# Gonococcal Infection as a Cause of Acute Abdomen

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**Abstract:** Gonococcal infection in the postantibiotic era continues to cause disseminated and severe disease in some patients. The differential diagnosis of pain in the lower abdomen in young women is difficult. Our case report described a 19-year-old patient who presented with acute abdomen as a result of Gonococcal infection, assessed as a local complication, pelveoperitonitis: pelvic inflammatory disease. The message of our case report is sexually transmitted infections should invariably be considered in young women and searched for accordingly.

## Introduction

The differential diagnosis of pain in the lower abdomen in young women is difficult. It is based on the patient's history, clinical investigation of signs of peritoneal irritation and laboratory tests (proinflammatory parameters of systemic bacterial infection: C-reactive protein, procalcitonin, leukocytosis). Abdominal ultrasound imaging makes standard part of the differential diagnosis. In young women, co-operation between a surgeon and a gynaecologist is needed. In some ambiguous cases, the surgeon has to carry out laparoscopy (laparotomy) to establish the diagnosis.

The incidence of sexually transmitted infections (STI) currently shows an increase. Gilliland reports the rectal form of gonorrhoea in more than 45% of homosexual men. This upward trend started in about 1989 [1]. Accurate data are not available because of inadequate reportability of gonorrhoea by GPs. Treatment is often based on clinical symptoms rather than on bacteriological detection [2]. Establishing the incidence of gonorrhoea also poses a problem for other reasons: gonorrhoea may change the clinical picture of diverse diseases and still remains a diagnostic and therapeutic challenge. The present communication is the case report of a 19-year-old woman with systemic Gonococcal infection who presented with acute abdomen.

## Case Report

A woman presented to an outpatient unit of gynaecology assuming her problems were due to staphylococcal toxic shock syndrome while using vaginal pads for her period. She reported weakness, general muscle pain and no fever. Her history on admission did not reveal any serious problems; she gave birth to a healthy child six months before. She reported pain in the lower abdomen, no radiating pain, no nausea or vomiting. On examination, she had fever ($T_{ax}$ 38.3°C), her heart rate was 90 beats/min and blood pressure was 120/80 mmHg. The gynaecologist found muscle contraction in the lower abdomen, more pronounced on the right side, without any signs of peritoneal irritation. Laboratory tests revealed leukocytosis of 19.8 and an elevated C-reactive protein level of 90 mg/dl. The patient was admitted to a gynaecology clinic for pain in the lower abdomen lasting for three days for observation. A native abdominal scan and ultrasound did not reveal any pathological changes. Staphylococcus epidermidis 10 was cultured from urine$^{3-4}$. The abdominal pain worsened over several hours and, after consultation with a general surgeon, the

patient was indicated for laparoscopy. It was carried out under general anaesthesia. The surgeon found purulent exudate in the small pelvis. The vermiform appendix did not show any signs of acute inflammation. Mesenteric lymph nodes were not enlarged. The uterus and ovaries were within the physiological range. The patient underwent appendectomy, without postoperative antibiotic prophylaxis. Postoperatively, the patient had fever of up to 38±0.5°C. Passage of gas and stool was restored postoperatively on days 2 and 3, respectively. The woman left the clinic on postoperative day 4 after signing the patient refusal form. No acute inflammatory changes in the appendix were reported by the pathologist.

On August 28, 2003, the woman presented for outpatient follow-up. She was afebrile, without subjective complaints. Her scar was quiet, solid, without secretion or erythema and painless on palpation. Stitches were removed.

On the following day, the patient was afebrile, but a purulent abscess of 1.5 cm in diameter formed in the proximal part of the scar. An incision was performed to evacuate pus, which was sampled for bacteriological investigation (aerobic bacteriological culture was negative), with subsequent lavage and drainage. The patient did not present for a bandage change on the next day.

The diagnosis of gonorrhoeal infection was not established until after the use of a screening programme. A male sexual partner of the patient was shown to have acute gonorrhoeal anterior urethritis; subsequent screening of his sexual partners included our patient shown to have gonorrhoea at 2 months after appendectomy.

Based on the bacteriological finding, the patient was referred to a STD department where she presented on September 8, 2003. On local examination, a purulent discharge from the fistula located in the upper part of the scar was found. A fistula discharge specimen was negative for Chlamydia trachomatis and positive for Neisseria gonorrhoeae in polymerase chain reaction (PCR) tests. Microscopy demonstrated Neisseria gonorrhoeae. Nitrocephin method revealed beta-lactamase production; agent testing showed the specimen to be resistant to penicillins, aminopenicillins and all fluoroquinolones (Table 1). Further, bacteriological investigation documented massive invasion by Staphylococcus aureus, resistant to penicillin and erythromycin.

