Chlamydia trachomatis as a possible cause of peritonitis and perihepatitis in a young woman

J PAAVONEN* AND V V VALTONEN†

From the *I-II Departments of Obstetrics and Gynaecology, University Central Hospital, Helsinki, and the †Department of Serology and Bacteriology, University of Helsinki, Finland

SUMMARY In a 20-year-old woman who developed pelvic peritonitis and perihepatitis, Chlamydia trachomatis was isolated from the lower genital tract. There was a significant rise in the antibody titre against C trachomatis, suggesting that an ascending infection by this organism may have been responsible for the condition.

Introduction

The syndrome of perihepatitis is not a rare complication of gonococcal pelvic inflammatory disease (PID).1 2 Perihepatitis associated with PID is also known as the Fitz-Hugh-Curtis syndrome.3 4 Recently, Müller-Schoop et al5 found serological evidence that Chlamydia trachomatis in the absence of gonococcal infection may play an important role in this peritoneal inflammation. A case of PID due to C trachomatis with exudative ascites and pain and tenderness in the right upper quadrant suggestive of perihepatitis in a young woman is presented.

Case report

A 20-year-old woman was admitted to hospital because of pelvic pain. The patient was para 1 with no miscarriages. She had previously been well with a regular menstrual cycle.

Past history

She had had a normal delivery eight months previously; during pregnancy she had been examined at the outpatient clinic several times because of lower abdominal pain. Laboratory examination had shown pyuria without significant bacteriuria on three separate occasions. At 21 weeks of pregnancy she had been treated with nitrofurantoin 50 mg three times daily for three weeks. Cultures for C trachomatis were not performed at that time. An intrauterine device (IUD) was inserted five months after delivery.

Address for reprints: Dr J Paavonen, I-II Departments of Obstetrics and Gynaecology, University Central Hospital, Haartmaninkatu 2, SF-00290 Helsinki 29, Finland

Received for publication 15 January 1980

On examination the following day the patient appeared well. Her temperature was normal and no pelvic mass could be found. However, Murphy's sign was still strongly positive. Her IUD was then removed; there was no growth of aerobic or anaerobic bacteria on culture. Urine culture for bacteria gave a negative result. Serum urea, serum and urine amylases, serum electrolytes, and serum transaminases (on four consecutive days) were all within normal limits. Cultures of stool specimens on three consecutive days gave negative results for yersinia, salmonellae, and shigellae. Blood culture for bacteria gave repeatedly negative results. Culture of the culdocentesis aspirate yielded a Peptostreptococcus, sensitive in vitro to penicillin and metronidazole but resistant to tetracycline; the fluid was not cultured for chlamydia. Gynaecological examination five days later was normal. The ESR was 25 mm in the first hour, WBC count, 5·9 x 10⁹/l, and the IFAT titre for antichlamydial antibodies on serum obtained nine days after the first specimen had risen to 1024 (an eightfold increase). The patient was discharged on the tenth day after admission. Follow-up examination showed no abnormalities and the patient was asymptomatic.

Discussion

Except by laparoscopy, there is no specific test for diagnosing perihepatitis. Severe right upper abdominal pain and tenderness may be indistinguishable from acute cholecystitis. It has been previously estimated that about 3% of patients with signs and symptoms of acute cholecystitis actually have gonococcal perihepatitis. On the other hand, the incidence of gonococcal perihepatitis has been estimated to be less than 10% in women with gonococcal infection. C trachomatis has been implicated as an important cause of non-specific genital infections, which are at present a major health problem.

Recent results have provided strong support for the view that acute salpingitis may, in many cases, be caused by C trachomatis. In this case, chlamydial infection was confirmed by positive culture results from cervical and urethral specimens. Furthermore, high antichlamydial antibody titres with a significant rise between the acute and convalescent phases suggested that C trachomatis actively participated in this systemic disease. Concomitant infection with N gonorrhoeae was definitely excluded by negative culture results. Further support for the aetiological role of C trachomatis was obtained from the poor response to the initial treatment with penicillin and metronidazole, both of which are known to be ineffective against chlamydia. However, when doxycycline treatment was started the patient rapidly responded clinically. Acute cholecystitis was an unlikely cause of the patient's symptoms as consecutive liver function tests showed normal results.

The fact that culture of the culdocentesis aspirate yielded Peptostreptococcus is interesting and raises the question of its possible aetiological role in the disease. Together with the modern techniques of anaerobic bacteriology, studies using culdocentesis have suggested that obligate anaerobes often play a part in the aetiology of acute PID. However, caution should be used in interpreting the bacteriological results from specimens obtained by culdocentesis because of possible contamination of the specimens by micro-organisms from the vaginal flora. In this case the Peptostreptococcus isolated was sensitive to penicillin and metronidazole in vitro, but this combined treatment was ineffective clinically, thus not supporting its aetiological role.

It is still uncertain how the infection reaches the peritoneum and the subphrenic space. A small amount of exudative fluid is not uncommon with PID and is thought to be produced by the inflamed tubal and ovarian tissue. The precise aetiology of ascites, or overproduction of fluid within the peritoneal cavity, is difficult to establish. However, it may result from the rapid spread of a genital tract infection to the peritoneum, which—secondary to inflammation and adhesion formation—blocks the normal lymphatic pathways for excretion of fluid from the cavity. Thus perihepatic inflammation is most likely caused by infection which has ascended from the pelvis.

In conclusion, C trachomatis, a prevalent sexually transmitted organism, may be a probable cause of exudative peritonitis and perihepatitis as a complication of acute PID. This syndrome has earlier been related only to gonococcal PID. This report may encourage clinicians to look for evidence of chlamydial infection in similar cases.

References

Chlamydia trachomatis as a possible cause of peritonitis and perihepatitis

Chlamydia trachomatis as a possible cause of peritonitis and perihepatitis in a young woman.

J Paavonen and V V Valtonen

*Br J Vener Dis* 1980 56: 341-343
doi: 10.1136/sti.56.5.341

Updated information and services can be found at: [http://sti.bmj.com/content/56/5/341](http://sti.bmj.com/content/56/5/341)

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to: [http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to: [http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to: [http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)