AETIOLOGY OF ECTOPIC TUBAL GESTATION-AN ANALYSIS

by

R. NARAYANAN,* M.D., D.G.O. Sr. T. Lillian,** M.B.,B.S., M.R.C.O.G., D.A.

and

SHIRLEY LIAO, *** M.B., B.S.

Analysis

Ectopic tubal pregnancy still remains potentially the most critical gynaecological emergency. It is not merely an isolated surgical episode in a woman's life, but has profound implications on her future reproductive performance. She will have only 1 in 3 chances of ever having a live child again and she confronts a 1 in 20 risk of another ectopic gestation according to Grant (1962).

An objective analysis of the aetiological factors could enable one to realistically assess the present trend in the incidence of this condition, particularly in relation to liberalization of abortion and the wide use of contraception. It is possible to work towards precautionary measures which would curtail the risk of this dreaded complication.

Material and Methods

Included in this analysis are 65 consecutive cases of ectopic tubal pregnancy treated at St. Martha's Hospital, Bangalore between August 1977 and April 1981. Full term deliveries during this period were 9159.

*Associate Professor.

*** Senior House Officer.

Department of Obstetrics & Gynaecology, St. John's Medical College & St. Martha's Hospital. Bangalore 560 034.

Accepted for publication on 1-10-1981.

1. Incidence: The incidence of tubal pregnancy varies widely between 1 in 85 to 1 in 378.5 deliveries even within our own country as has been pointed out by Ghose and Ghose (1968) Oumachigui (1976) Mitra (1975) and Rajan and Nair (1976). Our incidence works out to 1 in 140.9 births.

2. Age incidence: The maternal age in this series ranged from 13 to 40 years. 56.9% of cases belonged to the third decade of life. Similar age group incidence has been noted by Mitra *et al* (1980) and Bobrow and Bell (1962) who reported an incidence of 54.2% and 63.9%respectively. We had 6 patients in their teens suffering from this condition.

3. Parity

In 18 out of 65 patients, ectopic pregnancy was the first conception. This is comparable to the incidence in many other series, Riva *et al* (1962) 30.0%, Paranjothy (1962) 23.7%, Ghose and Ghose (1968) 29.4% and Wagh and Patel (1968) 24.0%.

Like in most other studies, a majority of our patients were multiparous women.

4. Infertility

There is a close relationship between the factors leading to infertility and to ectopic pregnancy. Further, the risk of

^{**} Assistant Professor.

ectocyesis increases eight-fold when the sent series, 3.2% had been operated for cording to Wyper (1962). Taylor (1978) quotes an incidence of 20.0% among those investigated for sterility. Mitra et al (1968) reports that 10.2% of their cases had diagnostic D & C and 0.8% had HSG done for infertility. The incidence of ectopic was 9.0% among 258 women treated with hydrotubation which makes the risk 27 times greater. A history of involuntary infertility of varying period beyond 2 years of married life was present in 28.0% in this series out of which 8.2% were primary and 19.7% were secondary infertility. Wagh and Patel (1968) found this to be 21.6%, whereas Oumachigui (1976) reported prolonged infertility of more than 10 years in 10.8% of her cases.

Dewhurst (1972) says that the chances of ectopic are very high if tubal surgery has been performed to correct old tubal damage. Grant (1962) gives a figure of 12.0% of ectopic following tubal surgery, while TeLinde (1970) reported that 10-20% developed ectopic following tubal plastic procedures.

5. Previous abortions and MTP

Obstetric history of 2 or more consecutive unsuccessful pregnancies was obtained in 8.3% of our series. History of abortion or MTP within the past one year was forthcoming in 23.3% of cases. Tubal dysfunction or damage following abortion, induced or otherwise appears to be a chief factor in these cases. 3.3% of cases in this series had habitual abortions.

6. Previous ectopic pregnancy

In concurrence with the studies of Mitra et al (1980), and Bobrow and Bell (1962) who reported 4.0% and 3.1% of repeat ectopics respectively. In the pre-

woman is investigated for infertility ac- previous ectopic pregnancy. Oumachigui reported 7.0% in her study, while Jeffcoate (1967) maintains that the risk is 15 times greater than in normal women. It is known that residual tubal damage is to be expected in the contralateral tube following an ectopic gestation. Since clotted blood left behind in the pelvis due to inadequate toiletting or haemostasis cannot be completely absorbed, the other tube may suffer further damage.

