Salpingitis isthmica nodosa: a review of the literature, discussion of clinical significance, and consideration of patient management

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Objectives: To examine and discuss the pathology, diagnosis, incidence, and patient profile of salpingitis isthmica nodosa and to question its natural history, propose management strategies, and identify areas of promising research.

Design: Over 50 studies were reviewed, evaluated, and compared to offer the clinician a foundation on which to generate treatment plans.

Results: Salpingitis isthmica nodosa is diagnosed by the pathological presence of isthmic diverticula and may be suggested by characteristic changes on hysterosalpingogram. Its incidence in healthy, fertile women ranges from 0.6% to 11%, but it is significantly more common in the setting of ectopic pregnancy and infertility. There are no studies, retrospective or prospective, that clearly dictate appropriate therapy.

Conclusion: Given its progressive nature and probable deleterious effects on fertility, we propose that microtubal surgery be the definitive treatment for qualified women who have salpingitis isthmica nodosa.

Key Words: Salpingitis isthmica nodosa, tubal diverticulosis, review, infertility, ectopic pregnancy, microtubal surgery

Salpingitis isthmica nodosa is a well-described pathological lesion of unknown etiology that typically afflicts women in their childbearing years. It seems to be associated with ectopic pregnancy (EP) and infertility. Much of the current literature debates possible etiologies and attempts to further clarify the disease as a pathological and radiologic entity. However, little is known about the true incidence, natural history, or clinical significance of salpingitis isthmica nodosa. Still less is known about appropriate management alternatives. This article seeks to summarize the pertinent characteristics of salpingitis isthmica nodosa and, more importantly, attempts to address the following questions of clinical relevance: Is salpingitis isthmica nodosa progressive? What is the association between salpingitis isthmica nodosa and EP? What is the association between salpingitis isthmica nodosa and infertility? Should patients with salpingitis isthmica nodosa be offered microtubal surgery? How should patients with salpingitis isthmica nodosa and patent tubes be treated? Is there a role for microendoscopic falloposcopy in identifying and treating the lesion?

BACKGROUND

The lesion that we today recognize as salpingitis isthmica nodosa was first described more than a century ago by Chiari (1) when he observed epithelial inclusions in the tubal walls of six of his patients. He believed the disease to be inflammatory in origin and hence proposed a descriptive name:
salpingitis, referring to the suspected etiology; isthmica, identifying the lesion’s prominent location within the fallopian tube; and nodosa, highlighting the gross nodular appearance of the affected tubes. Since its initial discovery, several synonyms for salpingitis isthmica nodosa have been suggested. Some names reflect pathological and radiologic manifestations of the disease (diverticulosis); others reflect possible origins (tubal adenomyosis or endosalpingiosis).

**PATHOLOGY**

Gross pathological examination of tubes with salpingitis isthmica nodosa reveals firm nodular enlargements ranging in diameter from several millimeters to 2 cm. The serosal covering is smooth and intact, and the nodules are gray, yellow, or brown on cut surface (2). The hallmark of this lesion is the presence of isthmic diverticula or outpouchings of tubal epithelium. These projections, which often communicate with the central tubal lumen, “in­vade” the surrounding muscularis and appear to in­cite secondary smooth muscle hypertrophy that re­sults in nodularity. Diverticula typically do not connect with the serosal surface but may be found in close proximity to it. Scarring is usually absent as are inflammatory changes (2–4) (Fig. 1). Kara­sick et al. (5) observe that muscular hypertrophy may occlude canaliculi, causing dilatation of the gland-like diverticular spaces and resulting in in­creased risk for secondary infection. Punnonen and Söderström (6) studied the tubal histology of 15

infertile patients who had been diagnosed with sal­pingitis isthmica nodosa. Specimens from two women were examined via electron microscopy. The diverticula were found to be lined by normal tubal epithelium. Each cell had a central nucleus, prominent nucleoli, abundant rough endoplasmic reticulum, secretory granules, and luminal micro­villi. Also, no endometrial stroma was detected, suggesting that salpingitis isthmica nodosa is not a form of tubal endometriosis.

