VASOVASOSTOMY FOR OBSTRUCTIVE AZOOSPERMIA DUE TO HERNIORRHAPHY IN CHILDHOOD

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ABSTRACT

Vasovasostomies for eight cases of obstruction of the vas deferens following herniorrhaphy in childhood were performed. Two pregnancies were obtained postoperatively. The following factors have been shown to be associated with a favourable result: a vasal obstruction in the subcutaneous inguinal ring, a dilatation of the proximal vas deferens, and the presence of sperm in the intravasal fluid that accumulates proximal to the obstructive site of the vas deferens. In the two successful cases, the time intervals after herniorrhaphy were 16 years and 20 years, respectively, which were considered to be long delays between vas occlusion and its reanastomosis.

Key Words: Vasovasostomy, Herniorrhaphy.

INTRODUCTION

Azoospermia due to obstruction of the vas deferens following herniorrhaphy in childhood is rare. Its therapy is technically difficult and its prognosis appears unfavourable. We examined eight patients with acquired obstructive azoospermia resulting from vas occlusion due to herniorrhaphy in childhood. Factors other than operative techniques might be expected to play a part in determining the outcome of vasovasostomy, and this paper attempted to analyse some of these.

CASES AND OPERATIVE TECHNIQUE

Eight men aged 23 to 36 years (mean 29.1 years) complaining of azoospermia were admitted to Nagoya University Hospital for vasovasostomy between September 1979 and February 1985. These patients had undergone bilateral inguinal herniorrhaphy in childhood. The interval from herniorrhaphy to vasovasostomy ranged from 16 to 30 years (Table 1). Seven testicular biopsies were performed and the biopsy specimens were placed in Carnoy's solution prior to fixation and to examination by light microscopy. Spermatogenesis was assessed by the Johnsen's score (healthy control's score 10). Light microscopy of six biopsy specimens taken from the testes showed the normal spermatogenesis to be well preserved. Impaired spermatogenesis was seen in only one biopsy specimen (case 8).

At the time of operation, an attempt was made to assess the length and position of vasal

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Table 1. Details of patient population and results in patients treated with vasovasostomy.

| | | | | | 1 | I | During surgery | | | After surgery | ıry |
|----------------|-----|--|------------------------------|--|--|--|---|---|-------|---|--------------------------|
| Name | Age | Diagnosis | Years after herniorrhaphy | Testicular biopsy (Johnsen's score) | Surgery (Date) | Site of obstruction | Dilatation of proximal vas deferens | Intravasal fluid from proximal vas deferens | Sperm | Appearance of sperm in semen | Delivery |
| 1 H.T. | 26 | obstruction of bil. vas deferens | 16 | 6 | bil. vasovasostomy (1980.6.5) | Lsubcutaneous inguinal ring Rsubcutaneous inguinal ring | ≢ । | ++ yellow-white - | + 1 | 5 months after 30 x 10 ⁶ /ml 40% (motility) 10% (deformity) | 27 months after, girl |
| 2 M.G. | 32 | obstruction of left vas deferens defect of right vas deferens | 20 | 9-10 | left vasovasostomy (1979.10.8) | Lsubcutaneous inguinal ring | ‡ | + yellow-white | ± | 5 months after 40 x 10°/ml 50%(motility) 25%(deformity) | 14 months after, boy |
| 3 S.S. | 35 | obstruction of left vas deferens right abnormal seminal vesicle | 30 | 9-10 | left vasovasostomy (1980.8.22) | Labdominal inguinal ring | ‡ | brown | ± | 8 months after _ | |
| 4 T.K. | 36 | obstruction of bil. vas deferens | 20 | 9-10 | bil. vasovasostomy (1980.9.24) | Labdominal inguinal ring Rsubcutaneous inguinal ring | ‡ ≀ | mikly-white + white | 1/HPF | 12 months after 2~3/HPF 1/HPF 16 months after | 1 |
| 5 S.S. | 26 | obstruction of bil. | 24.5 | | right vasovasostomy (1984.1.20) | Ldefect of distal vas deferens Ringuinal canal, and puncture site for vasography | ‡ | +++ yellow-white | ± ± | 1 month after 3~4/HPF 5 months after | |
| 6 S.I. | 30 | obstruction of left vas deferens right testis atrophy | 29 | 9-10 | left vasovasostomy (1984.3.1) | Lsubcutaneous inguinal ring | + | white | ‡ | 6 months after 60 x 10°/ml 0%(motility) 40~60%(deformity) | 1 |
| 7 S.I. | 26 | obstruction of bil. vas deferens | 23 | 9-10 | right vasovasostomy (1984.10.18) | Ldefect of distal vas deferens Rscrotal vas deferens | + + | yellow + yellow | 1 1 | 7 months after _ | 1 |
| % % N.N. | 23 | defect of right distal vas deferens left testis atrophy | 22 | 2-9 | anastomosis of right proximal vas deferens to left distal vas deferens (1985.2.14) | Rdefect of distal vas deferens | I | - white | 1 | 1 month after | 1 |

