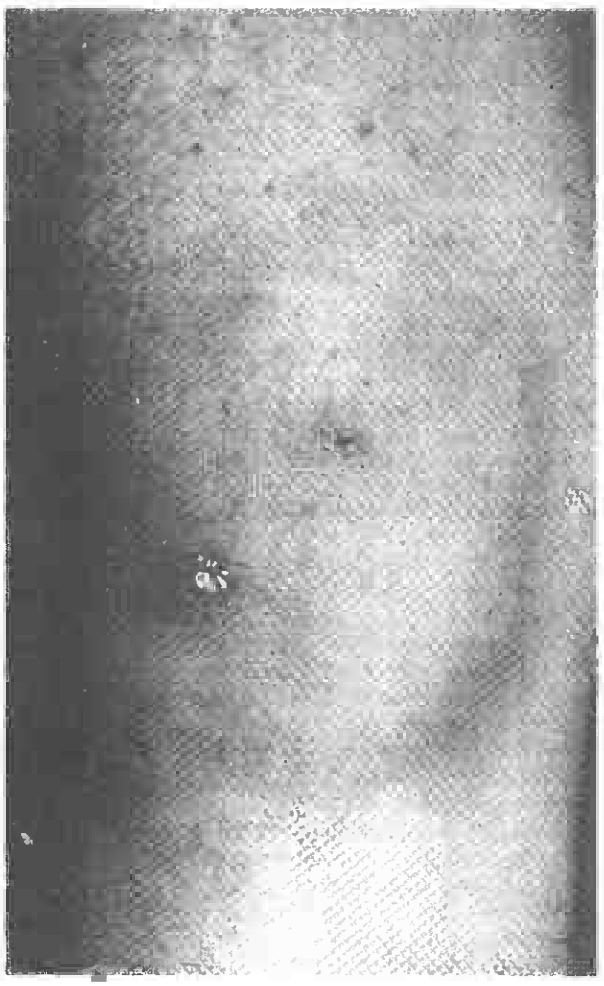
However, a survey by O'Brien and co-workers (9) suggests that there may be two different types of disseminated gonococcal infection — one associated with suppurative arthritis, in which blood cultures are negative, the other associated with tenosynovitis and skin lesions. In disseminated gonococcal infection, the knee is involved in more than 50% of cases, followed in decreasing order of frequency by the shoulders, ankles, wrists and small joints of the hand. If affected joints are not treated, the articular surface may be destroyed and fibrous or bony ankylosis may follow.

The skin lesions usually appear on the extremities (especially on the hands and around affected joints) and number between 5 to 20. They are often painful and have an asymmetrical distribution. The lesions start as erythematous papules which become pustular and haemorrhagic with necrotic centres. Lesions on the palms and soles may appear purpuric. Generally resolution occurs in 4–5 days but cropping may occur during febrile episodes. Other rarer manifestations include endocarditis with emboli to the cerebral, renal and peripheral arteries, myocarditis, pericarditis hepatitis and meningitis.

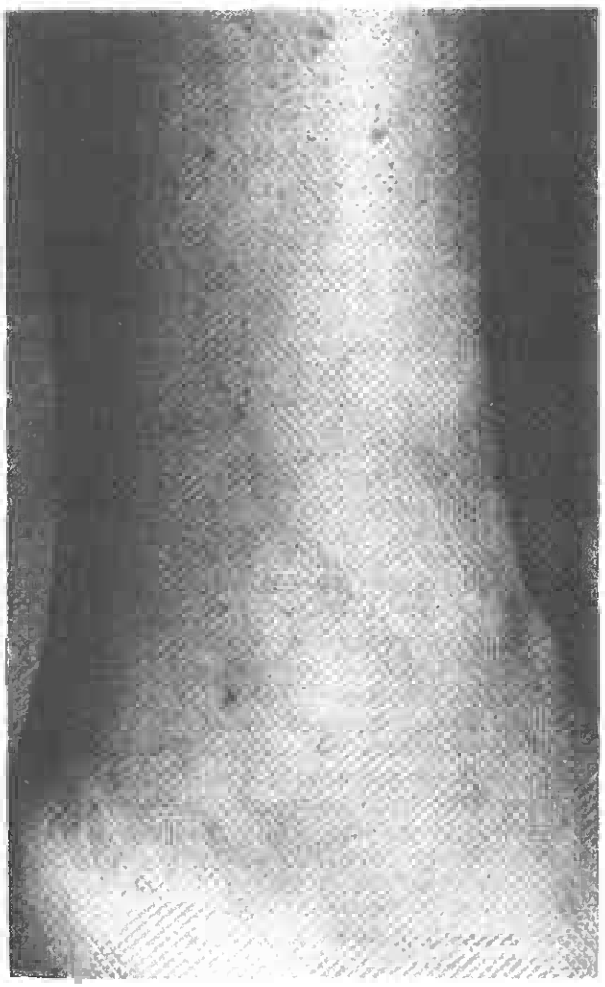
The pathogenesis of DGI is believed to be based on a circulating immune complex process. Daniel et al (10)



Haemorrhagic papules on the index and mid fingers of the right hand.



Haemorrhagic and erythematous papules on and around the right knee region.



Haemorrhagic papules around the right ankle and arthritis of the right ankle.

have shown that high levels of circulating immune complexes are found in a majority of patients with disseminated gonococcal infection as opposed to localised genital infection. They also showed that in patients with DGI, immune complexes closely paralleled the disease activity and negatively correlated with complement levels. Moreover, gonococcal antigen has been detected by immunoflourescence in sterile skin lesions and gonococci have been identified by electron microscopy in synovial membrane from which no organisms could be cultured (11).

Treatment of disseminated gonococcal infection (12.13) depends on the strains of the Neisseria gonorrhoeae. Penicillin is still the treatment of choice for non-PPNG strains. Strains which produce penicillinase are now best treated with parenteral third generation cephalosporins like ceftriaxone and cefotaxime. Ceftriaxone has the advantage of being an effective treatment for gonorrhoea of all sites. It is also effective for all the complications of gonorrhoea and can be administered as a single daily injection. Spectinomycin continues to be useful in PPNG infection except for its relatively poor results in pharyngeal gonorrhoea and the recent reports of development of spectinomycin resistant gonococcus (14.15).

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