Review Article

Current Perspectives on the Diagnosis and Management of Proximal Tubal Disease

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Evaluation of the fallopian tubes is an integral part of an infertility work up (Jones and Toner 1993). The first publication on the evaluation of tubal patency was by I. Rubin in 1920 (Rubin 1920). Rubin injected gas transcervically, and demonstrated its presence under the diaphragm on radiographs to provide indirect evidence of tubal patency. The first hysterosalpingogram (HSG) was performed almost 80 years ago with the use of bismuth paste as contrast medium (Yoder and Hall 1991). To date, HSG remains the most widely used test for tubal patency. Other techniques for testing tubal patency include laparoscopy (WHO 1986), radionuclide HSG (Osgur et al 1997) and ultrasound with the use of an echogenic contrast medium (Diechert et al 1989) or color Doppler (Peters and Culam 1991). The main thrust during evaluating the tubes has been establishing normal anatomy (tubal patency, tubal contour and the presence or absence of rugae). The true aim of an infertility work up, however, should be evaluation of the functional status of the fallopian tubes i.e. their ability to result in a pregnancy. The focus of this article will be to review the current literature on proximal tubal disease and evidence that strongly suggests that patent tubes may be functionally compromised i.e. have a reduced capability to result in pregnancy.

The exact incidence of proximal tubal occlusion (PTO) in an infertile population remains unknown (Risquez and Confino 1993). Historically, PTO was generally treated via surgery (Patton et al 1987). Currently, the inital approach towards treatment of PTO is transcervical tubal cannulation using fluoroscopic (Confino et al 1988) or hysteroscopic (Novy 1995) guidance. The first multi-center trial evaluating the use of a balloon tuboplasty catheter ws published by Confino et al. in 1988 (Confino et al 1990). They established tubal patency in 90% of patients and the subsequent pregnancy rate (PR) was almost 40%. An interesting group of patients in this study were patients that were false-positive for PTO i.e they were diagnosd with PTO on initial HSG as well as laparoscopy, but when they presented for transcervical balloon tuboplasty, the tubes were patent. These were patients with what is commonly referred to as "tubal spasm". On subsequent follow up, there were hardly any pregnancies in this group despite having patent tubes. The authors therefore raise the possibility that "tubal spasm" may not be a benign finding, but may be a harbinger of tubal disease. The balloon tuboplasty catheters, however, were not approved by the Federal Drug Administration and they are not commercially available here in the US. This has led to the use of wire-guides as the sole treatment modality to overcome PTO resistant to selective salpingography (SS).

Gleicher et al. (Gleicher et al 1994) initially reported that wire-guides alone were not useful for treatment of PTO. They reported on 25 patients with PTO (10 with bilateral, 12 with unilateral and 3 with a single tube) treated with wire-guides. Tubal patency was obtained in 27 of 35 (77%) tubes. The subsequent PR (1/25; 4%) was dismal and the only pregnancy was an ectopic. This was the first evidence suggesting that the establishment of tubal patency did not necessarily result in subsequent pregnancy.

A similar experience was reported by Woolcott et al (1995). They treated 66 patients with PTO using transcervical catheters. They initially attempted tubal recanalization with SS followed, if necessary, by tubal catheterization with a soft Teflon 2-French catheter and finally, if needed, wire-guide cannulation. Each procedure was terminated once patency had been established without recourse to the next technique. Tubal patency was successfully established in 90% of cases. However, there was significant difference in PR based on the technique needed to treat the PTO. PR were excellent in patients with "mild" PTO amenable to treatment with SS (6/22, 27.2%) or the soft Teflon 2 - French catheter (17/30, 56.6%). There were, however, no ongoing pregnancies in 7 patients with PTO severe enough to require wire-guide cannulation. The authors suggest that patients with PTO that require wire guide cannulation have more severe tubal pathology (salpingitis isthmica nodosa (SIN), periluminal fibrosis or intra-tubal adhesions).

