TUBAL FACTORS IN STERILITY

(A laparoscopic study of 697 cases of sterility)

by

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Introduction

The extent to which tubal block is responsible for infertility depends on the incidence of genital tuberculosis, venereal disease, puerperal sepsis and septic abortions in that community. Although efforts are made to restore that fertility by procedures such as hydrotubation in cases of mild intratubal adhesions and tuboplasty of various kinds in cases of tubal block of different varieties the success of these procedures is limited. The tubo-ovarian infections, puerperal and postabortal sepsis, therefore cause permanent damage and sterility in the majority of the cases.

Six hundred and ninety-seven cases of sterility have been examined clinically and laparoscopically. The incidence and type of tubal damage in primary and secondary sterility are assessed.

Material and Methods

Out of 697 cases of sterility, 491 had primary sterility and 206 had secondary sterility. The patients were chosen for laparoscopy after clinical examination and preliminary investigations. Only 1/4 of these patients had hysterosalpingography prior to laparoscopy. Diagnostic laparoscopy was carried out with all the necessary precautions and the procedure is not described here. The uterus, tubes, ovaries, pouch of Douglas and the pelvis were visualised. Chrome-pertubation was carried out in all cases except 40 cases with large tubo-ovarian masses or obvious tuberculosis with caseation or when visualisation of tubes and pouch of Douglas were difficult due to multiple adhesions. A second puncture to move the omentum or break the flimsy adhesions was carried out only in 4 cases. Previous laparotomy scars were present in 22 cases. adhesions made the procedure difficult in 2 cases. The vision was limited due to multiple adhesions in 4 other cases.

Results

The findings of laparoscopy were noted and the salient features were as follows:

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26.4% of these infertility patients had chronic pelvic inflammatory disease with tubal damage responsible for infertility.

17.5% had complete bilateral blocked tubes.

About 3% had partial block with delayed spill of the dye.

Table II indicates the incidence of tuboovarian masses, hydrosalpinx, thickened rigid tubes and peritubal adhesions as well as adhesions in pouch of Douglas in both primary and secondary sterility cases.

The overall incidence of pelvic tuberculosis was high in the patients with primary sterility—12.8% compared to cases of secondary sterility—4.3%. The ovarall incidence of pelvic tuberculosis was 10% (Table III).

Cases with frank tubercles and caseation were diagnosed as pelvic tuberculosis. It is difficult to easily distinguish pelvic tuberculosis in cases of hydrosalpinx or tube-ovarian masses or thickening of tubes from the non-specific chronic inflammatory disease, as the latter can also have a similar picture. The tuberculous tubes are more rigid, seem to be fixed to the uterus and their shape does not alter by moving the uterus back and

TABLE I Abnormal Findings at Laparotomy

| | Total No. of sterility cases | Primary | Secondary |
|--|---------------------------------|----------------------------|--------------------------|
| | 697 | 491 | 206 |
| abnormal findings of tubes clocked tubes (Thickened tubes T.O. | 184 (26.40%) | 130 (26.48%) | 54 (26.21%) |
| Masses, Hydrosalpinx, etc. | 122 (17.50%) 22 (3.16%) | 87 (17.72%) 14 (2.85%) | 35 (16.99%) 8 (3.88%) |

TABLE II
Nature of Tubal Block

| | Total No. of Primary sterility cases | | Secondary | |
|---|--------------------------------------|------------|-------------|--|
| | 697 | 491 | 206 | |
| Peritubal adhesions and adhesions in D.P. | 84 (12.05%) | 48 (9.78%) | 36 (17.48%) | |
| Cubo Ovarian masses | 42 (6.03%) | 30 (6.11%) | 12 (5.83%) | |
| Iydrosalpinx | 34 (4.88%) | 22 (4.48%) | 12 (5.83%) | |
| Blocked thickened tubes | 46 (6.60%) | 45 (9.16%) | 11 (5.33%) | |

A case with more than one pathology would be included in more than one column.

TABLE III
Incidence of Pelvic Tuberculosis

| | Total No. of sterility cases | Primary | Secondary |
|---------------------|---------------------------------|-------------|-----------|
| | 697 | 491 | 206 |
| Pelvic Tuberculosis | 72 (10.33%) | 63 (12.83%) | 9 (4.3%) |

forth. 'Blue uterus" corroborates, but does not clinch the diagnosis of genital tuberculosis unless other findings are associated. Tuberculous atrophy of the endometrium could be the commonest but not the only cause of intravasation. Although a conclusive diagnosis can be made only from a positive culture or histology of endometrium, laparoscopic diagnosis is likely to be correct in the majority of the cases if the collective data of history, clinical findings, other investigations and laparoscopic findings are put together. None of these cases had a laparoscopic tubal biopsy for histopathology for confirming the diagnosis of tuberculosis. Laparoscopy was, therefore, extremely useful to assess the extent to which tubal factors were responsible for infertility.

Discussion

In this series, tubal factor is responsible for 26% of cases of sterility. Kistner and Behrman find tubal factor to be responsible for 30-35% of cases with infertility.

