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Late failure of vasectomy after two documented analyses showing azoospermic semen

TIM PHILP, JOHN GUILLEBAUD, DAVID BUDD

Abstract

From April 1970 to December 1980, 14 047 men underwent vasectomy for sterilisation under local anaesthetic at this clinic. In each man sterility was confirmed by two analyses of semen showing azoospermia. Allowing for a minimum follow up of three years, the wives of six of these men subsequently became pregnant between 16 months and three years after vasectomy. Analyses of semen confirmed recanalisation of the vasa deferentia in all six men. Only five similar cases initially fulfilling the same criteria for sterility have previously been reported.

Full account of the rare possibility of recanalisation should be taken both when couples are counselled preoperatively and when pregnancy occurs after the male partner has been confirmed to be sterile.

Introduction

Vasectomy is one of the most reliable methods of birth control, but failure to achieve sterility because of spontaneous recanalisation of the vasa is well recognised and has previously been reported to occur after about 0·4-0·5% of operations.[1 2] Recanalisation has generally been thought to occur within a few weeks after surgery, and two consecutive analyses of semen showing azoospermia some weeks after surgery are therefore usually required to ensure sterility.[3] We report here on six patients in whom recanalisation occurred after zero sperm counts had been recorded at least four months postoperatively.

Elliot-Smith Clinic, Churchill Hospital, Oxford
TIM PHILP, FRCS, clinic surgeon
JOHN GUILLEBAUD, FRCSED, MRCOG, clinic surgeon
DAVID BUDD, FRCS, director

Correspondence and requests for reprints to: Mr J Guillebaud.

Methods

From April 1970 to December 1983, 16 796 men from the Oxford area underwent vasectomy at this clinic. They were referred by their general practitioners, and the operation was done under local anaesthesia. Patients were then provided with specimen bottles and advised to submit two ejaculates, the first to be obtained four months after vasectomy and the second to be obtained one to two weeks later. The standard criterion for sterility was two consecutive analyses showing semen to be completely azoospermic at least four months after surgery.

Until 1974 all vasectomies had been done by excision of a segment of vas and catgut ligation of the ends, with one or both ends doubled back, usually without fascial interposition. After 1974 and the successes reported by Schmidt,[4] most vasectomies were done by intraluminal diathermy. For this, a unipolar Hyfrecator probe (Birtcher Corporation, California) was passed proximally and distally about one centimetre down each end of the divided vas, coagulating for about three to four seconds until the muscle just became opaque, with minimal charring of the cut surface. Fascial interposition was done in only a few cases.

Results

About 4500 of the 16 796 vasectomies were done by ligation, the remainder by intraluminal diathermy. Side effects were rare, and early failures, identified by postoperative sperm counts, occurred in 71 men, giving an overall incidence of 0·4%. No clear difference in incidence of failure could be shown between ligation and fulguration.

During the same period six wives of men who had undergone vasectomy, and in whom zero sperm counts had been recorded after surgery, became pregnant. When each husband's ejaculate was analysed numerous sperm were found (table I).

Each vasectomy had been performed by a different surgeon; the surgeons were of varying degrees of experience. In each case the Schmidt technique had been used without any postoperative complications. The interval from operation to conception varied from 16 to 36 months. The sperm counts showed appreciable variation both between and within patients. Five of the six men underwent reoperation and showed recanalisation on either histological or radiological examination. Obvious sperm granulomas were seen in only two. The sixth couple opted for female sterilisation after termination of pregnancy.

TABLE I—*Details of men in whom late recanalisation occurred after vasectomy at this clinic*

Case No	Age (years)	No of vasectomies previously performed by surgeon	Time from vasectomy to tests for aspermia (months)		Time to conception (months)	Sperm counts after recanalisation	Outcome
			Test 1	Test 2			
1	36	100-999	4½	5½	36	"Present" motile 10 000 non-motile* 7×10^6 non-motile*	Termination of pregnancy; wife sterilised
2	38	>1000	4	4½	36	3×10^6 motile	Termination of pregnancy; second vasectomy (unilateral sperm granuloma)
3	35	100-999	4	4½	34	"Present" motile $1{\cdot}6 \times 10^6$ motile	Second vasectomy (bilateral sperm granulomas)
4	36	>1000	3½	4	20	190×10^6 non-motile* 52×10^6 motile	Termination of pregnancy; second vasectomy
5	44	100-999	4	7	16	9×10^6 non-motile*	Second vasectomy
6	29	100-999	4	4½	21	500 000 motile 750 000 non-motile*	Termination of pregnancy; second vasectomy

*Absence of motility explained by delay of two to four days before test performed.