Karande et al (1995) in the meanwhile, reported differential pregnancy rates in patients with patent tubes based on the evaluation of tubal perfusion pressures. They utilized a standardized technique for performing an HSG (which they call a "GynecoRadiological procedure") to detect even the most subtle abnormalities. These included delayed opacification of the tubes, asymmetrical spill, pocketing of dye, and of course unilateral or bilateral tubal occlusion. In these patients they measured tubal perfusion pressures (TPP) by performing SS and measuring resistance to the dye (which was injected by a pump) via a pressure transducer. The pressure data was transmitted to a computer and displayed on the monitor screen. Fortyseven women with normal HSG, by spot film underwent bilateral SS and were subdivided into those with normal (Group I, n=23) and abnormal (Group II, n = 24) TPP. Patients in both study groups underwent identical ovulation induction protocols with either gonadotropins or clomiphene citrate, independent of pressure measurements. Clinical pregnancy rates were then recorded over the ensuing 6 to 10 months. Both groups were similar in etiology of infertility, age, duration of infertility, and gravidity. Women with normal TPP demonstrated a significantly higher pregnancy rate (10/23) than patients with elevated TPP (4/24, $P<0.05$). These data are important because they present a techique to evaluate the capability of fallopian tubes to achieve pregnancy. Moreover, these data confirm the long held suspicion that tubal patency alone is unreliable as a predictor of pregnancy potential.

Karande et al (1995) next investigated the possible etiology of elevated TPP. They assessed 48 consecutive women who within a reasonably short time period had undergone an evaluation of TPP as well as a laparoscopy as part of their infertility work up (Karande et al, 1995). Patients with laparoscopically confirmed endometriosis showed a significantly increased incidence of asymmetrical tubal filling during initial HSG (12/26, 46.1%) compared to controls (2/14, 14.3% $P<0.03$). They also demonstrated significantly more frequently (22/26, 84.6%) elevated TPP than women without disease (2/14, 14.3%. $P<0.004$). Lastly, women with endometriosis also demonstrated significantly higher mean TPP than women with normal pelvises (576±264 versus 450±268 mm of Hg. $P<0.05$). They therefore concluded that asmmetrical tubal filling during initial HSG and elevated TPP during SS are highly suggestive of pelvic endometriosis (Karande et al, 1995).

The same authors next attempted to reduce elevated TPP by transvaginal catheterization procedures using wire-guides (Karande and Gleicher 1996). Such a maneuver

was of interest since it could have potential therapeutic value by increasing PR. In 17 patients (29 tubes) with elevated TPP, a Cope Mandril wire guide (Cook Ob/Gyn, Spencer, Indiana) with a diameter of 0.021 inch and length of 60 cm, was passed through the SS catheter into the tubal lumen and moved in a to and fro motion several times to improve upon tubal patency. TPP was reevaluated before and immediately after tubal catheterization with the wire guide. The TPP (mean ± SD) before wire-guide cannulation (779 ± 241 mm Hg) was reduced (to 474 ± 186 mm Hg) after wire-guide cannulation ($P < 0.0001$) by a mean difference of 305 ± 195 mm Hg (95% C.I. 231 - 379 mm Hg). Wire-guide cannulation was more effective in reducing TPP to normal in patients with mildly elevated TPP (≤ 600m Hg). In those instances, an elevated TPP appears due to a partial obstruction of the tubal lumen, which can often be relieved by the catheterization procedure. In most cases of severely elevated TPP, however, wire guide cannulation did not reduce TPP to normal. In such patients it appears likely that the increased resistance to the injected fluid column is reflective of a decreased tubal compliance, as one would expect with tubal infiltration by active endometriosis or tubal wall fibrosis as a consequence of endometriosis. It is tempting to speculate that balloon dilatation of fallopian tubes may be more effective in these patients than wire guide cannulation since it may lead to a break up of fibrotic fibers. This hypothesis could explain the reported success of transcervical balloon dilatation procedures in achieving pregnancy after severe tubal occlusion (Risquez and Confino, 1993).

