Isolation of *Neisseria gonorrhoeae* from urine obtained by suprapubic puncture of bladders of men with gonococcal urethritis

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SUMMARY The authors examined three urine specimens from each of 24 men with acute gonococcal urethritis. Gonococcal concentrations in urine were 7×10^3 /ml to 9×10^8 /ml in first samples, 1×10^2 /ml to 5×10^6 /ml in midstream samples, and, in the terminal samples from only 22 men, 8×10^4 /ml. A further 17 men with symptomless gonococcal urethritis were examined. Seven of them yielded 1×10^2 /ml to 2.5×10^5 /ml in first samples and 5×10^5 /ml in midstream samples, and only two yielded 5×10^1 /ml in final samples.

All 24 men with acute gonococcal urethritis, and seven of the men with symptomless gonococcal urethritis who had yielded *N gonorrhoeae* in midstream urine samples, were examined by suprapubic puncture before morning voiding. Four of the 24 men with acute gonococcal urethritis were found to have infection that had ascended into the bladder.

Several workers have discussed the complications, such as prostatitis and epididymitis, of gonococcal infection of the male genital tract.¹⁻⁶ Much less is known about infection of the bladder with *Neisseria gonorrhoeae*.

The main objective of this work was to discover whether such complications of gonorrhoea occur or not. We tried to answer this question on the basis of cultures of urine samples obtained by suprapubic puncture of the bladders of men with acute or symptomless gonococcal urethritis.

Patients and methods

We examined the urine of 24 men with acute and 17 men with symptomless gonococcal urethritis, all aged 18 to 26.

CULTURE

Samples were taken from the first portion, midstream, and terminal portion of urine (5 ml of each). They were diluted from 10^{-1} to 10^{-6} , and 0.1 ml of each sample was inoculated on MBA (modified blood agar

(Imuna Šarišské Michalany, Czechoslovakia) or MBA with LC (lincomycin hydrochloride) 5 µg/ml (Med-export, USSR) and colimycin 20 µg/ml (Laboratoire Roger Bellon). Inoculated plates were incubated for 48 hours at 37°C in 10% carbon dioxide. *Neisseria gonorrhoeae* was identified using standard methods. The concentration was expressed as the number of colony forming units (cfu) of gonococci per 1 ml of urine examined, where one colony equalled one cell of *N gonorrhoeae*. We calculated the arithmetic mean of colonies on three plates (readings of plates massively overgrown with colonies and those that remained sterile were excluded).

The same method was used to examine the urine samples obtained by suprapubic puncture of the bladder before morning voiding, which was performed only on men yielding *N gonorrhoeae* from the midstream specimen of urine. We thus examined all 24 men with acute gonococcal urethritis in that way, but only seven men with symptomless disease. We undertook suprapubic puncture of the bladder of one patient twice on two consecutive days, but only once in all the others.

Culture of all urine samples was started within 30 minutes after they were taken.

SUPRAPUBIC PUNCTURE TECHNIQUE

The prerequisite for suprapubic puncture of the

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bladder is its being well filled, which we secured in our patients by restricting voiding for 10 hours (from 21.00 to 07.00 on the day of the puncture). The filling of the bladder was checked by palpation over the suprapubic region and the imperative need of the patient to void. For each puncture we used a disposable needle (Braunula 2 G 14; B Braun Melsungen AG, West Germany), the puncture being performed, after local disinfection and anaesthesia, through the abdominal wall into the bladder of the recumbent patient, about 2 cm above the symphysis in the midline. We aspirated 10 ml of urine through the puncture needle and transported it immediately in a sterile test tube for bacteriological examination. After the puncture needle had been removed, the spot was briefly compressed with sterile cotton wool. The punctures were tolerated well by the patients, and no complications occurred.

TIMING

To minimise the interval between the diagnosis of gonorrhoea and treatment we decided on the following time schedule for taking samples. The suspicion of gonorrhoea in all the men examined was raised because of clinical symptomatology, gonococcal infection in their sexual partners, and on the basis of microscopy of Gram stained urethral smears. We obtained urethral material and urine sediment from

the first, midstream, and terminal micturition of each man, which we cultured for *N gonorrhoeae* on MBA and MBA with LC. *N gonorrhoeae* was identified from 24 hour culture by standard methods and by examination of the isolated colonies by a coagglutination test (Phadebact gonococcus test; Pharmacia Diagnostics AB). The day before the examination we asked each patient not to void during the night. On the basis of the results of 24 hour culture from these samples we then took from 24 men with acute and seven men with symptomless gonococcal urethritis first the urine samples by suprapubic puncture of the bladder, and then the samples from the first portion, midstream, and terminal micturition which were then examined by the methods already described. We did not wait for the results of these examinations, but started antibiotic treatment immediately. After receiving the results of cultures of urine samples and urethral swabs, we altered the treatment wherever necessary on the basis of tests of sensitivity to antibiotics. All patients were examined and treated as hospital inpatients. Thus the time from the first examination until the treatment of our patients never exceeded 24 hours.

