AETIOLOGY OF MALE INFERTILITY ANALYSIS OF 210 CONSECUTIVE CASES

by

R. RAJAN,* M.D., D.G.O.

The time has come when both gynaecologist and urologist should recognise the fact that the male bears a large share of responsibility in a childless marriage, that improvement can be anticipated in a reasonably large percentage of men, provided meticulous care is taken to study their problems individually. The causes of male infertility have been well delineated in numerous articles, but, the frequency with which these are encountered in the course of medical practice has not been well documented.

This presentation deals with the various causes of male infertility and the frequency with which these are encountered in the course of investigation of 210 consecutive cases of subfertile men during the 3 years period, from January 1973 to November 1975.

Material and Methods

All cases of proven male infertility as delineated by a complete physical examination and semen analysis were analysed as to the aetiology. Study of patient's history, complete physical examination and seminal analysis were the order in which each individual was analysed. Different samples of semen were studied when indicated. Those with 20 to 60 million count were considered as cases of mild oligospermia and below 20 million as severe oligospermia. Patients with normal semen qualities were eliminated from the study, except in those cases where sexual difficulties played a significant role in producing infertility. Testicular biopsy was performed in indicated cases to diagnose the obstructive lesions and various types of testicular failures. In a few cases of azoospermia, testicular biopsy could not be done since the patients were not willing for the procedure and these cases were grouped separately.

Results and Discussion

Two Hundred and ten cases of male infertility were studied during the last 3 years. Causes of the fertility problems as diagnosed by the investigations are listed in Table I. Results of semenal analysis are as follows: Azoospermia in 109 cases, mild oligospermia in 54, severe oligospermia in 38, normal sperm count in 7, necrospermia in 1 case and aspermia in 1 (Table II).

Testicular Failure: Testicular failure comprised 27.14% of this reported series of cases (Table III). Except for the 9 patients with spermatogenic arrest, all the others had irreversible testicular lesions. There were 9 cases of Klinefelter's syndrome, with long (disproportionate) lower limbs, gynaecomastia, azoospermia, small testicles showing hyalinised tubules with Leydig cell hyperplasia. In these cases the libido is quite often normal and these patients may require androgenic support at a later time in their lives. The

*Assistant Professor, Obstetrics & Gynaecology, Medical College Hospital, Kottayam.

Accepted for publication on 18-2-76.

TABLE I
Causes of Male Infertility in 210 Cases

Serial No.	Aetiological Factor	Number	Percentage
1.	Testicular failure	57	27.14%
2.	Varicocele	56	26.67%
3.	Oligospermia	48	22.89%
4.	Obstructive azoospermia	24	11.44%
5.	Impotence	4	1.90%
6.	Bilateral cryptorchidism	1	0.48%
7.	Unilateral cryptorchidism	2	0.95%
8.	Inguinal testicles (Surgical)	1	0.48%
9.	Necrospermia	1	0.48%
10.	Hypospadiasis	1	0.48%
11.	Aspermia	1	0.48%
12.	Azoospermia (cause not known)	14	6.67%
	Total	210	100.00%

TABLE II
Results of Seminal Analysis in 210 Infertile Men

No.	Semenal Cytology	No. of cases	Percentage
1.	Azoospermia	109	52%
2.	Mild oligospermia	54	25.50%
3.	Severe oligospermia	38	18.00%
4.	Normal sperm count	7	3.30%
5.	Necrospermia	1	0.50%
6.	Aspermia	1	0.50%
	Total	210	100.00%

TABLE III
Testicular Failure —57 Cases (27.14% of the Total)

