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Is ipsilateral testis mandatory for contralateral testicular deterioration encountered following spermatic cord torsion?

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Abstract Although deteriorating effects of unilateral spermatic cord torsion are generally accepted, the mechanism remains controversial. An experimental study was performed to evaluate the necessity of testicular and spermatogenetic material for contralateral testicular deterioration following unilateral spermatic cord torsion in rats. The animals were allocated to four groups: control, spermatic cord torsion, subepididymal orchiectomy, and spermatic cord torsion 14 days after subepididymal orchiectomy. The testes were removed on the 14th days and mean seminiferous tubular diameters and mean testicular biopsy scores were determined. Although contralateral testicular deterioration was more pronounced in the presence of testicular tissue, the absence of testicular tissue and/or spermatogenetic material did not prevent its occurrence. This is highly suggestive that autoimmune mechanism does not play a role in contralateral testicular damage following unilateral spermatic cord torsion.

Key word Spermatic cord torsion

Previous clinical and experimental studies have supported the idea that unilateral spermatic cord torsion may cause contralateral testicular damage and result in diminished fertility [4, 5]. However, explanations regarding the mechanism of contralateral testicular injury are still controversial.

One mechanism popularly proposed for contralateral testicular injury is an autoimmune phenomenon in which spermatogenetic cells enter the circulation after impairment of the blood–testis barrier and cause autoimmunization [12, 16]. According to this theory, the presence of a testis including spermatogenetic material is essential. On the other hand, it is reported that the presence of ipsilateral testicular tissue is not necessary for unilateral varicocele to reveal harmful effects on contralateral testis [10]. Therefore an experimental study was planned to investigate the necessity of the presence of ipsilateral testicular tissue for contralateral histological deterioration encountered following unilateral spermatic cord torsion.

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Materials and methods

Forty-eight adult male albino rats were used for the study. The rats were housed in a temperature- and light-controlled environment with ad libitum access to water and rat chow. The animals were randomly allocated to four experimental groups, each containing 12 rats. The surgical procedures were performed under ether anesthesia employing a sterile technique. Left-sided inguino-scrotal incisions were used, and clockwise torsions for 720° were applied.

Group 1. Control operations to expose the left testes and place a 4/0 atraumatic silk suture through the tunica albuginea were performed. The rats underwent bilateral orchiectomy 14 days after the control operations.

Group 2. Left testes were exposed and twisted. After maintenance of the spermatic cord torsion for a 24-h period with a silk suture, the testes were untwisted. The rats underwent bilateral orchiectomy 14 days after detorsion.

Group 3. The rats underwent left subepididymal orchiectomies. Fourteen days after subepididymal orchiectomy, reexploration (control operation) was performed. The animals underwent right orchiectomies 14 days after the control operations.

Group 4. The rats underwent left subepididymal orchiectomies. Fourteen days after the initial operations, remaining epididymis and spermatic cords were twisted. Following 24 h of torsion, epididymis and spermatic cords were untwisted. Fourteen days after the detorsion period, right orchiectomies were performed.

Testicular tissues were kept in Bouin's solution for histopathological examination. Histopathological evaluation was performed blind by