**DIAGNOSIS**

The diagnosis of salpingitis isthmica nodosa can be confirmed only by inspection of tubal histology. Classic findings are as described above. The diagnosis may be suspected by performing a hysterosalpingogram (HSG). The lesion was first demonstrated by HSG in 1951. (See McShane and Yeh [3]). In the last several decades, HSG-based diagnosis of salpingitis isthmica nodosa has been the premise for many studies that explore the incidence and clinical significance of the disease. Typically, 2-mm accumulations of contrast medium are ob­served in close approximation to the isthmic por­tion of the oviducts bilaterally (7) (Figs. 2 and 3).
Tubal obstruction and hydrosalpinx are common associated findings (5). Diseases other than salpingitis isthmica nodosa must be considered in the differential diagnosis of such tubal changes. Endometriosis typically produces a honeycomb appearance in the proximal tubal segment, manifests thicker-walled diverticula, and is associated with patent oviducts and a normal uterus (5). It can be differentiated from salpingitis isthmica nodosa by the presence of endometrial glands and stroma. Tuberculous salpingitis is associated with calcified pelvic lymph nodes, tubes, or ovaries, and shows endometrial changes. Ragged tubal borders, irregular tubal caliber, and multiple strictures are typically seen on HSG (8).

ETIOLOGY

The debate goes on as to the actual etiology of salpingitis isthmica nodosa. As stated above, Chiari (1) assumed an inflammatory origin when he named the lesion salpingitis. Since then, both congenital and a variety of acquired etiologies have been proposed. Creasy et al. (9) cite the first advancement of the congenital theory in 1896. It was believed that typical salpingitis isthmica nodosa findings were due to wolffian rests, because embryologically, the tubal isthmus is the site where the müllerian and wolffian ducts cross. Kuge (10), in 1927, also held that salpingitis isthmica nodosa represented mesonephric rests (10). Naisby's (11) recent article lends support to these observations. In 1987, he revealed an association of two mesonephric abnormalities in the same patient, a finding previously unreported. Naisby presents a 29-year-old woman with primary infertility and no previous history of pelvic inflammatory disease (PID). Her traditional infertility work-up, including semen analysis, was normal. Hysterosalpingogram revealed bilateral diverticula and a Gartner's duct (a remnant of the caudal end of the mesonephric duct or female equivalent of the vas deferens) extending to the left of the vagina with cervical communication. Aside from this unique discovery, all other recent studies have refuted the congenital hypothesis. Neumann (12) found no malformation or anlage when reviewing the embryologic development of the fallopian tubes in corroboration with the development of the müllerian ducts. He also studied 45 pairs of oviducts from full-term neonates and failed to find any abnormalities (12). Benjamin and Beaver (13) reviewed 51 cases of histologically-proven salpingitis isthmica nodosa and noted no congenital malformations. Likewise, Persaud (14) searched for congenital lesions from the autopsy specimens of 50 infants and children but found none. Only once has salpingitis isthmica nodosa been reported in a preadolescent girl (15). In summary, current literature fails to support a congenital etiology.

The more popular and probable etiologies are those that are acquired. Two researchers believed salpingitis isthmica nodosa to be neoplastic (16); others have seen pathology similar to human salpingitis isthmica nodosa in diethylstilbestrol-exposed neonatal rats, and thus have demonstrated a useful model for the study of hormonally induced oviductal lesions (17). Honoré (18) proposed a pathogenesis similar to that of colonic diverticulosis. He believed salpingitis isthmica nodosa to be a late manifestation of chronic tubal spasm because it almost exclusively involves "... the sphincter-like isthmus, which has a powerful muscle coat and dense adrenergic motor innervation . . ." (18). This tubal diverticulosis is thought to result from rising intramural pressure because of some disease stimulus and secondary weakness in the wall at the site of vascular bundles (19).

