Identification of Sexually Transmitted Bacteria in Tubo-Ovarian Abscesses through Nucleic Acid Amplification

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Tubo-ovarian abscess (TOA) consists of a purulent collection involving the fallopian tube, ovary, and, occasionally, other adjacent pelvic organs. TOA is clinically interrelated with pelvic inflammatory diseases (PID) and noncollected infection of the uterus, fallopian tubes, and other reproductive organs (1–4). Although TOA can be diagnosed without PID, TOA complicates the treatment of 15% of patients during the course of PID and is codiagnosed in 33% patients admitted for PID (5), suggesting a common pathogenesis (6). However, many authors distinguish these two clinical entities (6) because *Chlamydia trachomatis* and *Neisseria gonorrhoeae* have been identified only in the upper genital tracts of PID patients but never in TOA samples for *C. trachomatis* and only rarely for *N. gonorrhoeae* (6–9).

Using nucleic acid amplification (NAA) with the Roche Cobas 4800 CT/NG assay and a previously described in-house NAA protocol (10, 11) for the identification of *Mycoplasma hominis*, *Mycoplasma genitalium*, and *Ureaplasma urealyticum*, we report NAA detection of *C. trachomatis*, *N. gonorrhoeae*, and *M. hominis* in TOA transvaginal-ultrasound-guided drainage samples (Table 1). Briefly, following antiseptic disinfection, an ultrasound probe is placed in the vaginal lateral fornix closest to the TOA, and the operator then inserts a sterile needle (18 gauge) through a coupled guide allowing drainage.

NAA was also added to the standard culture for both TOA-transvaginal drainage samples and endocervical swabs in screening for associated sexually transmitted disease (STD) performed prior to drainage in accordance with the approval granted to this study by the Obstetrical Gynecology Research Ethical Committee.

Thirty-one TOA samples and related endocervical STD screening samples from 31 patients were also studied by NAA. The median age for the population was 33 years (range, 26 to 45 years), the median corporal temperature was 37.6°C (range, 37 to 38.35°C), the median white blood count was 15.42 G/liter (range, 12.6 to 16.96 G/liter), the median C reactive protein level was 149.5 mg/

liter (range, 67 to 213.5 mg/liter), and the median tubo-ovarian abscess diameter was 56 mm (range, 46 to 70 mm).

C. trachomatis, *N. gonorrhoeae*, and *M. hominis* were identified in 18/31 (58%) TOA samples (Table 1). Vaginal contamination represents a potential limit to our study. However, when the same microorganism was identified in both TOA samples and vaginal swabs, amplified DNA quantities in TOA samples were 10-fold higher than in vaginal swabs (data not shown), suggesting that contamination by *C. trachomatis*, *N. gonorrhoeae*, and *M. hominis* DNA from the vagina could be considered unlikely.

NAA identified for the first time *C. trachomatis*, *N. gonorrhoeae*, or *M. hominis* in TOAs. *M. hominis* is found in approximately 40% of healthy vaginal flora (12), both *C. trachomatis* and *N. gonorrhoeae* are known to induce lower genital tract infections such as cervicitis (6), and we found by a culture-based method several bacteria known to inhabit the vagina in TOA (Table 1). Our data also suggest that TOA infections, similarly to what is known in PID (6), may be caused by upper genital tract invasion by bacteria, sexually transmitted or not, coming from the lower genital tract.

Taken as a whole, our results suggest a common pathogenesis between TOA and PID implicating for both entities common pathogens that reside in the vaginal cavity.

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TABLE 1 Results of nucleic acid amplification (NAA) and bacterial culture in tubo-ovarian abscesses and nucleic acid amplification in vaginal samples

Sample	Antibiotics ^a (h)	Endocervical NAA assay result					Tubo-ovarian abscess NAA assay result					Tubo-ovarian abscess bacterial culture result
		<i>Chlamydia trachomatis</i>	<i>Neisseria gonorrhoeae</i>	<i>Mycoplasma genitalium</i>	<i>Mycoplasma hominis</i>	<i>Ureaplasma urealyticum</i>	<i>Chlamydia trachomatis</i>	<i>Neisseria gonorrhoeae</i>	<i>Mycoplasma genitalium</i>	<i>Mycoplasma hominis</i>	<i>Ureaplasma urealyticum</i>	
1	19	—	—	—	+	—	—	—	—	+	—	No bacterial growth
2	10	—	—	—	—	—	—	—	—	—	—	<i>Escherichia coli</i>
3	15	—	—	—	—	—	—	—	—	—	—	<i>Escherichia coli</i>
4	15	—	—	—	—	—	—	—	—	—	—	No bacterial growth
5	11	+	—	—	+	—	+	—	—	—	—	No bacterial growth
6	2	—	—	—	+	—	—	—	—	+	—	<i>Corynebacterium jeikeium</i>
7	12	—	—	—	—	—	—	—	—	—	—	<i>Escherichia coli</i>
8	53	—	—	—	—	—	—	—	—	—	—	No bacterial growth
9	11	+	+	—	+	+	—	+	—	+	—	No bacterial growth
10	27	—	—	—	—	—	—	—	—	—	—	No bacterial growth
11	25	—	—	—	—	—	—	—	—	—	—	No bacterial growth
12	12	—	—	—	—	—	—	—	—	—	—	No bacterial growth
13	12	—	—	—	—	—	—	—	—	—	—	No bacterial growth
14	15	—	—	—	—	—	—	—	—	—	—	<i>Escherichia coli</i> , <i>Bacteroides fragilis</i>
15	9	—	—	—	—	—	—	—	—	+	—	<i>Escherichia coli</i>
16	24	—	—	—	—	—	—	—	—	+	—	<i>Propionibacterium acnes</i> , <i>B. fragilis</i> , <i>Lactobacillus jensenii</i>
17	16	—	—	—	—	—	—	—	—	+	—	<i>Prevotella bivia</i> , <i>Fusobacterium nucleatum</i>
18	14	+	—	—	—	—	+	—	—	—	—	No bacterial growth
19	18	—	—	—	+	—	I ^b	I ^b	—	+	—	No bacterial growth
20	24	—	—	—	—	+	—	—	—	—	—	No bacterial growth
21	10	—	—	—	—	—	—	—	—	+	—	<i>Klebsiella pneumoniae</i>
22	0	+	—	—	—	—	+	—	—	—	—	No bacterial growth
23	6	—	—	—	+	—	I ^b	I ^b	—	—	—	<i>Haemophilus influenzae</i>
24	21	—	—	—	—	—	—	—	—	+	—	<i>Streptococcus pneumoniae</i>
25	12	—	—	—	+	—	—	—	—	+	—	<i>Streptococcus constellatus</i>
26	12	+	—	—	—	—	+	—	—	—	—	No bacterial growth
27	144	—	—	—	—	—	—	—	—	—	—	<i>Enterococcus faecium</i>
28	12	—	—	—	—	—	+	—	—	—	—	No bacterial growth
29	48	—	—	—	—	—	—	—	—	—	—	<i>Actinomyces neuii</i>
30	20	+	—	—	—	—	+	—	—	—	—	No bacterial growth
31	9	—	—	—	—	—	—	—	—	—	—	<i>Streptococcus intermedius</i> , <i>Eikenella corrodens</i>

^a Data represent the time from antimicrobial initiation to abscess sampling. Antimicrobial treatment was as follows: ceftriaxone at 2 g/24 h, doxycycline at 200 mg/24 h, and metronidazole at 1,500 mg/24 h.^b NAA inhibitor.

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