An oropharyngeal specimen tested culture-positive for Neisseria gonorrhoeae, which was also PCR-detected from the uterine endocervix and urethra.

**Table 1 – Antibiotic susceptibility of Neisseria gonorrhoeae strain 3269/03**

| Antibiotic agent | Limit concentrations for susceptible strains | MIC values for strain 3269/03 | S-susceptible R-resistant |
|---|---|---|---|
| Penicillin | ≤ 0.063 mg/l | >2 mg/l | R |
| Cefotaxime | ≤ 0.5 mg/l | ≤ 0.008 mg/l | S |
| Ciprofloxacin | ≤ 0.063 mg/l | >2 mg/l | R |

Gonococcal culture of a rectal sample was negative. PCR screening for Chlamydia trachomatis was negative. The patient also showed negativity in VDRL, TPHA, Syphilis EIA screen and anti-HIV 1,2 + p 24.

In view of the results obtained, she was admitted to the STD clinic of the General Teaching Hospital, Prague 2 (U nemocnice 2) to be hospitalised from September 12, 2003 to September 19, 2003. Based on the recommendation of an ATB centre, the patient was treated with Rocephin 2.0 g i.m. daily for 7 days. When discharged from the clinic, she was followed up at the STD outpatient department and at 3 obligatory check-ups tested culture and PCR-negative for Gonococcal infection from the specimens as specified above (except for the fistula that resolved). Another STI was detected in the endocervix and urethra during follow-up after treatment: Mycoplasma hominis and Ureaplasma urealyticum, cured by Doxybene 100 mg t.i.d. for 10 days. At a subsequent follow-up visit, the patient was negative.

In December 2003, the patient had a final serological investigation for syphilis and HIV 1, 2 testing as recommended. All tests were negative and the patient was considered "a case" following treatment of gonorrhoea.

**Discussion**

In the Czech Republic, about 7,000 cases of gonorrhoea per year (i.e., 70–80 cases per 100,000 pop.) were reported in the mid-fifties of the twenties century. The incidence of gonorrhoea markedly declined – approximately by a third – in the late 1950's to show a steep rise since 1961, peaking in the late 1960's with 12,000 to 16,000 cases per year (i.e., 120–140 cases per 100,000 pop.). Since then, the incidence showed a progressive downward tendency up to the mid-1980's when the incidence rates became stable and was about the same as in the 1950's with about 6,000 cases reported per year. In 1990–1992, the incidence increased to 7,455 cases per year (i.e., 72.3 per 100,000 pop.). Since 1993, a sharp decline followed with the lowest number of cases, i.e. 880 in 2001, and then a slight rise with 911 cases was reported in 2002 (Table 2).

Although the reported incidence of syphilis compared to gonorrhoea is slightly higher, the rates of both infections are becoming closer one to the other.

The decline in the incidence of gonorrhoea is explained by reduced virulence of the causative agent, possible misdiagnosis, bias due to therapy of other infections, self-instituted therapy, and underreporting. Although women are more prone to acquire gonorrhoeal infection through sexual intercourse with an infected partner than men, the infection has been diagnosed less frequently in women whose complaints are attributed to vaginal discharges. Chronic and asymptomatic forms of gonorrhoea are recorded more frequently in women.

In 2002, out of the 911 reported cases, 277 and 634 were diagnosed in females and males, respectively, 182 and 580 were acute infections, respectively, and 95 and 54 were chronic infections, respectively.

The highest rates of gonorrhoea are detected in women aged between 15 and 34 years and in men aged between 20 and 34 years.

In a controlled study, women using oral contraceptives showed a 70% higher incidence of Gonococcal infection compared to those with no contraception [3].

Many infected patients reported a history of crack or injectable/intravenous illegal drug use or of sexual contact with a drug user. Gonococcal infection in the postantibiotic era continues to cause disseminated and severe disease in some patients. Disseminated infection occurs more often in women than in men and is associated with pregnancy and pharyngeal gonorrhoea [2]. Clinical presentations of disseminated disease include Gonococcal endocarditis [4], meningitis caused by N. gonorrhoeae [5], acute arthritis-dermatitis syndrome, [6] and rare cases of acute maxillary sinusitis and acute respiratory distress syndrome [7].