No.	Testicular Histology	No.	Percentage
1.	Sertoli-Cell-Only syndrome	21	10.00%
2.	Tubular hyalinization	15	7.14%
3.	Spermatogenic arrest	9	4.28%
4.	Klinefelter's syndrome	9	4.28%
5.	Multiple lesions	3	1.36%
	Total	27.14%	57

apparent Leydig cell hyperplasia in biopsy should not be mistaken for Leydig cell hyperactivity, and if the Leydig cells are counted it may be only normal or even less than normal. Fifteen patients had tubular hyalinisation with peritubular fibrosis and some amount of Leydig cell hypertrophy. Many of them in this group gave a history of mumps orchitis after the age of puberty, and these patients had smaller testicles. Mumps orchitis led to bilateral testicular atrophy in 2.5% of

the series reported by Dubin and Amelar (1971), and in their series 70% were having testicular swelling. However, mumps is undoubtedly a very rare cause of sterility, largely because the condition is so rarely bilateral (White and Green-Armytage, 1962).

Twenty-one patients had Sertoli-cell-only syndrome, with seminiferous tubules populated only by Sertoli cells, while spermatogenesis was absent. In these patients the testicular size was normal, though many had very soft testicles. White and Green-Armytage (1962) are of the opinion that the consistency of the testicles is a much more reliable guide to spermatogenic potential than the size. In 3 cases, there were multiple lesions, consisting of tubular hyalinisation, spermatogenic arrest, sloughing and disorganization. This condition is one of progressive tubular degeneration rather than a separate entity (Girgis *et al*, 1969).

Varicocele: Varicocele was the second commonest cause of infertility. This condition was detected in 56 infertile men (26.67%). Care must be exerted in the examination of all male infertility patients so that a varicocele is not missed. The patient must be examined in the upright position, and the reflux into the scrotal venous system can be determined by palpation while the patient performs valsalva maneuver.

Dubin and Amelar (1971) had reported a very high incidence of varicocele in their series (39%), which probably may be due to increased referral of such cases because of the author's interest in this problem. The effect of varicocele is probably secondary to retrograde flow of blood down the incompetent internal spermatic vein and in 99% it is seen on the left side. In our series there were no right sided lesions. The seminal cytology ranged from azoospermia to oligospermia. The typical stress pattern described by Mcleod (1965) was the commonest abnormality in the seminal cytology. This includes abnormal sperm morphology with tapering and amorphous cells and exfoliation of immature cells of the germinal line into the ejaculate, in addition to oligospermia and hypokinesis. The semen defect is secondary to premature sloughing of immature cells of the germinal epithelium in the seminiferous tubules (Dubin and Hotchkiss, 1969).

Oligospermia: In this group are included 48 cases (22.89%), who had no abnormalities like varicocele, hydrocele or trauma. The seminal count varied from very few sperm to 20 million per cc (severe oligospermia) and from 20 to 60 million (mild oligospermia). The etiologic factors involved in this group may be endocrine problems or environmental factors. 8.6% of the cases documented by Dubin and Amelar (1971) had definite evidence of hormonal abnormalities. Of these cases, 7.1% had hypopituitarism, 0.9% had adrenal dysfunction and 0.6% had thyroid dysfunction.

Oligospermia could theoretically be caused by a partial obstruction, but in practice this must be extremely rare, and one must assume that low sperm density and poor morphology are nearly always due to defect in spermatogenesis. But, Charny (1963) had reported oligospermia in men with obstructive lesions.

Obstructive Azoospermia: Twenty-four patients in this group had azoospermia with normal spermatogenesis proved by testicular biopsy. This was the fourth common cause of sterility, comprising of 11.4% of the total cases. There were 2 cases of surgical block due to vasectomy in this series, and the cause was not known in the other cases. Dubin and

Amelar had reported an incidence of ductal obstruction in 7.4% of infertility cases (1971). Grigis *et al* (1969). in their analysis of 843 cases of azoospermia, had reported obstructive azoospermia in 466 cases (55%), and the commonest site of obstruction was in the distal part of the epididymis and/or the adjoining part of the vas deferens, usually congenital or due to epididymitis from gonorrhoea or tuberculosis.