7. Pelvic Inflammatory Disease

A tube that is not completely blocked by disease but severely damaged with destruction of cilia can contribute to ectopic gestation. A 'peritoneal factor' is described where tubes are patent but the peritubal adhesions interfere with the normal tubo-ovarian relationship required for tubal pick up.

Westrom (1975) points out that the ectopic is 6 times higher in treated cases of acute PID. 13.8% of cases in our series gave history suggestive of PID. Bobrow and Bell (1962) reported the incidence to be as high as 56.0% along with Jeffcoate (1967) who gave an incidence of 50-60%. Riva et al (1962) reported 12.3% and Ghose and Ghose's (1968) figure was 27.5%.

Salpingitis, gonococcal or tuberculous also plays a major role in causing ectopic gestation. Current treatment of acute salpingitis with antibiotics may be responsible for ecopic gestation as this leads to agglutination of cilia and synechial bands in place of completely blocked tubes in untreated cases. Halbrecht (1957) drew attention to the fact that of the few pregnancies that occurred after medical treatment of TB endometritis, two thirds were ectopic. Kleiner and Roberts (1967) came to the conclusion that chronic endosalpingitis was the most significant aetiological factor. They noticed in 53% of their cases, chronic follicular salpingitis on histopathology, though no bacterial growth was seen on culture. In 37 of cases, postabortal infection preceding the ectopic gestation was found to be the causative factor.

8. Previous Pelvic surgery

In our series, 21.1% of patients had undergone previous pelvic surgery— D & C 8.2%, puerperal sterilization 4.6%, previous ectopic 3.2%, appendicectomy 1.6%, LSCS 1.0% and others 0.6%. The incidence is comparable with the figures of most workers, ranging from Mitra *et al* (1980) 6.4% to Paranjothy's (1962) 25%. The peritubal inflammation and ahesions could explain the high incidence ectopic gestation in these cases.

9. Previous sterilization

4.6% of patients in this series, had tubectomy earlier. Pendse (1981) in his series of 110 cases, had 3 cases with previous tubectomy. Here, the possibilities are that the sperms could be trapped in the distal part of the tube, prior to surgery and later being fertilised, or failed sterilisation due to recanalisation of tubes or improper surgery, and again due to development of tubo-peritoneal fistula. Chakravarthy *et al* (1975) reported that one third of subsequent pregnancies following sterilisation were ectopics.

10. Contraception

Throughout literature we find reports linking various types of contraceptive devices to the onset of ectopic pregnancy and also the implication of prolonged use of IUCD—Jones (1975) Parks (1975) Halatt (1976) Saafan (1976) Burke (1977) Snowden (1977) and Vessey (1979). Tietze (1966) reported that 26

ectopics resulted in relation to 588 intrauterine gestations which occurred with IUCD in situ. In an ICMR study (1962) it was quoted by Mukherji and Mukherji that 0.46% women with IUCD developed ectopic pregnancy. In the present study, 6.6% had used IUCD. Te Linde and Mattingly (1970) noted that in his series of 162 patients who became pregnant while on the "pill", 12% developed ectopic pregnancy.

11. Other factors

Congenital anomalies of tubes like diverticulae and accessory ora could be contributing factors. Here the diagnosis is difficult as they may be missed even at laparotomy. Altered tubal physiology, where ampulla and isthmus behave differently could also be an important factor. The role of oestrogen and progesterone with their control on tubal motility, has also been noted as significant, though the reports available are conflicting on the issue. Te Linde (1970) showed transperitoneal migration of ovum in 15% of cases, a fact put forward also by Jeffcoate (1967). Moore (1979) reported an interesting and rare case of leiomyma of the fallopian tube which was the cause of ectopic pregnancy. However, it must be stressed that no aetiological factor could assigned in many cases. Taylor be (1978) brought to attention certain recent reports of damaged tubes showing histological evidence of viral infection which could explain the absence of traditional causes.