Our case report described a 19-year-old patient who presented with acute abdomen as a result of Gonococcal infection, assessed as a local complication, pelveoperitonitis: pelvic inflammatory disease, or PID. Factors supporting infection ascension in this particular case included menses, vaginal pad, intercourse during period, hygiene, promiscuity/prostitution, unprotected intercourse.

Culture and/or PCR positivity was documented in specimens from the oropharynx, endocervix, and rectum. She had sex with about 50 men within a period of 6 months. The pain in the lower abdomen in women may be associated with inflammation in the pelvis due to salpingitis, endometritis,

**Table 2 – STD cases reported in 1990–2002. Chancroid, lymphogranuloma venereum and granuloma inguinale have not been reported since 1994**

| | Total | | Per 100,000 pop. | |
|---|---|---|---|---|
| Diagnosis | Syphilis | Gonorrhoea | Syphilis | Gonorrhoea |
| year | A 50–53 | A 54 | A 50–53 | A 54 |
| 1990 | 164 | 6463 | 1.6 | 62.4 |
| 1991 | 249 | 7283 | 2.4 | 70.7 |
| 1992 | 265 | 7455 | 2.6 | 72.3 |
| 1993 | 233 | 4700 | 2.3 | 45.5 |
| 1994 | 369 | 2948 | 3.6 | 28.5 |
| 1995 | 433 | 2036 | 4.2 | 19.7 |
| 1996 | 551 | 1194 | 5.3 | 11.6 |
| 1997 | 604 | 1098 | 5.9 | 10.7 |
| 1998 | 687 | 1055 | 6.7 | 10.3 |
| 1999 | 731 | 995 | 7.1 | 9.7 |
| 2000 | 967 | 888 | 9.4 | 8.6 |
| 2001 | 1376 | 880 | 13.4 | 8.6 |
| 2002 | 976 | 911 | 9.6 | 8.9 |

parametritis, oophoritis (or adnexitis) or pelvic peritonitis. Functional metrorrhagia occurs frequently.

A possible explanation may be ascending infection that spreads to the ovaries and peritoneum, possibly caused by many bacterial species including STD causative agents: N. gonorrhoeae, C. trachomatis, Mycoplasma hominis, Ureaplasma urealyticum, anaerobes, etc.

All plausible causes of peritonitis should be taken into account in the differential diagnosis. A history of prostitution or crack or injectable/intravenous illegal drug use and rapid microbiological diagnosis of Gonococcal infection are suggestive of primary peritonitis due to Gonococcal infection. Nevertheless, in the presence of objective sings of peritonitis and inconclusive diagnosis, the surgeon has to perform a laparoscopy (or laparotomy).

When suggestive pathology is found in the abdominal cavity, an abdominal exudate sample should be investigated microbiologically to allow institution of antibiotic therapy as early as possible. Our recommendations for the treatment of Gonococcal infection are ceftriaxone 1.0 g IV daily, or cefotaxime 1.0 g IV every 8 h, or ceftizoxime 1.0 g IV every 8 h. Unfortunately, the behaviour of our patient is typical of the given population group showing sexual incontinence, lack of self discipline and health care non-compliance (she was discharged from the surgical department on postoperative day 4 after signing the patient refusal form). She did not present for outpatient follow-up appointments. Her sexual life did not change (she had an unprotected vaginal intercourse on postoperative day 5). When followed up after the treatment of gonorrhoea, she was instructed to avoid sexual intercourse but reported to be non-compliant. Even over the following period, the patient does not change her high-risk sexual behaviour and goes on living on prostitution.

**Conclusion**

The Czech Republic has a system of mandatory screening for gonorrhoea (see Bulletin of the Ministry of Health of the Czech Republic 12/1997, Article 10, Investigational Procedure Standards). Proper treatment of gonorrhoea requires co-operation on the part of the patient. Our patient developed oropharyngeal gonorrhoeal re-infection, including non-sexual transmission to her one-year-old child (incidence of oropharyngeal gonorrhoea). At the same time, our patient's subsequent pregnancy free of any problems attests to cure of the gonorrhoea without any subsequent post-inflammatory sequel (if untreated: infertility, sterility, chronic epigastric discomfort, extrauterine pregnancy, death).

The message of our communication is STI should invariably be considered in young women and searched for accordingly. Another critical consideration is combined infection (in our case report: gonorrhoea, ureaplasma urealyticum, mycoplasma hominis, and Staphylococcus aureus) and a risk for ascending infection in women.

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