Near fatal puerperal fever due to *Mycoplasma hominis*

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Summary: We describe a case of severe septicaemia caused by *Mycoplasma hominis* in a 23 year old patient following childbirth. She developed disseminated intravascular coagulation and acute respiratory distress syndrome which have not hitherto been described in association with septicaemia due to this organism. Investigation and treatment leading to full recovery is outlined.

Introduction

Mycoplasma hominis is a common inhabitant of the female genital tract and is a well recognized cause of post-partum fever.¹

Typically patients are not severely ill. They have a low grade fever for up to 48 hours after delivery, recovering uneventfully without antibiotic treatment.² We have been unable to discover any previous reports of life-threatening sepsis with disseminated intravascular coagulation and adult respiratory distress syndrome due to this species of *Mycoplasma*.

Case report

A 23 year old patient, gravida 3, para 2, was delivered of a normal male infant after induction at term and a 4-hour labour. Her pregnancy had been normal apart from folic acid deficiency anaemia. The placenta was intact. Forty eight hours after delivery she was discharged from hospital but within 4 hours she became feverish with rigors, headache, abdominal pain, thirst and polyuria. Three days later, having failed to respond to amoxycillin prescribed for a presumed urinary tract infection, she was readmitted to hospital.

On admission she was dyspnoeic, feverish (39.4°C) and hypotensive (100/60 mmHg) with a rapid pulse (240 beats/min). Examination revealed pulmonary crepitations and hepatosplenomegaly. The uterus was palpable up to the umbilicus and was not well contracted. The cervix was widely dilated and slight uterine tenderness was present, more on the right side than the left. The lochia was not offensive. The blood count showed typical

changes of disseminated intravascular coagulation (DIC) with haemoglobin 7.0 g/dl, platelets $71 \times 10^9/l$, reticulocytes 23% and fragmented distorted red cells on the peripheral blood film. Subsequent coagulation studies were consistent with this diagnosis. The prothrombin time was prolonged to 20 seconds (control 14 seconds) and kaolin-cephalin clotting time to 133 seconds (control 30 seconds). Fibrin degradation products were 40 mg/l. The white blood cell count was $7.3 \times 10^9/l$ and a differential count showed 95% neutrophils with left shift. The C-reactive protein was 324 mg/l; liver function tests revealed a cholestatic picture. Her $PaCO_2$ was 2.7 kPa, PaO_2 5.6 kPa on air and arterial pH 7.51. The chest X-ray showed widespread opacity described as 'ground glass'.

A provisional diagnosis of septicaemia was made and after blood cultures and a high vaginal swab had been taken, treatment was commenced with a one gram bolus of methylprednisolone together with metronidazole, gentamicin and ampicillin. The DIC was treated with intravenous heparin and blood products. Despite 100% oxygen via a face mask, her oxygenation deteriorated to a PaO_2 of 4.4 kPa and ventilation was commenced 8 hours after admission. Right atrial and later pulmonary wedge pressure monitoring was used to monitor fluid balance and haemodynamics.

After 48 hours her coagulopathy and infection seemed to be controlled but she developed further pyrexia. Intravenous ciprofloxacin was added after further blood cultures had been taken. She had no further episodes of pyrexia and continued to improve. Throughout the illness her renal function remained good. There was some indication of a reduction in glomerular filtration rate on the second day of illness when the serum creatinine rose to 150 $\mu\text{mol/l}$ (upper limit of normal range 124 $\mu\text{mol/l}$), but this had returned to normal the following day.

After 8 days ventilation she was extubated when

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her chest was clinically and radiologically clear and her hepatosplenomegaly had improved. She received a total of 14 days' antibiotics and was discharged 16 days after admission with a PaO_2 of 15 kPa and haemoglobin 13.3 g/dl. Her son had remained well.

A urine specimen taken on the day of admission was sterile. A high vaginal swab yielded *Bacteroides* species and numerous pus cells were seen on Gram film. *M. hominis* was not isolated despite the use of a selective/enrichment medium. A slow-growing pleomorphic Gram-negative bacillus was isolated from the anaerobic bottles of two blood cultures, one taken on the day of admission (when the patient was receiving amoxycillin) and one taken the day after admission (after commencement of gentamicin, metronidazole and ampicillin).

The bottles required 3 and 6 days' incubation respectively before the radiometric blood culture system gave a 'positive' reading. Subculture on to blood agar produced minute colonies visible with a hand lens after a further 3 days' incubation in anaerobic/ CO_2 enriched atmosphere. Attempts to identify the organism using conventional laboratory techniques were unsuccessful and it was identified as *M. hominis* by the Mycoplasma Reference Facility of the National Collection of Type Cultures.

Discussion

The association between *M. hominis* and infections of the genital tract have been recognized for at least 30 years.³ It was first reported to cause post-partum septicaemia by Tulley and Smith in 1968.⁴ Since then a number of investigators have drawn attention to the importance of *M. hominis* in post abortion and post partum fever.^{1,2,5} Platt *et al.*¹ followed up 535 patients after vaginal delivery and found that *M. hominis* septicaemia was the commonest cause of fever.

Almost all reports of *M. hominis* septicaemia following abortion or childbirth have emphasized the benign nature of the infection. Even a patient with severe immunosuppression secondary to

malignant lymphoma, recovered from *M. hominis* septicaemia without antibiotic treatment effective against the infecting organism.⁶

DIC is a recognized complication of *M. pneumoniae* infections⁷ but has not been recorded in association with *M. hominis* septicaemia, or with other conditions such as pelvic inflammatory disease and pyelonephritis where *M. hominis* acts as a primary pathogen.

It is likely that the source of *M. hominis* in our patient was the genital tract. Studies of vaginal flora in asymptomatic premenopausal, non-pregnant women have shown that this organism was present in 12 to 22% of the women studied.⁸ We failed to isolate *M. hominis* from a high vaginal swab taken on the day of admission. Possibly overgrowth of *Bacteroides* species obscured the tiny colonies of *M. hominis* on blood agar. No growth occurred on the selective medium we use to detect *M. hominis* in genital specimens. This medium contains thallos acetate at a concentration of 0.01% and did not support the growth of the blood culture isolates.

It is possible that our patient's acute respiratory failure was a direct result of *M. hominis* pneumonia and that her pregnancy and childbirth were incidental to her illness. *M. hominis* can be isolated from the respiratory tract of up to 3% of healthy adults.⁹

However, unlike pneumonia due to *M. pneumoniae*, that due to *M. hominis* seems to be extremely rare in immunocompetent individuals, although cases have been reported in patients with profound immunosuppression.¹⁰

M. hominis is sensitive to tetracycline, but many strains are resistant to gentamicin and other antibiotics used for the treatment of septicaemia. Ciprofloxacin has been shown to be active *in vitro* against *M. hominis*, but evidence of its clinical efficacy is lacking.¹¹ It is difficult to know whether it contributed to our patient's recovery.

We therefore feel that in patients with severe post partum septicaemia, *M. hominis* should be considered as a possible cause and antimicrobial treatment should be selected with this in mind.

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