Undoubtedly pelvic inflammatory disease is the largest single factor to cause tubal infertility. Though infantile tubes and congenitally long tortuous tubes are other mechanical causes of sterility, there was no case with the above diagnosis in our series. Although gonococal, post-abortal, puerperal sepsis as well as some iatrogenic factors as I.U.C.D., Rubins test, etc. are supposed to be etiologic factors in the nontuberculous group of pelvic inflammatory disease, even laparotomy and histopathology can rarely pin point the exact etiology. Eighty

TABLE IV

| | Total cases | Primary | Secondary |
|--|-------------|----------|-----------|
| Sterility | 697 | 491 | 206 |
| Tubal pathology as per scopic findings | 184 | 130 | 54 |
| A STATE OF THE PARTY OF THE PAR | (26.40%) | (26.48%) | (26.21%) |
| Clinically abnormal findings, e.g. T.O. | 58/697 | 24/491 | 34/206 |
| masses, thickening, etc. | (8.32%) | (4.89%) | (16.50%) |
| Clinically detected abnormal cases out of | 58/184 | 24/130 | 34/54 |
| laparoscopically detected abnormal | (31.52%) | (18.46%) | (62.96%) |
| cases | | | |

Table IV shows that only 8% of these sterility cases had abnormal clinical findings, but 26% of the cases had tubal pathology as diagnosed by laparoscopy. Therefore, only 31% of the cases with pelvic pathology would have been diagnosed with clinical examination alone. Moreover, the kind of pathology could not have been clarified. It is also seen that the incidence of abnormal clinical findings in primary sterility is much less as compared to secondary sterility as clinically silent tuberculosis is commoner in primary sterility.

of our patients had undergone an operative procedure such as dilatation and curettage, induction of abortion, Rubins test, hysterosalpingography or laparotomy for operations such as ovarian cyst removal, wedge resection of ovaries, cervicopexy, ventral sspension, appendicectomy, etc. One can easily guess to what extent these procedures could be responsible for introduction of sepsis and causing tubal pathology. Therefore, prophylaxis and curative therapy both have a major role to play in pelvic inflammatory disease.

Prompt diagnosis and management of genital tuberculosis is very important. The incidence of this pathology varies throughout the world. Schaefer (1976) states that 5-10% of cases attending infertility clinics have genital tuberculosis. In our series the incidence is 10.33%. In India the overall incidence of tuberculosis is 0.76% to 1.1%. Unfortunately despite early diagnosis and therapy the incidence of pregnancy is practically nil (0.31%).

Endoscopy plays a unique role in detecting pelvic inflammatory disease. Laparoscopy is useful not only to diagnose but also to prognosticate and also to observe the results of treatment. A tuboplasty should always be preceded by a laparoscopy and can also be followed up by one. This procedure has even been shown to be helpful in cases of acute pelvic inflammatory disease. Mardh et al (1977) collected material for culture and studied the microbiology of pelvic infection in the acute phase. The same workers have recently cultured Chlamydia Trachomitis from cases with acute pelvic infections indicating a new aspect in the etiology of acute pelvic inflammatory disease.

Although Culdoscopy is helpful in the visualisation of the pelvis, it is more helpful in the detection of endometriosis which is often present on the under surface of the ovaries. Laparoscopy is preferable when chronic pelvic infection especially tuberculosis is suspected.

Hysterosalpingography can diagnose blocked tubes as well as intra-uterine leiomyomas and intravasation of dye in tuberculosis. Peritubal adhesions are often missed on performing this procedure. Moghissi and Sim (1975) conclude that endoscopy and hysterosalpingography are supplementary procedures.

Keirse and Vandervellen (1973) use hysterosalpingography in patients with normal findings, clinically, because they believe that hysterosalpingography is less imposing, gives valuable information about the uterus and tubes and may be of therapeutic value. Sheth and Krishna (personal communication) studied 100 cases of infertility with laparoscopy and hysterosalpingography and found laparoscopy more informative. In 17 out of 48 cases (35.4%) one or both tubes were found to be patent on laparoscopy though detected to be blocked radiologically. Moreover, in 58 patients laparoscopy gave additional information regarding the tubal pathology, e.g. hydrosalpinx or pelvic tuberculosis.

Treatment of tubal obstruction has limited success. Hydrotubation in suitable cases and tuboplasty in selected cases have success varying from 10-50%. Campos da Paz found that 62.7% patients benefit from prolonged hydrotubation in achieving patency but succeed in achieving pregnancy in only 25.5% of cases. This shows that demonstration of tubal patency does not ensure tubal functional competence. The complex functions such as ovum pick-up, sperm transport, early blastocyst development and its transport to the uterine cavity may imply some biochemical and ultrastructural functions which are as yet incompletely understood.

In our study, only 5% of the patients with tubal pathology were found to be suitable for tuboplasty. The follow-up of these patients and the results of tuboplasty are not presented here. As we have a considerable number of patients with genital tuberculosis, we have only a limited number suitable for tuboplasty. Encouraging results following tuboplasty have been reported by various workers. Umezaki et al (1974) found a pregnancy

rate of 39.4%. Speroff (1970) following 40 cases with pelvic inflammatory disease who had undergone tuboplasty detected 4 term pregnancies, 3 ectopics and 1 abortion. Lamb and Moscovitz (1972) had 27 pregnancies from 100 cases of tuboplasty. Only 19 delivered viable infants, 3 ended as ectopics and 5 had first trimester abortion. Loss of peristallic function and cilia even in a small hydrosalpinx, undiagnosed endometriosis, possibility of tuberculous etiology and recurrence or formation of peritubal adhesions after the operation are the important causes of failure.

The dismal results of tuboplasty prove the point that "prevention is better than cure". This paper stresses the importance of prevention of pelvic infections and aggressive management of acute pelvic infection.

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