Conclusion

An analysis of aetiological factors in ectopic pregnancy indicates that even though ectopic gestation is not totally preventable, it may be feasible to bring down the incidence by observing certain precautionary measures which are listed below. A high index of suspicion will help one pick up likely cases early so that the morbidity is brought down to negligible levels and mortality totally avoided.

(i) Prevention of self induced and criminal abortions.

(ii) Keeping infection in check in spontaneous abortion and MTP.

(iii) Prevention and treatment of postabortal and puerperal sepsis.

(iv) Early, vigorous and thorough treatment of all pelvic infections including gonorrhoea and tuberculosis.

(v) Care in tissue handling and heamostasis during all pelvic surgery, followed by thorough peritoneal toilet before closure of abdomen.

(vi) Awareness of the diagnosis in the high-risk group including women under investigation for sterility IUCD users and women who have had tuboplasty.

(vii) Awareness of damage in the contralateral tube following the first ectopic pregnancy or pelvic surgery. Tubal patency tests at a later date may be indicated.

Acknowledgement

We thank Dr. S. Krishna Kumari, Professor and Head of the Department of Obstetrics and Gynaecology and the Medical Superintendent of St. Martha's Hospital for permission to publish this paper.

References

- 1. Bobrow, M. L. and Bell, H. G.: Obstet. Gynec. 20: 500, 1962.
- 2. Burke, M: Brit. Med. J. 1: 169, 1977.
- Chakravarthy, S. and Shardlow, J.: Brit. J. Obstet. Gynec. 82: 58, 1975.
- Dewhurst, C. J.: Integrated Obstet. Gynec., for Postgraduates, Ed. 2, p. 239,

Blackwell Scientific Publication, Oxford, 1976.

- 5. Douglas, C. P.: Brit. Med. J. 2: 838, 1963.
- Ghose, N. and Ghose, J.: J. Obstet. Gynec. India 18: 375, 1968.
- Grant, A.: Clin. Obstet. Gynec. 5: 861, 1962
- 8. Halatt, J. G.: Obstet. Gynec. 6: 754, 1956.
- 9. Halbrecht, I.: Obstet. Gynec. 10: 73, 1957.
- Jeffcoate, T. N. A.: Principles of Gynec., Ed. 4, p. 208, Butterworths, London and Boston, 1975.
- 11. Jones, J.: Brit. Med. J. 3: 467, 1975.
- 12. Kleiner, G. J. and Roberts, T. W.: Am. J. Obstet. Gynec. 99: 21, 1967.
- Mitra, S., Sikdar, K. and Mandal, G. S.: J. Obstet. Gynec. India 30: 25, 1980.
- Moore, O. A.: Am. J. Obstet. Gynec. 134: 101, 1979.
- Mukherjee, S N. and Mukherjee, S. I.: I.C.M.R. report 1962, J. Obstet. Gynec. India 18: 241, 1968.
- 16. Oumachigui, A.: Antiseptic. 73: 9, 1976.
- Paranjothy, D.: J. Obstet. Gynec. India 12: 459, 1962.
- 18. Parks, S.: Lancet. 2: 1261, 1975.
- Pendse, V.: J. Obstet. Gynec. India 31: 100, 1981.
- Rajan, R. and Nair, M. S.: J. Obstet. Gynec. India 26: 118, 1976.
- Riva, H. L., Kammeraad, L. A. and Anderson, P. S.: Obstet. Gynec. 20: 189, 1968.
- 22. Saafan, S. T.: Lancet. 1: 193, 1976.
- Snowden, R.: Brit. Med. J. 2: 1600, 1977.
 Taylor, M. L.: Infertility, P. 25, Grune
- and Stralton, London, 1978.
- Te Yinde, R. W. and Mattingley, R. F.: Operative Gynaecology, E. 4, P. 324, J. B. Lippincott Company, Philadelphia, Toronto, 1970.
- Tietze, C.: Brit. Med. J. 2: 302, 1966. 1962.
- 27. Vessey, M. P.: Lancet. 2: 501, 1979.
- Wagh, K. V. and Patel, S.: J. Obstet. Gynec. India 18: 370, 1968.
- Westrom, L.: Am. J. Obstet. Gynec. 121: 707, 1975.
- 30. Wyper, J. F. B.: Brit. Med. J. 1: 275, 1962.