Urol Res (1996) 24:329–331

ORIGINAL PAPER

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Serum creatine kinase enzyme levels in the early diagnosis of spermatic cord torsion

Received: 16 February 1996 / Accepted: 12 April 1996

Abstract Delay in the diagnosis of spermatic cord torsion (SCT) is still a significant cause of testicular loss in children. The aim of this experimental study was to assess the diagnostic value of serum creatine kinase (CK) in the early period following SCT. Forty male rats were assigned randomly into five similar groups: group A, control; group B, sham, right testis exposed, manipulated, and blood sampling at 6th h; group C, right SCT, blood sampling at 2nd h; group D, right SCT, blood sampling at 4th h; and group E, right SCT, blood sampling at 6th h. CK and its isoenzymes were measured in the sera of all animals. All testes were removed and examined histopathologically. Significant increases in serum CK levels compared to control and sham groups were observed at 4 and 6 h following SCT. The major increase in CK was observed in the CK-MM isoenzyme fraction. Histologic pictures showed varying degrees of edema, vascular congestion, and hemorrhage in the testicular tissue, but no necrosis in any of the study groups. These results showed that serum CK levels in rats in the early period following SCT increase significantly before necrosis of testicular tissue. This may be of value as a diagnostic test, to corroborate findings from clinical studies.

Key words Spermatic cord torsion · Testis · Creatine kinase · Serum enzymes · Children

Introduction

Early diagnosis of spermatic cord torsion (SCT) is mandatory for a successful outcome. Delay in the diagnosis, however, is still a significant cause of testicular loss in children. Doppler ultrasonic flow detector and radioisotope scanning have been the main diagnostic tools; yet neither are without limitations [2, 8]. The aim of this experimental study was to assess the diagnostic value of serum creatine kinase (CK), which has been shown previously to increase in experimental testicular torsion, and to verify any possible relation between this increase and early pathologic changes in the testicular tissue [3, 4]. The isoenzyme fractions of CK were also investigated to further elucidate the source of this increase in CK following SCT.

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Materials and methods

Forty male albino rats, weighing 200 g on average, were used in the experiment. Rats were diagnosed randomly into five similar groups with eight animals in each: group A: control, anesthesia alone, and blood sampling at 6th h; group B: sham, right testis exposed, manipulated, and blood sampling at 6th h; group C: right spermatic cord torsion, blood sampling at 2nd h; group D: right spermatic cord torsion, blood sampling at 4th h; and group E: right spermatic cord torsion, blood sampling at 6th h. Experiments were performed with the rats under intraperitoneal ketamine anesthesia (50 mg/kg). The right testis was exposed via a longitudinal scrotal incision. Following a clockwise rotation of 1080°, it was fixed to the dartos from the epididymis with a 5/0 silk suture in the torsion groups. No torsion was applied to the control and sham groups. CK levels were calculated in the sera of all animals. Its isoenzymes (CK-BB, CK-MB, CK-MM) were also determined at sham and at 6 h in the torsion groups. The results were compared statistically between groups using the *t*-test. To relate any structural changes in the testicular tissue to the possible increases in the enzyme levels, all testes were examined histopathologically.

Results

Significant increases in serum CK levels compared to control group were observed at 4 and 6 h following SCT. There was also a significant difference between the sham and 6-h torsion groups (Fig. 1). The major

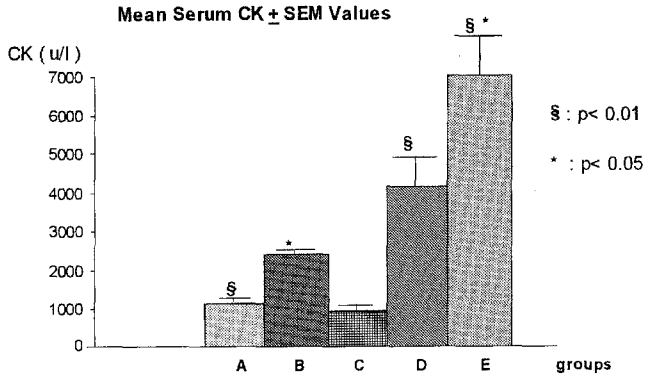

Fig. 1 Mean (±SEM) CK values of study groups (*CK* creatine kinase, *SEM* standard error of the mean). Torsion groups (*C, D, E*) were compared with both control (*A*) and sham (*B*) groups