TABLE II—*Cases of recanalisation in men who had undergone vasectomy and in whom two analyses had shown azoospermic semen reported by four centres. (Minimum follow up of three years to December 1983)*

Centre	Source of information	No of cases identified with numerous motile sperms	Manner of recognition	Vasectomy technique	Total No of vasectomies done at clinic before 31 December 1980	Estimated incidence of late recanalisation
Simon Clinic, Swindon	Mr J K Monro	3	Pregnancy	Excision and catgut ligation	21 919	1:7300
Marie Stopes Centre, London	Dr T Black	3	Pregnancy	Hyfrecator diathermy	18 000	1:6000
Croydon Vasectomy Clinic	Dr P Mortimer	1	Pregnancy	Excision and catgut ligation	4 043	1:4000
Elliot-Smith Clinic, Oxford	This series	6	Pregnancy	Hyfrecator diathermy	14 047*	1:2300

*This number performed between 31 March 1970 and 1 January 1981.

TABLE III—*Previously reported cases of recanalisation*

Reference	No of cases	Time from vasectomy to recognition of recanalisation	Manner of recognition	Details of vasectomy	Follow up details	Sperm counts after recanalisation	Findings at operation
Cases in which two aspermic counts reported							
Pugh and Hanley, 1969[6]	2	10 months 10 months	Pregnancy Pregnancy	Excision and catgut ligation 1 double back	Aspermic at two and three months		Unilateral recanalisation through scar
Livingstone, 1971[7]	1		Pregnancy		Two aspermic counts then "failed"		Reoperated (no details)
Esho *et al*, 1974[10]	1	11 months	Routine test		Two aspermic counts at two and three months	$39{\cdot}8 \times 10^6$	Unilateral recanalisation
Spencer, 1981[11]	1	4½ months	Pregnancy	Diathermy	Two aspermic counts at about three months		Successfully reoperated
Cases in which only one sperm count, or no details, reported							
Bunge, 1968[5]	1	6 years	Pregnancy			45×10^6	Bilateral recanalisation; unilateral sperm granuloma
Franzblau, 1973[8]	1	10 years	Pregnancy			Normal	Bilateral recanalisation; sperm granulomas
Esho *et al*, 1974[10]	2	16 months 14 months	Routine test Routine test		One aspermic count at six months One aspermic count at two months	7×10^6 $52{\cdot}3 \times 10^6$	Unilateral recanalisation
Jina *et al*, 1977[9]	1	3 years	Pregnancy			Normal	Unilateral recanalisation through scar
Hayashi *et al*, 1983[12]	1	3 years	Pregnancy			39×10^6	Unilateral recanalisation through scar

Discussion

The overall incidence of early failure (0·4%) at this clinic was comparable with that reported in previous British series.[1,2] The incidence of late failure, as defined here, was also similar to that experienced by other centres (table II), allowing for a minimum follow up of three years. Altogether we could find reports of 11 previous cases of apparent late recanalisation, but in only five were there details of two postoperative analyses showing azoospermia (table III).[5–12]

The aetiology of this phenomenon is not known. Early recanalisation is said to be associated with sperm granulomas, which may be asymptomatic.[13] These, however, were seen in only two of our six patients and in two of those reported on previously; histological examination otherwise showed the formation of one or more channels through scar tissue.

Although intraluminal diathermy was used in all our patients, the ligation method was probably used in most of the cases previously reported[6–12] and certainly in four cases reported in table II. Moreover, individual technique did not seem to be important as each man in our series had undergone vasectomy by a different surgeon; two of these surgeons had already performed well over 1000 operations. We could therefore find no obvious aspect of technique to explain late recanalisation.

Some of the less well documented cases of recanalisation have occurred up to 10 years after vasectomy. In the absence of any long term follow up studies with regular analyses of semen, it must be assumed that restoration of fertility may occur at any time.