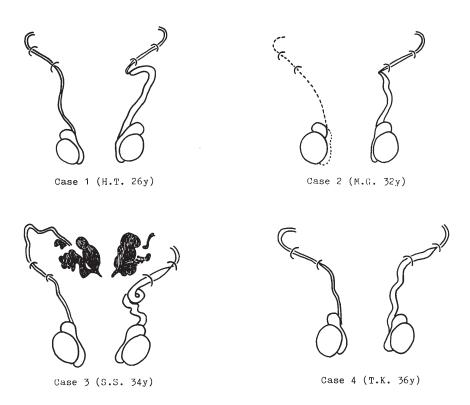


Fig. 1. Illustrated initial pathological state of vas deferens, epididymis, and testis in four patients. Dotted line indicates disappearance of epididymal tail and right vas deferens. Bilateral cystic seminal vesicle demonstrated by vasography during operation is illustrated in case 3.

obstruction. The results were shown in Fig. 1 and 2. The most common obstructive site of the vas deferens was the proximal area to the subcutaneous inguinal ring. Vasal obstruction in the abdominal inguinal ring was disclosed in two cases (cases 3 and 4). We failed to find the vas deferens in the part indicated by dotted lines as shown in Fig. 1 and 2. The vas deferens distal from the obstructive site had disappeared in most cases. Both the right vas deferens and the right testicular vessels had disappeared above the subcutaneous inguinal ring in case 5. In case 8, the right vas deferens had disappeared above the subcutaneous inguinal ring, while testicular vessels existed in the right spermatic cord. Testicular atrophy was seen in cases 6 and 8. This atrophy was probably due to a damaged testicular vascular system at the time of herniorrhaphy.

The grade of dilatation of the proximal vas deferens, the presence or absence of sperm in the intravasal fluid from the proximal vas deferens and its amount and gross appearance were shown in Table 1. In the patients who had a deep yellowish intravasal fluid, the appearance of sperm in this fluid was recognized except in case 7.

The vasovasostomies were performed under epidural anesthesia. A 10 cm incision was made in the scrotal sac and the distal and proximal vas deferens were sufficiently exposed. If the obstructive site was above the abdominal inguinal ring, the incision was extended upward to the inguinal canal.

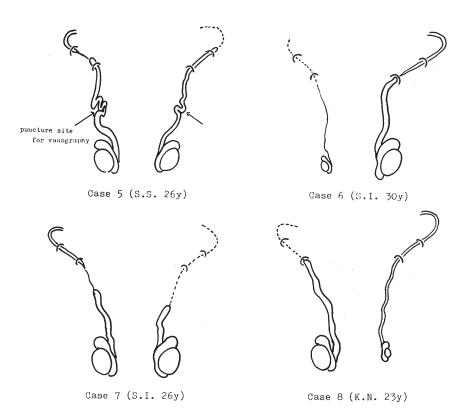


Fig. 2. Illustrated initial pathological state of vas deferens, epididymis, and testis in remaining four patients. Arrows in case 5 show puncture site for preoperative vasography causing vasal obstruction. Dotted lines indicate disappearance of distal vas deferens. Atrophic testis is illustrated in cases 6 and 8. Obstruction of right vas deferens in case 7 was about 7 cm in length.