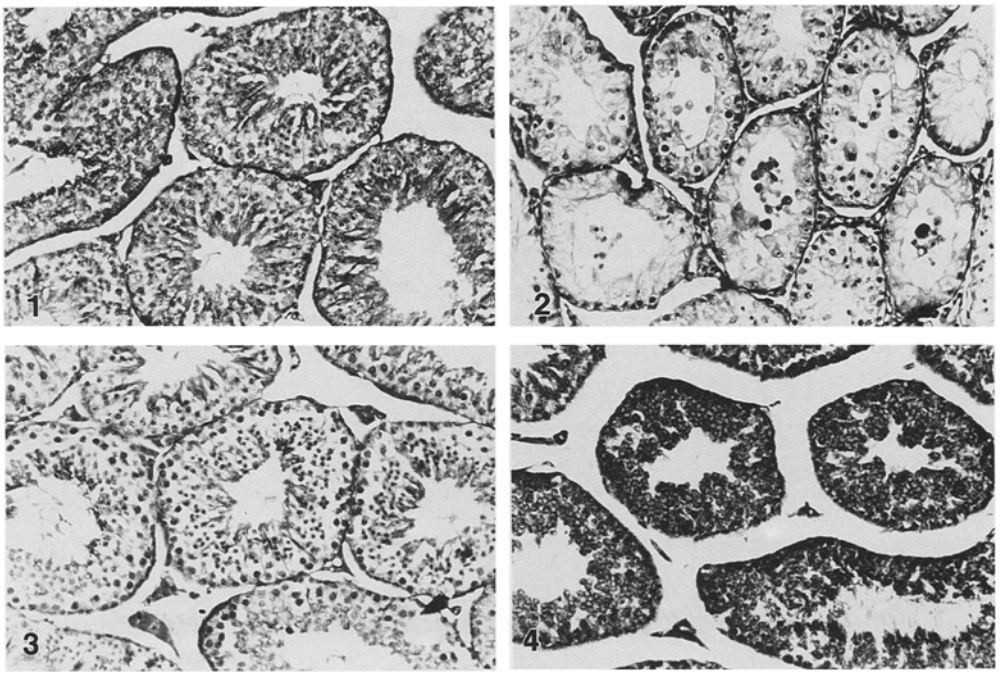

Fig. 1 Normal contralateral testicular architecture characterized by complete spermatogenesis 14 days after control operation. Group 1; HE, ×230. **Fig. 2** Histology of contralateral testis 14 days after unilateral spermatic cord torsion showing detoriorated testicular architecture with tubular atrophy, few spermatocytes and no spermatids. Group 2; HE, ×230. **Fig. 3** Normal contralateral testicular histology 14 days after control operation, performed two weeks after unilateral subepididymal orchiectomy. Group 3; HE, ×230. **Fig. 4** Contralateral testicular histology 14 days after unilateral spermatic cord torsion in the absence of ipsilateral testis showing diminished MSTD, many normal spermatids and no mature spermatozoa in most tubules. Group 4; HE, ×230

Table 1 Comparative mean (±SD) seminiferous tubular diameters (μm) of left and right testes

	Left testes	Right testes (μ ± SD)
Group 1	226.52 ± 6.60	222.96 ± 10.92
Group 2	–[a]	164.24 ± 25.95
Group 3	223.92 ± 5.34	222.16 ± 15.20
Group 4	224.84 ± 7.73	185.04 ± 13.41

[a] The testes could not evaluated because of severe necrosis

the pathologist of the research group. Two criteria consisting of mean seminiferous tubular diameters (MSTD) and mean testicular biopsy scores (MTBS) were used to evaluate testicular tissues.

To determine the MSTD, the 25 most nearly circular tubules were chosen in each biopsy section, and diameters of the tubules were measured using an ocular micrometer and a 10× objective.

Randomly chosen tubular cross sections with clear lumens were used for scoring. Maturity of tubular epithelium was graded using the modified Johnson testicular biopsy score [13]. Herein 40 tubules were scored under 40× objective and a score of 0–10 was given to each tubule according to epithelial maturation.

All measurements were recorded as mean ± SD. Statistical data were evaluated by SYSTAT and SPSS computer programs. Comparisons among the groups were performed with one-way analysis of variance, and a paired *t*-test was used to compare left- and right-side values. *P*-values lower than 0.05 were considered to be statistically significant.

Table 2 Comparative mean (±SD) testicular biopsy scores of left and right testes

	Left testes	Right testes
Group 1	9.8 ± 0.4	9.9 ± 0.3
Group 2	–[a]	5.9 ± 1.4
Group 3	9.7 ± 0.5	9.8 ± 0.4
Group 4	9.9 ± 0.3	9.2 ± 1.0

[a] The testes could not evaluated because of severe necrosis

Results

Forty rats, 10 in each group, were available for evaluation.

Left (ipsilateral) testes atrophied following the experimental procedure in group 2, and biopsies from remaining tissues showed necrosis without any testicular architecture. Therefore, MSTD and MTBS of left testes were not available for group 2.

MSTD did not reveal any significant difference ($P > 0.05$) between right and left testes in groups 1 and 3 (Table 1). However, MSTD of right testes in group 4 were significantly lower ($P < 0.05$) than the left testicular values obtained from testes before torsion procedure. MSTD of right testes were significantly lower ($P < 0.05$) in groups 2 and 4 than in groups 1 and 3. When the right testes of groups 2 and 4 were compared the values for group 2 were found to be significantly lower ($P < 0.05$) than those for group 4.

MTBS of right and left testes did not reveal any significant difference ($P > 0.05$) in groups 1, 3 or 4 (Table

2). The MTBS of right testes in group 2 were significantly lower ($P<0.05$) than those of the right testes of other groups (Figs. 1–4).

Discussion

Since unilateral testicular pathologies such as torsion, non-descent, hemorrhagic necrosis resulting from incarcerated hernia and varicocele also affect the contralateral testis, a common pathway for contralateral testicular deterioration seems possible.

The contralateral testicular effects of the above-mentioned pathologies have been investigated separately [10, 13–15]. The theories proposed include autoimmunity, underlying congenital defect affecting the contralateral testis and release of acrosomal enzymes [12, 16]. Additionally, recent studies performed by our group have revealed a decrease in contralateral testicular blood flow during unilateral spermatic cord torsion [11, 15]. Unilateral spermatic cord torsion also resulted in elevated levels of lactic acid and hypoxanthine in contralateral testicular tissue [2]. These findings led us to conclude that contralateral testicular damage may occur through a reflexive arc that results in a decrease in blood flow.