The most obvious (but highly disputed and as yet unproven) etiological theory is infectious. Several retrospective studies (6, 7, 9, 13, 14, 20, 21) have demonstrated an association between salpingitis isthmica nodosa and other infectious lesions or historic, radiologic, or surgical evidence of previous inflammation. None have been able to show a cause-and-effect relationship. One gynecological textbook reports inflammation to be associated with salpingitis isthmica nodosa histology in 75%
to 80% of cases (3). Punnonen and Söderström (6) found a slight inflammatory cell infiltrate of small lymphocytes in all of the 15 salpingitis isthmica nodosa patients that they examined by pathological inspection. They (6) proposed that a chronic infection, perhaps chlamydia, might be the inciting event in a colonic-like diverticulosis theory akin to Honore’s (18). Dubuisson et al. (20) studied 148 tubal pregnancies histologically, and of the 12 isthmus, perhaps chlamydia, might be the inciting event in a colonic-like diverticulosis theory akin to Honore’s (18). Dubuisson et al. (20) studied 148 tubal pregnancies histologically, and of the 12 isthmic EPs, 36.4% revealed salpingitis isthmica nodosa. In all but one case, when the isthmus showed pathology, the ampulla showed chronic salpingitis. Likewise, Green and Kott (21) found salpingitis isthmica nodosa to be associated with postinflammatory changes in 89% of their ectopic study group. Creasy et al. (9) found that 89% (62 of 70) of the salpingitis isthmica nodosa patients in their retrospective review of 1,194 HSGs had evidence of previous inflammation by either history, HSG, or surgery. There remains a major problem with this theory: if infection causes the lesions typical of salpingitis isthmica nodosa, why is salpingitis isthmica nodosa so relatively uncommon compared with PID and chronic salpingitis? Also why is there not evidence of tubal scarring in salpingitis isthmica nodosa patients (9)? Perhaps salpingitis isthmica nodosa predisposes to subsequent infection. It is difficult to determine which condition actually comes first. To do this, a prospective study of fertile women without a history of PID, PID risk factors, or evidence of inflammation is needed. The women would necessarily be followed with serial HSGs, and any evidence of infectious salpingitis that then arose could be correlated with HSG findings. This, however, would be difficult to accomplish because it would subject the pelvis of normal fertile women to unnecessary radiation (9). Thus, a cause-and-effect relationship may never be confirmed. The final acquired theory of origin refers to salpingitis isthmica nodosa as an adenomysis-like process. Uterine adenomyosis results from endometrial invasion of the myometrium. Likewise, salpingitis isthmica nodosa demonstrates invasion of the tubal musculature by tubal epithelium with subsequent muscular hypertrophy. Interestingly, unilateral salpingitis isthmica nodosa is often accompanied by ipsilateral uterine adenomyosis (4).

INCIDENCE

The reported incidence of salpingitis isthmica nodosa in a healthy, fertile, control group ranges from 0.6% to 11%, depending on study design. Some researchers suggest a racial predilection within this population (21, 22), whereas others show an interesting association between race and the incidence of salpingitis isthmica nodosa. For instance, Persaud (14) reports an incidence of 11%; all patients in his study were from Jamaica. Honore (18) reports an incidence of 0.6%; all his patients were white. Niles and Clark (23), however, report the same incidence of 0.6%, and all their patients were black. Larger, controlled investigations are needed. The incidence of salpingitis isthmica nodosa among women with EPs and primary infertility appears to be significantly greater and will be discussed later. Table 1 summarizes the authors who have addressed the question of incidence and their findings. In each study, the diagnosis of salpingitis isthmica nodosa was confirmed by pathological examination.

PATIENT PROFILE

Salpingitis isthmica nodosa is usually discovered during the investigation of primary infertility, often by HSG, laparoscopy, laparotomy, or by pathological examination. Except for the associations with infertility and EP, the lesion is asymptomatic. There is no typical pattern of gynecological problems that is associated with this diagnosis (25). It afflicts women ages 25 to 50, with a mean age of initial presentation of 30 to 35 years (3). Bilateral salpingitis isthmica nodosa is common. Benjamin and Beaver (13) reported bilateral lesions in 18 of 51 patients (35%), whereas Karasick et al. (5) documented 80% bilaterality. Creasy et al. (9) reviewed 1,194 HSGs over 5 years and discovered salpingitis isthmica nodosa to be bilateral in 55.5% (25 of 45). Several studies (5, 9, 13, 21, 26) suggest that blacks are more commonly affected than whites in the setting of EP and infertility. Freakley et al. (26) examined 918 HSGs with roughly a 1:1 race distribution and found salpingitis isthmica nodosa to be 9 times more common in blacks. Creasy et al. (9), Karasick et al. (5), and Benjamin and Beaver (13) found between a 2 and 3.5 times greater incidence in blacks. Only one study contradicts these findings. Green and Kott (21) found salpingitis isthmica nodosa to be more common in whites in a 3:2 ratio. Finally, a history of PID is often elicited in the salpingitis isthmica nodosa patient (5, 6, 21), but the cause-and-effect relationship between the two entities has yet to be decided.
Table 1 The Incidence of Salpingitis Isthmica Nodosa in a Healthy, Fertile Population