If the distal vas deferens was not detected, the incision was extended further to examine the retrovesical space and to determine the presence or absence of distal vas deferens. After the obstructive site was confirmed, the distal and proximal ends of the vas deferens were cut. If the proximal vas deferens was dilated, distal end was cut obliquely in spatulated form to give more surface area as shown in Fig. 3A and B.

The emission of intravasal fluid from the proximal vas deferens and the existence of sperm in this fluid were evaluated. Patency of the distal vas deferens was determined next by injecting normal saline solution into it through a blunted 23-gauge hypodermic needle. This was done easily if no obstruction existed. Patency of the proximal vas deferens was proved by the appearance of spermatic fluid.

Anastomosis was performed using a technique similar to that described by Amelar and Dubin² (Fig. 3). The full thickness of the vasal wall was sutured with 9–0 Dexon at 12 and 6 o'clock positions of the vas stump, and the outer layer of the vas end was then sutured interruptedly with 6 to 9 stitches using 7–0 Dexon. These sutures were made as tightly as possible. The distal and proximal vas deferens were stabilized by utilizing the relatively sturdy fascia in the surroundings to avoid tension on the anastomosis site. A sterile dressing and a scrotal support were applied. The supprot was worn for seven days to immobilize the scrotum. Testing for sperm was commenced after one month.

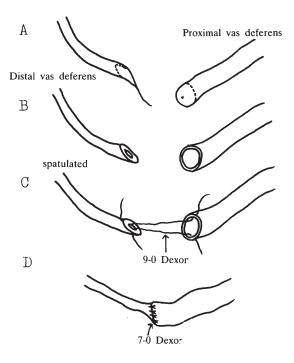


Fig. 3. Vasovasostomy: A) Ends of vas deferens are exposed and obstructed ends are excised; B) After patency has been tested, distal vas deferens may require spatulation; C) Vasovasostomy with interrupted 9-0 Dexon atraumatic sutures; D) Completed anastomosis.

POSTOPERATIVE COURSE

We have noted five cases of sperm appearance in semen following vasovasostomy. Two patients impregnated their wives (cases 1 and 2).

In case 3, the preoperative vasography showed normal right vas deferens and obstructive left vas deferens; therefore, vasovasostomy was performed only on the left side. Vasography during operation indicated a left cystic seminal vesicle, and a large number of sperm were found in the intravasal fluid from the left proximal vas deferens. However, sperm did not appear in the semen eight months after operation. Postoperative vasography revealed stenosis of the anastomosis site due to scarring.

In case 4, the sperm number in the intravasal fluid from the proximal vas deferens was small, but sperm was seen in the semen 12 months after surgery. However, there was an actual decrease in number and motility, and, ultimately, a total absence of sperm in the ejaculate.

The patient in case 5 was unlucky. Before receiving treatment at our hospital, this patient underwent bilateral vasography at another hospital. Both induration and obstruction of the vas deferens in the puncture site for vasography were identified at the time of vasovasostomy; therefore, anastomosis was performed at two places. The treatment resulted in severe oligozoospermia and the patient became azoospermia five months later.

In case 6, a sufficient number of sperm were found in the semen after operation, but they had low motility and a high percentage of deformity. This patient had undergone herniorrhaphy at 1 year

old. It is conceivable that some mild disturbance in the spermatogenesis may have occurred during the 29 years after herniorrhaphy.

In case 7, the left distal vas deferens had disappeared. Accordingly, only a right vasovasostomy was performed. The stricuture of the vas deferens was 7 to 8 cm in length. Induration in the caudal part of the right epididymis was discovered during operation. This induration remained even after release from vasal obstruction. There was no sperm appearance in the semen seven months after operation.

In case 8, the right vas deferens had disappeared above the subcutaneous inguinal ring and the left testis was atrophic. However, the right testicular volume was normal and the left vas deferens maintained patency. Thus, we performed a transseptal crossed anastomosis³ on the right proximal vas deferens to the left distal vas deferens. During operation, we failed to find any sperm in the intravasal fluid from the right proximal vas deferens. There was no appearance of sperm in the semen one month after operation.

