EJACULATORY DUCT OBSTRUCTION

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ABSTRACT

The diagnosis, long-range pathophysiology and surgical approach to ejaculatory duct obstruction are presented. Of 5 cases 3 had epididymal extravasation from long-term pressure buildup and secondary epididymal obstruction. This is similar to the epididymal findings after long-term vasectomy.

Obstruction is a frequent cause of azoospermia. When the semen volume is <1.0 cc and fructose is absent, congenital absence of the vas deferens is the most likely diagnosis. However, if a vas deferens can be palpated in the scrotum, the problem is more likely to be congenital obstruction of the ejaculatory duct. We have seen and treated 5 patients who have clarified the natural history of congenital ejaculatory duct obstruction. Long-term pressure buildup from years of obstruction frequently leads to epididymal dilatation, perforation, sperm extravasation into the epididymis and secondary epididymal occlusion. In these cases microsurgical vasoepididymostomy is required in addition to transurethral resection of the ejaculatory duct.

CASE REPORTS

Case 1. A 31-year-old man with no other health problems had been trying to impregnate his wife for 5 years. He was noted to have azoospermia and underwent a testicile biopsy and vasogram. The vasogram showed patency on the left side and absence of the vas on the right side. A testicile biopsy revealed normal spermatogenesis.

The patient was first referred to this hospital for a possible vasoepididymostomy. Physical examination revealed no abnormality except for an absent vas deferens on the right side and a cyst-like abnormality on rectal examination in the region of the seminal vesicle. Semen analysis revealed a volume of only 0.5 cc, negative fructose and no sperm in the semen. Scrotal exploration was done on the left side with an operative vasogram and a left testicular biopsy. Testicular biopsy again showed normal spermatogenesis. The vasogram revealed a dilated but intact vas deferens up to a blind-ending, dilated ejaculatory duct with a severely dilated, congested left seminal vesicle (fig. 1). There was no spillage of contrast medium into the prostatic urethra or bladder. The ejaculatory duct appeared to be obstructed at its orifice just within and beneath the prostate gland.

There was a copious amount of creamy, white fluid noted in the vas deferens coming from the testicular side of the vasectomy, which harbored no sperm or sperm parts but only amorphous debris. Exploration of the epididymis revealed marked congestion. The epididymis was transversely sectioned, working from the tail proximally towards the corpus. At the distal corpus region there was somewhat less congestion, with translucent-appearing epididymal fluid that harbored many normal sperm. Sections of the epididymis distal to this site revealed interstitial sperm granuloma and obstruction. A microsurgical anastomosis of the inner lumen of the vas deferens directly to the epididymal tubule leaking normal sperm fluid was obtained using 4, 9-zero nylon interrupted sutures. The outer muscularis of the vas was then separately sutured to the outer epididymal tunic.1 Postoperatively, the semen still had a volume of only 0.5 cc, negative fructose and no sperm. At cystoscopy 9 days later the prostatic urethra, verumontanum, bladder and bladder neck appeared to be normal. No ejaculatory duct orifice could be identified. A resection of the floor of the prostate was performed in a small area just proximal to the external sphincter and distal to the bladder neck, on the left side of the verumontanum. The first of several bites revealed only normal prostatic tissue and no evidence of the obstructed ejaculatory duct. With persistent resection a large, gaping, cyst-like opening was revealed on the prostate floor. The resectoscope could then be slipped into this cavity and the seminal vesicle and ejaculatory duct were identified. All bleeders were cauterized carefully and the resectoscope was removed.

Postoperatively, the semen had a normal fructose and the volume was consistently between 3 and 4.5 cc. Sperm were noted in the ejaculate by 6 months. It is still too early to know if fertility will recover. At 1 year postoperatively there are several million sperm per cc with only 5 per cent motility.

Case 2. A 41-year-old general surgeon from Iran, with a 10-year history of azoospermia, had normal spermatogenesis on a testicile biopsy. Semen analysis revealed negative fructose and a semen volume of 0.8 cc. There was no sperm or cellular material in the semen. Physical examination revealed a dilated, indurated epididymis with a palpable vas in both scrotal sacs.