Sexual Problems: Sexual problems in couples, where the male has normal semen quality, was observed in 4 cases (1.9%). In this group the organic cause of impotence must be differentiated from the psychologic cause. The common causes include diabetes, neurologic disease, endocrine disorders, sympathectomy, perineal trauma and surgery, drugs such as anticholinergics, tranquillizers and certain antihypertensive agents.

Cryptorchidism: The literature on cryptorchidism is vast (Snyder and Greeney, 1962). Dubin and Amelar (1971) had reported 56 cases of azoospermia due to cryptorchidism. Some undescended testes are pathologic to begin with and the same developmental error may be responsible for the non-descent as well as the azoospermia. Hansen (1949), Mack (1953), and Scott (1961) have found that there is a high incidence of impaired fertility associated with postpubertal unilateral cryptorchidism. In the present series there were 3 cases of cryptorchidism, 2 were unilateral and 1 was bilateral. The patient with bilateral cryptorchidism presented with azoospermia. In the unilateral variety, one was azoospermic and the other oligospermic.

Necrospermia: There was one patient with necrospermia, who was completely normal with excellent sperm count and good morphology. Since there was no motility the infertility was irreversible.

Hypospadiasis: There was one case of hypospadiasis, in an otherwise normal person with normal seminal cytology, preventing proper deposition of semen into the vagina. The female investigation proved to be normal, but for a negative PCI, done repeatedly. There was no sexual problem for this couple. Dubin and Amelar (1971), had 2 cases of hypospadiasis, in their series, where husband insemination was performed successfully.

Apsermia: No ejaculate was present in one patient investigated for infertility. He had normal development and had no sexual problems. He had sensed orgasm at the time of intercourse but there was no emission. The exact cause of this condition was not known in the case. Retrograde ejaculation, due to bladder neck dysfunction following sympathetic blockade, is the commonest cause of absence of ejaculate (Green *et al*, 1963; Dubin and Amelar, 1971). Under such circumstances, semen can be recovered by bladder lavage, immediately after intercourse. Aspermia can be rarely caused by obstructive lesions like congenital absence of the deferens and seminal vesicles or inflammatory block at the level of ejaculatory duct.

In addition to the above mentioned cases, there were 14 cases of azoospermia in whom testicular biopsy could not be performed, hence a proper diagnosis was not arrived at.

Summary

The etiology of male infertility in 210 consecutive cases, studied over a 3-year period has been outlined and discussed. Testicular failure due to various causes and varicocele are found to be the two common causes of male infertility, constituting more than 50% of the cases. Other important factors are oligospermia

due to endocrine causes, and ductal obstruction. Impotence, as a cause of infertility, was seen only in 4 cases (1.9% of total cases).

Acknowledgement

I am grateful to the Pathologist, Dr. Malini Thomas for the co-operation, and the Surgeon, Dr. Chacko Valayil for the help rendered in preparing this paper.

References

1. Charny, C. W.: Fetil. & Steril. 14: 610, 1963.
2. Dubin, L. and Hotchkiss, R. S.: Fertil. & Steril. 20: 50, 1969.
3. Dubin, L. and Amelar, R. D.: Fertil. & Steril. 22: 469, 1971.
4. Girgis, S. M., Etriby, A., Ibrahim, A. A. and Kahil, S. A.: Fertil. & Steril. 20: 467, 1969.
5. Greene, L. F., Kelalis, P. P. and Weeks, R. E.: Fertil. & Steril. 14: 617, 1963.
6. Hansen, T. S.: Proc. Roy. Soc. Med. 42: 645, 1949.
7. Mack, W. S.: Proc. Roy. Soc. Med. 46: 835, 1953.
8. McLeod, J.: Fertil. & Steril. 16: 735, 1965.
9. Scott, S. L.: J. Reprod. Fertil. 2: 54, 1961.
10. Snyder, W. H. and Greaney, E. M.: Paed. Surgery. Year Book. 2: 1041, 1962.
11. White, M. M. and Green-Armytage, V. B.: The management of impaired Fertility, Oxford University Press, Part V: 273, 1962.