Wiedemann et al (1996) reported on their experience with falloposcopy in the management of PTO. They classified PTO as: 1) non-nodular occlusion (complete fibrotic obstruction as a result of an inflammatory process); 2) nodular occlusion (in cases of SIN or endometriosis); 3) pseudo-occlusion (which is descriptive for debris, polyps or hypoplastic tubes). They suggest treatment based on the falloposcopic findings. For non-nodular occlusion they recommend a surgical approach with resection of the diseased segment of the tubes. Patients with nodula and pseudo-occlusion were treated with gonadotropin-releasing hormone analogues. This resulted in subsequent tubal patency, but, a low spontaneous pregnancy rate. They recommend this group of patients be further treated with ovulation induction or some form of assisted reproductive technologies.

The findings of these three approaches are similar although stated differently. The vast majority of patients that require wire guides to treat PTO, subsequently have elevated TPP. These are the patients that on falloposcopy are diagnosed with "non-nodular" occlusion. Then, there are the patients with PTO that is easily treated with SS. These are the patients with "nodular" occlusion (or "pseudo-occlusion") on falloposcopy. And finally there are patients with patent tubes but elevated TPP which may reflect an early stage of non-nodular occlusion.

Use of ultrasound to treat PTO has been reported (Risquez and Confino 1993); it is possible that this approach may subsequently replace the fluoroscopic approach.

We thus have three different approaches for evaluating the proximal tube. Firstly, there is the "GynecoRadiological Procedure" with the measurement of TPP. The equipment for measuring TPP, however, is not freely available commercially. Then, there is Woolcotts' data showing differential PR based on the severity of PTO and the technique required for its treatment. Finally there is the falloposcopic approach. All three approaches strongly suggest that between a "normal" tube and severe PTO there lies a intermediate stage of tubal "disease". These are patients with tubes that may be anatomically patent (or obstructed, but, treatable with plain SS) but functionally compromised. The challenge for today's clinician is to not merely evaluate tubal "patency" but to evaluate tubal disease, and perhaps consider some form of assisted reproduction at an earlier stage in these patients (Gleicher and Karande 1996).

References

1. Confino E, Friberg J, Gleicher N. Am J Obstet Gynecol 59:370-375, 1988.
2. Confino E, Tur-Kaspa I, DeCherney A, Corfman R, Coulam C, Robinson E, JAMA 264:2079-2082, 1990.
3. Diechert U, Schlief R, Van de Sandt M, Juhnke I. Hum Reprod 4:418-424, 1989.
4. Gleicher N, Redding L, Parrilli M, Karande V, Pratt D. Hum Reprod 9:1109-1111. 1994.
5. Gleicher N, Karande V. Hum Reprod 11:1825-1828. 1996.
6. Jones HW, Toner J. N Engl J Med 329:1710-1715. 1993.
7. Karande VC, Pratt DE, Rabin DS, Gleicher N. Fertil Steril 63:1167-1171, 1995.
8. Karande VC, Pratt DE, Rao R, Balin M, Gleicher N. Fertil Steril 64:1070-1073. 1995.
9. Karande V, Gleicher N. Hum Reprod Update 2:5-8. 1996.
10. Novy MJ. Gynecol Obstet pp 67-71. 1995.
11. Osgur K, Yildiz A, Uner M, Erklic M, Trak B, Erman O. Fertil Steril 67:751-755, 1997.
12. Patton PE, Williams TJ, Coulam CB. Fertil steril 47:35-39, 1987.
13. Peters AJ, Coulam CB. Am J Obstet Gynecol 164:1530-1534, 1991.
14. Risquez F, Confino E. Fertil Steril 60:211-226, 1993.
15. Rubin I. JAMA April 10, 1920.
16. Wiedemann R, Montag M, Sterzik K. Hum Reprod 11:1823-1825, 1996.
17. Woolcott R, Petchud A, O'Donnell P, Stanger J. Hum Reprod 10:1423-1426, 10:1995.
18. World Health Organisation. Fertil Steril 46:1101-1107, 1986.
19. Yoder IC, Hall DA. AJR 157:675-683, 1991.