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Fig. 1. Vasogram in case 1 demonstrates markedly dilated vas deferens and seminal vesicle on left side with ejaculatory duct ending in blind cyst-like cavity just beneath prostate urethra. Epididymal obstruction also was encountered owing to blow-outs and extravasation in epididymis.
A cyst-like mass was palpated posterior to the prostate gland bilaterally.

The patient underwent scrotal exploration. The findings in each scrotal sac were essentially identical. The vas deferens was markedly dilated. A vasogram demonstrated the vas to be intact up to the area of the seminal vesicles with a blind-ending, cyst-like dilated ejaculatory duct and massively congested seminal vesicles (fig. 2). There was a copious amount of creamy, yellow fluid coming from the testicular side of the vasotomy site on the right and left sides. None of this fluid contained any sperm. Exploration of the epididymis under the operating microscope revealed massive dilatation. Serial transsections from the tail of the epididymis towards the mid corpus eventually demonstrated fluid that was more translucent in appearance and contained many normal sperm. Histologic sections of the epididymis distal to this site revealed extensive interstitial sperm extravasation and secondary epididymal obstruction. The inner canal of the vas deferens was then anastomosed to the epididymal tubule.

A transurethral resection was performed 2 weeks later to open up the congenitally obstructed ejaculatory ducts. On each side the cyst-like dilatation of a blind-ending ejaculatory duct was unroofed and a wide open channel into the prostatic urethra was created. The seminal vesicles and ejaculatory duct could be observed directly through the scope. Because of political problems in this country, followup has been difficult to obtain. The semen volume has increased but we have no formal semen analysis report.

Case 3. A 31-year-old man has had azoospermia for the last several years. Scrotal exploration at another institution 1 year previously revealed a normal testicle biopsy, congenital absence of the right vas deferens and "a totally fibrosed vas on the left".

Physical examination was unremarkable except for an absent vas deferens on the right side and a large cyst-like mass located superior rather than posterior to the prostate on the left side.

Semen analysis revealed a volume of 0.5 cc, negative fructose and no sperm. Scrotal exploration and a vasogram revealed no sperm in the vas fluid and a markedly dilated congested vas deferens. A vasogram showed that the vas deferens and seminal vesicles came to a blind ending, in this case behind the ureteral orifice of the bladder 2 cm. above the prostate gland. Serial transsections of the epididymis again revealed massive dilatation but at a point near the junction of the tail of the epididymis and the corpus translucent rather than creamy fluid was found, which harbored many normal sperm. The sections of the epididymis distal to this region showed interstitial sperm granuloma. A microscopic vasectomy and vasectomy was done. Later that week cystoscopy was performed with an effort to form a channel transurethrally from the blind-ending ejaculatory duct into the prostatic urethra. Postoperatively, the semen volumes at first slowly increased to 2.0 cc but then returned to 0.5 cc by 6 months. The difficulty in establishing a reliable channel between the seminal vesicles and the prostatic urethra (because of the large separation between them) spelled the failure in this patient.

Case 4. A 28-year-old man was referred from Central America with a diagnosis of epididymal obstruction because of azoospermia and a normal testicle biopsy. However, the semen volume was <1.0 cc, fructose was negative and he had a vas deferens palpable bilaterally. Scrotal exploration revealed normal sperm in the vas deferens and a vasogram showed a small blind-ending ejaculatory duct obstruction. There was no secondary epididymal obstruction and transurethral resection of the ejaculatory duct was done the following day. Because the blind-ending ejaculatory duct was so small it was uncertain if the resection would be successful in this case. Semen volume stayed low and the patient remains azoospermic.

Case 5. A 31-year-old man was referred for an artificial spermatocele because of azoospermia and presumed bilateral congenital absence of the vas (diagnosed because of a low semen volume and negative fructose). However, on physical examination the vas was absent only on the left side. On the right side the vas was normal and the epididymis was dilated. On rectal examination a cyst-like mass was palpated just behind the
prostate, which turned out to be a blind-ending dilated ejaculatory duct and seminal vesicle. Therefore, an artificial spermatocele will not be created but a transurethral resection of the ejaculatory duct will be done.