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Total no. of patients</th>
<th>Specimen source</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Niles and Clark (23)</td>
<td>1969</td>
<td>161</td>
<td>Postpartum tubal ligation</td>
<td>0.6</td>
</tr>
<tr>
<td>Persaud (14)</td>
<td>1970</td>
<td>100</td>
<td>Postpartum tubal ligation</td>
<td>11.0</td>
</tr>
<tr>
<td>Honoré (18)</td>
<td>1978</td>
<td>167</td>
<td>Elective sterilization*</td>
<td>0.6</td>
</tr>
<tr>
<td>Majmudar et al. (22)</td>
<td>1983</td>
<td>100</td>
<td>Autopsy and surgical pathology</td>
<td>5.0</td>
</tr>
<tr>
<td>Homm et al. (24)</td>
<td>1987</td>
<td>39</td>
<td>Tubal ampullae of ectopic patients</td>
<td>10.2</td>
</tr>
<tr>
<td>Green and Kott (21)</td>
<td>1989</td>
<td>100</td>
<td>Tubal ligation or hysterectomy</td>
<td>5.0</td>
</tr>
</tbody>
</table>

* A minority (21) of specimens were taken from women with unilateral hydrosalpinx, pyosalpinx, or tubo-ovarian abscess.

NATURAL HISTORY

Competence in treating a disease depends on knowledge of its natural history. Unfortunately, little is known about the behavior of salpingitis isthmica nodosa over time. McComb and Rowe (27) attempted to document the progressive nature of salpingitis isthmica nodosa in six infertile patients. All six were found to have salpingitis isthmica nodosa on initial HSG. Their length of infertility varied between 2 and 8 years, and the age at detection ranged from 24 to 31 years. These women were followed by sequential HSGs. All six demonstrated progression, defined as either an increase in size of the lesion or newly acquired tubal obstruction. The time of progression ranged from 12 to 60 months. Because of these findings, McComb and Rowe observe: “One cannot exclude evolution of salpingitis isthmica nodosa with an HSG study done more than 12 months previously (27).” Although this is not a large, prospective study with controls, it serves an important purpose in defining disease behavior. Indeed, it does suggest that salpingitis isthmica nodosa will progress if left untreated. Whether or not all lesions progress, over what time period, and the potential mounting threats to fertility are all issues that await further study.

ASSOCIATION WITH EP

In the last decade, research has been focusing on the association between salpingitis isthmica nodosa and tubal EPs. At least 10 studies over the past quarter-century have supported this claim. Table 2 lists these studies, the incidence of salpingitis isthmica nodosa in patients with EPs, and the incidence of the disease in controls where applicable. All of these reports support the same basic conclusion: women who have EPs are more likely to be found to have salpingitis isthmica nodosa than controls. The strength of the association varies. Honoré (18) found the association to be minimal (2.86% incidence of salpingitis isthmica nodosa in the ectopic population, compared with 0.6% in controls), whereas Majmudar et al. (22) reported evidence of salpingitis isthmica nodosa in 57% of his ectopic population, compared with 5% in controls. The study of Homm et al. (24) is especially interesting. He and his colleagues reviewed the charts of 285 patients with EPs and excluded all patients with previous tubal manipulation. Of 255 total, 39 were isthmic in location. These isthmic ectopics were compared with ampullary ectopics to determine the incidence of salpingitis isthmica nodosa shown in Table 2. Thus, this study was location-specific and probably shows a better correlation between the disease and the outcome. When considering this association, however, other variables come into play. For instance, questions of race, size of study populations, design of study, and presence or absence of control group cloud the issue. Also, some researchers cannot be as selective in choosing their study population and hence must include patients with another pathology such as PID. Ideally, a project must be designed to include women with evidence of salpingitis isthmica nodosa by HSG and no other pelvic pathology. These women would then have to be followed, and their incidence of EP determined as compared with an age- and race-matched control group. Unfortunately, this would not be extremely practical. As it stands now, salpingitis isthmica nodosa has not been proven to cause EP. It does, however, make intuitive sense that salpingitis isthmica nodosa would alter the tubal architecture enough so as to inhibit the transport of either the sperm, ovum, or zygote. Its diverticular character might serve as a nidus for an ectopic implantation. Based on the above evidence, the association between salpingitis isthmica nodosa and ectopic gestations definitely exists. It seems
Table 2 The Incidence of Salpingitis Isthmica Nodosa in Patients With EPs