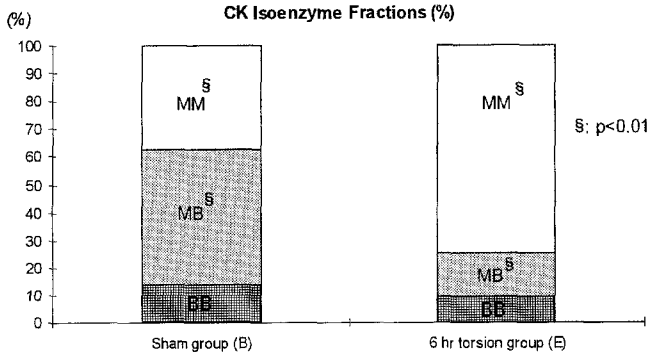

Fig. 2 Comparison of CK isoenzyme fractions in the sham (*B*) and 6-h torsion (*E*) groups

increase in CK was observed in the CK-MM isoenzyme fraction (Fig. 2). Histopathologic pictures showed variable degrees of edema, vascular congestion, and hemorrhage in the testicular tissue in the torsion groups, but no clue as to the loss of viability and necrosis. The specimens from the control and sham groups were completely normal. The histopathologic examinations failed to demonstrate any specific macroscopic or microscopic findings pertinent to any specific group, which made histologic grading redundant. There were also no significant differences in seminiferous tubule diameter between the groups.

Discussion

Many surgeons still advocate emergent exploration of all cases of acute scrotal inflammation, although there is a growing skepticism as to the necessity of this type of management in an era of minimally invasive surgery [6, 7]. Scans using ^{99m}Tc-pertechnetate have proved sensitive in differentiating surgical from nonsurgical causes of acute scrotum, but in practice it may not be so easy to perform scrotal scintigraphy properly within an acceptable time [5, 7, 8]. Doppler flow detectors are more readily available and the technique takes less time to perform, but, due to the high error rates, their help is complementary at best [1, 6, 10]. The limited assistance from diagnostic modalities, and the grave outcome of the involved organ when the treatment is delayed, have probably been the main factors in the widespread acceptance so far of the early surgical exploration method in boys with acute scrotal swellings [7].

There are hundreds of different enzymes present in the human body, synthesized intracellularly, and most of them carry out their functions within the cells in which they are formed. Enzymes are normally present in the serum with low activities only, and it is sometimes possible to infer the location and nature of pathologic changes in the tissues by measuring changes in the activities of these enzymes in disease states. There is a considerable body of data regarding the enzymatic changes in the hypoxic states of various tissues [9]. However, testicular tissue has less frequently been subjected to such enzymatic studies [3, 4].

CK catalyzes the transfer of high-energy phosphate from creatine phosphate to ADP, a reaction necessary for resynthesis of ATP, which is the primary energy source in the organism, especially in the muscle tissue [9]. It is suggested that ischemia and infarction of the cremaster muscle following spermatic cord torsion is the source of increased serum CK levels, which is released from tissue [3]. The significant increase in the CK-MM isoenzyme fraction following spermatic cord torsion in the present study, which was shown for the first time, supports this hypothesis. The MM fraction of CK (CK-3) is known to predominate in skeletal and cardiac muscle, while CK-BB (CK-1) predominates in brain, prostate, gut, lung, bladder, uterus, placenta, and thyroid [9].

We have shown that serum CK levels in rats increase in the early period, at 4 h, following SCT before necrosis of testicular tissue occurs. This finding is of vital importance because any diagnostic method in testicular torsion is valuable only if it helps in the salvage of the testis. Together with the degree of torsion, 10 h seems to be the critical time point for irreversible structural changes to occur in the testicular tissue following SCT [11]. It is shown in this study that serum CK levels increase not as early as 2 h, but at 4 h following torsion. However, the histopathologic specimens even at 6 h showed no signs of necrosis, instead there were varying degrees of edema, vascular congestion, and hemorrhage in the testicular tissue.

Disorders of muscles due to infection, inflammation, degenerative changes, drugs, and alcohol may also cause enzyme leakage from myocytes [9]. However, it has been clearly shown in two different previous studies

that in experimental epididymitis there was no significant increase in serum CK levels, which is very important in the differential diagnosis of SCT [3, 4].

In conclusion, the results of this experimental study support the previous findings regarding the value of CK as an early indicator of SCT, and add to them by showing that the increase in CK levels occurs before necrosis of testicular tissue, and that CK is most probably released from the cremaster muscle according to the changes in the isoenzyme compositions following torsion.

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