On the other hand, studies investigating the autoimmune mechanism revealed controversial results [3, 7]. For the autoimmune mechanism to be responsible for contralateral testicular damage, ipsilateral testicular tissue containing spermatogenetic material would have to exist. However, some reports have suggested that the presence of ipsilateral testicular tissue is not mandatory for varicocele to cause deterioration in the contralateral testis [8, 10]. Furthermore, Akgür et al. suggest that ipsilateral testis is not mandatory for the contralateral biochemical changes after unilateral spermatic cord torsion [1].

Although contralateral testicular effects of ipsilateral spermatic cord torsion are opposed by some authors [9], it is generally accepted that unilateral spermatic cord torsion causes contralateral testicular deterioration. The present study revealed that ipsilateral spermatic cord torsion caused contralateral testicular deterioration by way of diminished MSTD and MTBS.

Subepididymal orchiectomy did not cause any adverse effect on the contralateral testis. When the spermatic cord and epididymis were twisted in the absence of testicular tissue after a few weeks had been allowed for the disappearance of spermatogenetic material from the epididymis [6], contralateral testicular damage also occurred. Although MSTD was better than after spermatic cord torsion, MSTD was significantly lower after twisting of the epididymis and spermatic cord than in controls.

MTBS did not reveal a significant difference after twisting of the epididymis and of the spermatic cord. However, in some testes affected no statistically significant differences were found.

Spermatic cord torsion in the presence of ipsilateral testicular tissue produced more pronounced effects on the contralateral testis. Although the contralateral testicular effect was not as pronounced, torsion of epididymis and spermatic cord also had contralateral deteriorating effects in the absence of ipsilateral testicular and/or spermatogenetic material.

Therefore, the presence of ipsilateral testicular tissue seems not to be essential in the causation of contralateral testicular deterioration in the spermatic cord torsion model, but does seem to augment such damage. This makes mechanisms other than immunological, e.g., decreased blood flow, more likely for the contralateral testicular deterioration encountered following unilateral spermatic cord torsion.

References

1. Akgür FM, Kilinç K, Aktuğ T (in press) Is ipsilateral testis mandatory for contralateral biochemical changes after unilateral spermatic cord torsion? Urology
2. Akgür FM, Kilinç K, Tanyel FC, Büyükpamukçu N, Hiçsönmez A (in press) Ipsilateral and contralateral testicular biochemical changes after unilateral testicular torsion and detorsion. Urology
3. Anderson JB, Williamson RCN (1990) Fertility after torsion of spermatic cord. Br J Urol 65:225
4. Chakraborty J, Hikim APS, Jhunjhunwala JS (1985) Quantitative evaluation of testicular biopsies from men with unilateral torsion of spermatic cord. Urology 25:145
5. Cosentino MJ, Nishida M, Rabinowitz R, Cockett ATK (1985) Histological changes occurring in the contralateral testes of prepubertal rats subjected to various durations of unilateral spermatic cord torsion. J Urol 133:906
6. Ewing LL, Chang TSK (1987) The testis, epididymis and ductus deferens. In: Walsh PC, Gittes RF, Perlmutter AD,Stamey TA (eds) Campbell's urology. Saunders, Philadelphia, p 200
7. Fraser I, Slater N, Tate C, Smart JG (1985) Testicular torsion does not cause autoimmunization in men. Br J Surg 72:237
8. Green KF, Turner TT, Howards SS (1985) Effects of varicocele after unilateral orchiectomy and sympathectomy. J Urol 134:378
9. Henderson JA IV, Smey P, Cohen MS, Davis LP, Payer AF, Parkening TA, Warren MM (1985) The effect of testicular torsion on the contralateral testicle in prepubertal Chinese hamsters. J Pediatr Surg 20:592
10. Hurt GS, Howards SS, Turner TT (1987) The effects of unilateral, experimental varicocele are not mediated through the ipsilateral testis. J Androl 8:403
11. Kizilcan F, Bernay I, Tanyel FC, Büyükpamukçu N, Bekdik C, Hiçsönmez A (1992) Ipsilateral and contralateral testicular blood flows during unilateral testicular torsion by 133 Xe clearance technique. Int Urol Nephrol 24:515
12. Merimsky E, Orni-Wasserlauf R, Yust I (1984) Assessment of immunological mechanism in infertility of the rat after experimental testicular torsion. Urol Res 12:179
13. Salman T, Adkins ES, Fonkalsrud EW (1988) Morphologic effects of unilateral cryptorchidism on the contralateral descended testis. J Pediatr Surg 23:439
14. Tanyel FC, Ayhan A, Büyükpamukçu N, Hiçsönmez A (1989) The fate of testes following unilateral incarcerated inguinal hernia in adult rats. Br J Urol 64:176
15. Tanyel FC, Büyükpamukçu N, Hiçsönmez A (1989) Contralateral testicular blood flow during unilateral testicular torsion. Br J Urol 63:512
16. Williamson RCN, Thomas WEG (1984) Sympathetic orchidopathia. Ann R Coll Surg Eng 66:264