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Diagnosis</th>
<th>Total no. of ectopic patients</th>
<th>Incidence in ectopics</th>
<th>Incidence in controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wroth and Broders (28)</td>
<td>1942</td>
<td>Histology</td>
<td>100</td>
<td>13.0</td>
<td>NA*</td>
</tr>
<tr>
<td>Niles and Clark (23)</td>
<td>1969</td>
<td>Histology</td>
<td>436</td>
<td>6.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Persaud (14)</td>
<td>1970</td>
<td>Histology</td>
<td>100</td>
<td>49.0</td>
<td>11.0</td>
</tr>
<tr>
<td>Honoré (18)</td>
<td>1978</td>
<td>Histology</td>
<td>35</td>
<td>2.9</td>
<td>0.6</td>
</tr>
<tr>
<td>Gonzalez and Waxman (29)</td>
<td>1981</td>
<td>Histology</td>
<td>394</td>
<td>9.9</td>
<td>NA</td>
</tr>
<tr>
<td>Majmudar et al. (22)</td>
<td>1983</td>
<td>Histology</td>
<td>100*</td>
<td>27.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Majmudar et al. (22)</td>
<td>1983</td>
<td>Histology</td>
<td>100‡</td>
<td>57.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Creasy et al. (9)</td>
<td>1985</td>
<td>HSG</td>
<td>45</td>
<td>9.4</td>
<td>NA</td>
</tr>
<tr>
<td>Dubuisson et al. (20)</td>
<td>1986</td>
<td>Histology</td>
<td>132</td>
<td>32.6</td>
<td>NA</td>
</tr>
<tr>
<td>Homm et al. (24)</td>
<td>1987</td>
<td>Histology</td>
<td>37§</td>
<td>45.9</td>
<td>10.2</td>
</tr>
<tr>
<td>Green and Kott (21)</td>
<td>1989</td>
<td>Histology</td>
<td>129</td>
<td>43.0</td>
<td>5.0</td>
</tr>
</tbody>
</table>

* NA, not applicable.
† Majmudar evaluated 100 patients with EPs retrospectively and found a 27% incidence of salpingitis isthmica nodosa.
‡ In the same study as above, Majmudar (22) evaluated 100 patients with EPs prospectively and found a 57% incidence of salpingitis Isthmica nodosa.
§ All were isthmic EPs.

likely that salpingitis isthmica nodosa could indeed be an underlying cause of tubal pregnancies as well.

ASSOCIATION WITH INFERTILITY

As important as the association between salpingitis isthmica nodosa and tubal pregnancy is the association between the disease and infertility. Unfortunately, given that infertility is most commonly multifactorial in origin, this relationship is even harder to establish than the one just discussed. Because of the many confounding variables, few investigators have tackled the question of salpingitis isthmica nodosa and its role in causing infertility. Indirect evidence is more prevalent. In 1985, Fortier and Haney (25) inspected the isthmic tubal segments of 42 infertile women who underwent surgical procedures to correct proximal tubal obstruction. All patients had been infertile for at least 2 years and had undergone a standard infertility work-up, including laparoscopy. Their mean duration of infertility was 3.7 years. Of the 42 patients studied, 10 (24%) were found to have microscopic evidence of salpingitis isthmica nodosa. In Honoré's (18) study to determine the overall incidence of salpingitis isthmica nodosa in various populations, he examined the tubal specimens of 6 infertile women undergoing tubal surgery. Although this group was small and biased toward surgery, Honoré (18) demonstrated salpingitis isthmica nodosa in 3 of the 6 (50%). Two patients had bilateral disease. Karasick et al. (5) found a history of either primary or secondary infertility together with radiologic evidence of salpingitis isthmica nodosa in 13 of 150 (8.7%) consecutive patients undergoing HSG. Finally, Creasy et al. (9) discovered a history of primary infertility along with salpingitis isthmica nodosa in 17 of 45 (37.8%) women having HSGs for various reasons over a 5-year period. Thus, much as with EPs, salpingitis isthmica nodosa has not been proven to directly cause infertility simply because it is so difficult to eliminate the various other states contributing to the final common pathway of infertility. The association, however, has been documented and is compelling.