DISCUSSION

Iatrogenically acquired ductal obstruction may be seen after herniorrhaphy, varicocelectomy, inguinal division of the spermatic vein, lower ureteral lithotomy, and hydrocelectomy. This form of obstruction is amenable to surgical correction after the site of obstruction has been established and normal ductal structures have been demonstrated to exist proximal and distal to the obstruction.^{4,5} There have been many reports on vasectomy reversal in regard to the condition of the intravasal fluid from the proximal vas deferens, which effects the results of vasovasostomy.⁶ Irreversible obstruction of the seminal duct in the epididymis due to chronic inflammation and fibrosis is one reason for intravasal azoospermia.⁶ After the operation, sperm counts are often low and sometimes sperm do not appear until months later even if sperm was detected in the intravasal fluid from the proximal vas deferens during operation. Two reasons for these phenomena are possible: (a) After vasal obstruction, the tubules of the epididymis dilate. Their walls are thin and, when dilated, become even thinner. Just as a chronically dilated ureter seldom regains its tone or its ability to peristalse after obstruction is corrected, the epididymis may be the site of a similar condition slowing sperm transport and causing many sperms to die before they reach the vas deferens. (b) After vasal obstruction, a balance is reached between sperm production and sperm absorption. Phagocytosis accounts for sperm absorption and is far more active than normal. Phagocytosis may also remain hyperactive so that only few sperm escape it.

Vasography should always be avoided in patients who have vasal obstruction caused by herniorrhaphy. Performing a vasography as an isolated diagnostic procedure creates many problems as seen in case 5. It should not be used for diagnosing obstruction or for deciding to perform surgery for obstruction. Ross insists that routine vasography is to be avoided, since it may damage the vas deferens or the delicate epididymal tubule causing scarring and obstruction. If a vasography is needed, it should only be performed as part of the whole operative procedure for corrrecting obstruction.

The interval between vasal obstruction and vasovasostomy usually does not appear to influence the rate of success in vas reanastomosis, but this interval may be critical in the individual patient who has suffered irreversible obstructive damage to the testes or epididymides after vasal obstruction. Schmidt has reported successful reanastomosis as long as 21 years after vasectomy. Silber reported that he obtained poor results in patients who had vasectomies performed more than 10 years before reanastomosis and excellent results when the vasectomy was performed within two years of the reversal operation. The data that would allow conclusions with respect to a critical interval beyond which reconstruction of the vas deferens should not be attempted are not available presently.

REFERENCES

- 1) Johnsen, S.G.: Testicular biopsy score count: a method for registration of spermatogenesis in human testis. Normal values and results in 335 hypogonadal males. *Hormones*, 1, 2–25 (1970).
- 2) Amelar, R.D. and Dubin, L.: Vasectomy reversal. J. Urol., 121, 547-550 (1979).
- 3) Lizza, E.F., Marmar, J.L., Schmidt, S.S., Lanasa, J.A.Jr., Sharlip, I.D., Thomas, A.J., Belker, A.M. and Nagler, H.M.: Transseptal crossed vasovasostomy. *J. Urol.*, **134**, 1131–1132 (1985).
- 4) Matz, M., Zepnick, H., Adler, D. und Kuster, P.: Zur samenleiterobstruktiven Azoospermie nach inguinaler Herniotomie. Z. Urol. u. Nephrol., 77, 543-547 (1984).
- 5) White, R.D. and Paulson, D.F.: Obstruction of the male reproductive tract. *J. Urol.*, 118, 266-268 (1977).
- 6) Sharlip, I.D.: The significance of intravasal azoospermia during vasovasostomy. *Fertil. Steril.*, **38**, 496–498 (1982).
- 7) Silber, S.J.: Microsurgery for vasectomy reversal and vasoepididymostomy. *Urology*, 23, 505-524 (1984).
- 8) Ross, L.S.: Diagnosis and treatment of infertile men: a clinical perspective. *J. Urol.*, **130**, 847-854 (1983).
- 9) Wicklund, R. and Alexander, N.J.: Vasovasostomy: evaluation of success. *Urology*, 13, 532-534 (1979).
- 10) Schmidt, S.S.: Anastomosis of the vas deferens: an experimental study. II Success and failure in experimental anastomosis. *J. Urol.*, **81**, 203–205 (1959).
- 11) Silber, S.J.: Vasectomy and vasectomy reversal. Fertil. Steril., 29, 125-140 (1978).