All strains of *N gonorrhoeae* isolated were tested for susceptibility to antibiotics by the plate dilution method,^{7,8} and were screened for production of penicillinase by a rapid iodometric test.⁹

Results

The figure shows the concentrations of gonococci in the four urine samples from (a) the 24 men with acute and (b) the 17 men with symptomless gonococcal urethritis. The first urine samples from the men with acute infection contained 7×10^3 /ml to 9×10^6 /ml, the midstream samples contained 1×10^2 /ml to 5×10^6 /ml, and the last samples contained no organisms (in two patients) to 8×10^3 /ml. These findings differed considerably from those for the 17 symptomless men. Their first urine samples yielded only 1×10^1 /ml to 2.5×10^3 /ml, and only seven midstream and two final samples yielded *N gonorrhoeae*.

The table shows that gonococcal infection of the

Figure 1 Number of gonococci/ml of urine in three samples and in urine obtained by suprapubic puncture from (a) 24 men with acute and (b) 17 with symptomless gonococcal urethritis. Patients without (●), and with (○), infection in the bladder, as shown by suprapubic puncture.

Table Analysis of bacterial flora isolated in urine obtained by suprapubic puncture of bladders of five men with acute gonococcal urethritis

Organism	Case Nos				
	1	2	3	4	5
<i>Neisseria gonorrhoeae</i>	+	+	+	+	-
<i>Escherichia coli</i>	-	+	-	-	+
<i>Streptococcus faecalis</i>	+	-	-	+	-
Plasmacoagglutination negative <i>Staphylococci</i> spp	-	-	-	-	+

Results positive (+) or negative (-).

urinary bladder proved by suprapubic puncture was observed in four patients with acute gonococcal urethritis. Another man with acute gonococcal urethritis yielded 1×10^6 /ml *Escherichia coli* from the bladder, but no gonococci.

Discussion

According to Wiesner and Thompson, 80% of men with gonococcal urethritis present with a purulent urethral discharge and dysuria.¹⁰ These signs confirm those described by Glingar,¹¹ and were also seen in 24 of our patients.

In 7–43% patients with gonorrhoea, however, the disease has an atypical course from the very beginning with mild symptoms such as a slight mucous discharge mostly before the first morning voiding.^{6 10 12–14}

Gonorrhoea is symptomless in 2–10% of men.^{10 15 16} Symptomless gonorrhoea occurred in 17 of our men. Microscopy of Gram stained urethral material showed leucocytes and numerous epithelial cells. Gram negative diplococci were found individually or in small clusters, mostly located extracellularly.

Summing up the number of gonococci in the urine in men with acute gonococcal urethritis, they were most numerous in the first portion. In cases where infection had not ascended to the bladder, 10^4 /ml gonococci were found in midstream and terminal urine specimens. Higher concentrations were highly indicative of infection ascending to the upper parts of the urinary tract, though some of our patients' midstream samples had 10^3 /ml when the culture of urine taken by suprapubic puncture remained sterile. The evaluation of urinary sediment and the test of two or three glasses of urine in men with acute gonococcal urethritis provided only an indication of the need for further examination. This examination is not practicable for the aetiological diagnosis of cystitis, cystopyelitis, or cystopyelonephritis, as all three urine samples from men with acute gonococcal urethritis were opaque. Sediment contained many leucocytes, Gram negative diplococci, or other bacteria and in cases of haemorrhagic urethritis it also contained many erythrocytes. We must emphasise, however, that the urine taken from these men by suprapubic puncture was sterile.

Suprapubic puncture of the bladder was a valuable contribution to the diagnosis of gonococcal infection ascending to the upper urinary tract. It may also help to diagnose infection with other bacteria, such as *E coli* and *Streptococcus faecalis* (table). One of our patients with acute gonococcal urethritis (case 5 in the table) yielded 1×10^6 *E coli* from the bladder. In this patient we undertook suprapubic puncture of the bladder twice a day on two consecutive days, as we could not find *N gonorrhoeae*. We do not think that the finding of plasmacocagulase negative *Staphylococcus* spp in this patient was important as the urine was probably

contaminated during the puncture or in the laboratory. We included its isolation in our results for completeness.

All men whose urine obtained by suprapubic puncture yielded bacteria on culture had acute gonococcal urethritis.

On the basis of our observations we assume that the membranous urethra does not create any obstacle to gonococcal infection penetrating to the prostatic part of urethra. Similarly, the sphincter of the bladder does not prevent the propagation of gonococcal infection beyond the neck of the bladder to the whole bladder. We therefore assume that infection with *N gonorrhoeae* and other bacterial microflora ascends from the urethra to the bladder by the endocanalicular route. In such cases, however, the urethra is supposed to be inflamed throughout its length. Urethroscopy would have shown whether this was the case in our patients, but such an examination is considered to be contraindicated in patients with acute gonococcal urethritis.

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