DISCUSSION

All 5 of these patients had a congenital obstruction of the ejaculatory duct as their primary problem. Originally, they did not have obstruction of the epididymis. Obstruction of the epididymis occurred in 3 patients because of leakage and blowout after many years of high intratubular pressure building up owing to the primary ejaculatory duct obstruction. Thus, these patients had the same sort of secondary epididymal obstruction as seen in long-standing vasectomy patients. It would be difficult to understand the separate locations of obstruction, at the ejaculatory duct and also in the epididymis, or the different causes of the obstruction (congenital versus inflammatory), without the previous studies on vasectomy reversal that have been reported.2,4 Obstruction of the vas deferens or ejaculatory duct for many years leads to dilatation, increased pressure and eventual blowouts in the epididymal tubule, which result in interstitial sperm granuloma and secondary obstruction in the epididymis. Correcting only 1 of these 2 sites of obstruction would not be sufficient.

All 5 of these patients had undergone previously scrotal exploration with an attempted vasogram but their diagnosis remained obscure. A low semen volume and a negative semen fructose should lead to a diagnosis of either ejaculatory duct obstruction or total absence of the seminal vesicles. A cyst-like mass palpated posterior to the prostate gland on physical examination gave a clue to the actual diagnosis but it was not certain until the time of scrotal exploration. A vasogram established the diagnosis and the secondary damage in the epididymis was hinted at by the absence of sperm in the vas fluid. Then serial sections of the epididymis confirmed that epididymal perforation and interstitial inflammation led to secondary epididymal occlusions. Thus, a vasoepididymostomy was performed as well.

Successful unroofing of the ejaculatory duct obstruction resulted in an increase of the semen volume and a positive fructose test, despite up to 41 years of low semen volume, azospermia and a negative fructose. The transurethral technique for unroofing a blind-ending ejaculatory duct and obstructed seminal vesicle is not easy. The indications for attempting it usually have been infection in an obstructed seminal vesicle and most such patients have eventually required an open pelvic operation.4 The difficulty in the transurethral operation is that one cannot reach the ejaculatory duct cavity after only 1 or 2 bites with the resectoscope. A somewhat more radical and deeper resection is needed to be successful. This requires experience and skill with the resectoscope to avoid inadvertently entering the rectum or damaging the sphincter.

Figure 3 is a normal vasogram demonstrating how the ejaculatory duct would appear if it were not obstructed. Approximately 1½ cm. of this duct are within the prostate gland posterior to the area visualized with the resectoscope just to the side of the verumonatanum. By comparing this normal vasogram to the vasograms of ejaculatory duct obstruction, one can see where the resection starts to open the blind-ending channel. Figures 4 and 5 demonstrate the various steps in approaching and resecting this area of the prostatic floor transurethrally. Resection distal or proximal to this point could lead to retrograde ejaculation or incontinence.

These 5 cases appear to demonstrate that as in long-standing...
FIG. 5. Patient with bilateral ejaculatory duct obstruction. Opening resected with transurethral resection is so large that it does not scar and congenital defect is, thus, treated effectively.

Vasectomy many of these patients undergo secondary epididymal disruption owing to pressure buildup from the proximal obstruction. This results in 2 sites of obstruction, both of which must be corrected. Because there are 2 problems that are usually long standing, each of which is difficult to correct surgically, these patients have a poorer prognosis than the usual case of obstructive azoospermia.

REFERENCES


EDITORIAL COMMENT

Another entity to be considered in the differential diagnosis of the infertile man who has palpable vasa but azoospermia with a low semen volume and absent fructose is the atypical form of retrograde ejaculation. The ejaculatory ducts are not blocked but are directed so that the products of the vasa and seminal vessels empty into the bladder, while the prostatic fluid is ejaculated outward. Therefore, when azoospermic patients with palpable vasa but scanty outward ejaculate that does not contain fructose are evaluated the post-ejaculation urine should always be checked for sperm and fructose. If this test is unrewarding then vasography should be done.

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