We think that regular sperm counts to avoid late recanalisation being recognised only when a pregnancy occurs should not be undertaken routinely; the laboratory time and expense of these analyses together with the risk of creating unnecessary anxiety among many couples outweigh the possible benefit to a small number, in whom return of fertility might not even be detected in time.

Our six patients came from a population of 14 047 men who underwent vasectomy at one centre before the end of 1980.

Allowing, as seems reasonable from our data, a minimum of three years for the recanalisation to become apparent, this gives an incidence of one in 2300. Other possible estimates of incidence are given in table II. All must, however, be considered to be minimal estimates because, firstly, motile sperm may return to the ejaculate without the man seeking medical advice because no pregnancy follows; secondly, some pregnancies may not be reported to the operating surgeon or centre; and, thirdly—and most relevantly as two zero sperm counts after vasectomy have been widely believed to confirm permanent sterility—once a woman becomes pregnant there is reluctance to perform a sperm count in her partner for fear of precipitating marital breakdown.

Only Esho *et al* have reported an attempt to collect annual specimens of semen.[9] Only 215 out of 1000 men who had undergone vasectomy complied, in three of whom recanalisation was found to have occurred. These limited data suggest that late restoration of fertility could occur in as many as 1% of patients. The rarity, however, of reported conceptions occurring after vasectomy brings such a high incidence into question. A re-evaluation of this work is required, and, pending more data, our preferred estimate would be roughly one in 2000.

The important practical conclusions from this information are that, firstly, the rare possibility of late failure of vasectomy should be taken into account during preoperative counselling and in the wording of letters confirming the achievement of sterility (though it should be emphasised that abstinence remains the only method of birth control that is more effective than vasectomy judged to be successful by two zero sperm counts); and, secondly, if the partner of a man who has undergone vasectomy becomes pregnant a semen analysis should be performed before it is assumed that the fertile sperm came from another source.

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Blood thiamine and thiamine phosphate ester concentrations in alcoholic and non-alcoholic liver diseases

M DANCY, G EVANS, M K GAITONDE, J D MAXWELL

Abstract

Thiamine state was investigated in patients with alcoholic liver disease, patients with various non-alcoholic liver diseases, and controls using a direct technique (thiochrome assay) to measure thiamine, thiamine monophospate, and the active coenzyme thiamine pyrophosphate in whole blood after isolating the fractions by ion exchange chromatography. Overall nutrition was similar in all groups as assessed by anthropometry, and no patient had clinical evidence of thiamine deficiency.

There was no significant difference among the groups in mean concentration of any form of thiamine. The scatter was much greater in patients with alcoholic liver disease but only 8·7% had biochemical thiamine deficiency (defined as a blood concentration of the active coenzyme greater than 2 SD below the mean control value). An unexpected finding was of abnormally high total thiamine concentrations (>2 SD above the mean control value) in 17·4% of patients with alcoholic liver disease, the highest concentrations being found in two patients with severe alcoholic hepatitis and cirrhosis. The ratio of phosphorylated to unphosphorylated thiamine was calculated as an index of phosphorylation and, although the mean did not differ significantly among the groups, the range was greatest in alcoholic liver disease. The lowest ratios occurred in the two patients with severe alcoholic hepatitis, but neither had evidence of thiamine pyrophosphate deficiency.

Contrary to studies using indirect assay techniques, these results suggest that thiamine deficiency is unusual in well nourished patients with alcoholic liver disease. The new finding of unexpectedly high thiamine concentrations in some patients may be due to abnormalities of hepatic storage or release in liver disease, particularly in severe alcoholic hepatitis. There was no convincing evidence of impaired thiamine phosphorylation in any patients with liver disease. Conclusions from studies using indirect assays on the prevalence and mechanisms of thiamine deficiency in liver diseases may not be valid.

Departments of Medicine and Biochemistry, St George's Hospital and Medical School, London

M DANCY, MA, MRCP, senior registrar in medicine
G EVANS, BSC, research assistant
M K GAITONDE, MSC, PHD, senior scientist
J D MAXWELL, MD, FRCP, senior lecturer in medicine

Correspondence and requests for reprints to: Dr J D Maxwell, Department of Medicine II, St George's Hospital Medical School, London SW17 0RE.

Introduction

In affluent countries overt thiamine deficiency resulting in clinical syndromes such as Wernicke's encephalopathy, peripheral neuropathy, or beriberi heart disease is most often a