MANAGEMENT

In advising treatment for salpingitis isthmica nodosa, one must assimilate all of the factors discussed above and then formulate a plan catered to the individual patient. Unfortunately, there is a paucity of literature to assist the physician in making management decisions. It is reasonable to argue that prevention is the only definitive treatment. If one believes that salpingitis isthmica nodosa is wholly or partially caused by infection or inflammation, one would attempt to educate the patient regarding risk factors for PID, the need for yearly gynecological exams with appropriate cultures, and prompt treatment of infections. Hopefully, this is already the standard of care for all patients. Assuming a woman is found to have radiologic evidence of salpingitis isthmica nodosa on HSG in the face of a normal infertility work-up (medical and gynecological history and physical assessment of ovulatory
function, semen analysis, and postcoital test), what is her best treatment option? Overall, microtubal surgery is a favorable procedure. Gomel (30) treated 48 patients who had tubocornual obstruction with microsurgical tubocornual anastomosis, an alternative to the older method of tubouterine implantation. One year after the procedure, 27 of 48 (56.2%) women reported a term intrauterine pregnancy (IUP), whereas only 3 of 48 (6.2%) reported an EP. Success rates for sterilization reversals involving healthy isthmic tissue are much greater. McComb confirmed Gomel’s results in a similar study (31). Of the 26 women undergoing unilateral or bilateral tubocornual anastomosis, 15 (57.7%) achieved viable IUPs with a conception interval of 3 to 29 months. Additionally, McComb (31) examined the tubal histology of these patients and found 5 of the 26 patients to have salpingitis isthmica nodosa. Three of the 5 went on to have viable IUPs, whereas 2 did not. Although the numbers are small (not statistically significant) and the chance of a term IUP is 60% at best, it is important to note that salpingitis isthmica nodosa pathology was not associated with a worse outcome after tubocornual anastomosis when compared with other lesions such as fibrosis or chronic salpingitis. A third study attempts to address success rates of tubocornual anastomosis based on the underlying pathology and preoperative HSG. Donnez and Casanas-Roux (32) evaluated 82 women with bilateral cornual blockage (164 pathological tubes) and found 6 to have evidence of diverticular lesions suggestive of salpingitis isthmica nodosa and occlusion on HSG. Only 1 of 6 (16%) women went on to have a term IUP, compared with an overall 44% rate for all 82 women with varying tubal pathology. Although these data seem to speak against microtubal surgery for salpingitis isthmica nodosa patients, the pathological evidence is not helpful because a diagnosis of endometriosis was made in each case when the HSG demonstrated diverticular lesions. As previously stated, salpingitis isthmica nodosa and tubal endometriosis must be distinguished as two separate entities based on the presence or absence of endometrial stroma. Because the diagnosis of salpingitis isthmica nodosa was not confirmed in this study, it provides little help in answering our original question.

Further studies are needed before we can confidently advise our patients with salpingitis isthmica nodosa that microtubal surgery is their cure for infertility. However, certain patients are likely to benefit from such a procedure with either an improved chance for fertility or a decreased chance of ectopic gestations. The first step in treating a patient with salpingitis isthmica nodosa is to determine whether or not her tubes are occluded. This may be accomplished by HSG or may require laparoscopy and chromopertubation. All of the studies described above deal with patients who already have occluded tubes. Certainly this procedure should offer the patient some hope for improvement in the face of otherwise occluded oviducts. If, however, the patient also has extensive distal tubal disease (33) that could not easily be corrected at the time of surgery, IVF might be the most reasonable option. If the patient’s tubes are patent and the disease is suspected based on HSG, one should consider other factors before offering microtubal surgery. First, has the couple had an infertility work-up to this point that has failed to identify other correctable causes? Second, has the patient’s infertility been prolonged, for example, >2 to 3 years? Third, can the lesion be resected by removing 1.5 cm. or less of tubal length (32)? Fourth, is the patient a good surgical candidate? Finally, is microtubal surgery a cost-effective alternative for the individual patient? If the answer to all of these questions is yes, microtubal surgery should be offered to the patient. How does each of these questions affect the decision to pursue surgery? The first is the most influential. For microtubal surgery to offer any hope of improved fertility, there must be no other infertility factors identified or left untreated. Clearly, if a woman is not ovulating or a man’s semen analysis is less than adequate, surgery would be an unnecessary risk, unless these factors could be remedied. Question two allows for the possibility that salpingitis isthmica nodosa could be present and pregnancy still occur, especially if the lesion is in the early stages. There is no evidence to suggest that salpingitis isthmica nodosa is self-limited or retreats. Thus, based on McComb and Rowe’s (27) article and lack of findings to the contrary, one must assume that the natural history of salpingitis isthmica nodosa is to progress and decrease fertility proportionally. Therefore, the longer the duration of infertility, the less likely the couple will conceive despite the disease. Question three is also quite influential. Two separate studies (32, 34) have proven that isthmic residual length is a prognostic factor, significantly influencing pregnancy rates after surgery. Although this is not an essential prerequisite to microtubal reanastamosis, it clearly improves the chance of obtaining a viable pregnancy. Questions four and five are self-
explanatory. A physician and his patient must balance the risk of surgery (including complicating medical factors) with the desire and reasonable chance for fertility. Funds must also be available. Based on the above reasoning, an affirmative answer to question one is essential before surgery should be offered. Not so for questions two and three. An affirmative response to either or both would proportionally increase the couple’s chance of obtaining an IUP, but negative responses should not exclude surgery as an option. Given the compelling association between salpingitis isthmica nodosa and both EP and infertility and the evidence that salpingitis isthmica nodosa lesions are likely progressive in nature, segmental resection and reanastomosis are the best alternatives one can offer the couple desiring a term IUP.

NEW DEVELOPMENTS

A new method of identifying and perhaps treating tubal disease is gaining favor: microendoscopic falloposcopy, along with tubal recanalization via selective ostal salpingography and transcervical balloon tuboplasty. In the future, this technique might serve as the noninvasive method of choice for identifying salpingitis isthmica nodosa-afflicted tubes and thus help to determine who might be a candidate for microtubal surgery. Proximal tubal obstruction is believed to be responsible for 20% of infertility because of tubal disease (35). Thurmond and Rosch (36) successfully recanlized at least one of two tubes in 86 of 100 patients with proximal obstruction. Based on their data, they suggest that women have at least a 30% chance of achieving an IUP soon after fluoroscopic oviduct recanalization. Confino et al. (35) achieved similar results. They were able to recanlize (via selective ostal salpingography and tuboplasty) at least one of two tubes in 71 of 77 (92%) patients, 17 of whom went on to have IUPs and healthy infants. Of the 25 who had not conceived in 6 months, 17 still demonstrated tubal patency on HSG. Although intramural plugging, submucosal leiomyomas, tubal spasm, or tubal debris might be responsible for the high success rates in these two studies, progress is being made without having to resort to surgery. Most recently, Letterie and Sakas (37) examined 27 cornual/isthmic tubal segments from 15 patients who failed fluoroscopically guided tubal recanalization and thus went on to microtubal surgery to correct a proximal obstruction. Forty-two percent revealed histologic evidence of salpingitis isthmica nodosa, suggesting two things. First, salpingitis isthmica nodosa is a relatively common cause of proximal tubal obstruction. Second, failed recanalization might lead one to doubt a functional cause of obstruction and suspect an intrinsic cause amenable to surgery (e.g., salpingitis isthmica nodosa). As yet, there are no investigations that specifically address the efficacy of these methods for women with salpingitis isthmica nodosa, and it is unclear whether or not dilating such a lesion would have any affect on future fertility.

SUMMARY

In conclusion, salpingitis isthmica nodosa is a well-characterized lesion of the proximal fallopian tube. Its pathologic hallmark is the presence of isthmic diverticula, outpouchings of tubal epithelium, with secondary hypertrophy of the surrounding muscularis. Although salpingitis isthmica nodosa is diagnosed based on these typical histologic findings, the lesion may be suspected by characteristic changes on HSG. Its etiology is likely acquired. Salpingitis isthmica nodosa affects women between the ages of 25 and 50, is often bilateral, and may be more common in blacks. Its incidence in a healthy, fertile population ranges from 0.6% to 11%. The disease is strongly associated with both EP and infertility. There are no studies that dictate appropriate treatment for women with salpingitis isthmica nodosa, but segmental resection and microtubal reanastomosis are likely the best options for qualified patients with tubal obstruction or patent oviducts. In the future, microendoscopic falloposcopy with selective ostal salpingography and transcervical balloon tuboplasty may aid in identifying salpingitis isthmica nodosa, determining surgical candidacy, and perhaps treating or temporizing the disease.

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