EXERCISE INDUCED ACUTE RENAL FAILURE IN 4 CASES

Pages with reference to book, From 314 To 316

Q.A. Shaikh, S.A.J. Naqvi (Department of Nephrourology, Jinnah Postgraduate Medical Centre, Karachi.)

Acute exertional rhabdomyolysis is a syndrome that occurs in apparently healthy individuals as a result of performing strenuous exercise to which he is not conditioned. Exercise induced acute renal failure after rhabdomyolysis is well reported[1-4]. Four cases of acute renal failure induced by myoglobinuria due to unaccustomed heavy exercise are reported.

CASE I

A 44 years male, cashier, previously in good health was referred from cardiology department on 26th February, 1990. He was drowsy and short of breath and was unable to stand due to pain in legs. Twelve days prior to the admission he was punished with 100 nonstop sit ups after which he collapsed and developed severe pain and cramps in the lower limbs and was, therefore, bedridden. Fle passed dark brownish coloured urine and became severely oliguric with associated nausea, vomiting and anorexia. There was no past history of diabetes mellitus or hypertension. Physical examination revealed pulse of 88/minute and blood pressure of 150/100mmHg. JYP was not raised, bilateral basal crepts were present, abdomen was tense and his quadriceps were tense and tender. His hemoglobin was 12.3G/dl, BUN 92mg/dl, serum creatinine 9.2mg/di, serum sodium 124mEq/L, serum potassium 6.0mEq/L, total bilirubin was 0.8mg/dl, SGOT 22 IU/L, SGPT 31 IU/L, LDH 996 U/L, CPK 108 U/L, HBsAg was negative. Flemoglobin was present in urine. His kidneys were normal on ultrasonography and he refused renal biopsy. Peritoneal dialysis was started on the same day and continued for 6 days with maintenance of fluid and caloric requirement. Arterio-venous shunt in left leg was made on 5/3/90 and he was then hemodialysed on three occasions. His hypertension was controlled by Tab. Aldomet and Cap. Adalat. His renal functions returned to normal gradually and tenderness of quadriceps vanished. FIe was discharged on 12/3/90 on Tab. Aldomet for the control of hypertension.

CASE II

a 25 years male, taxi driver, previously in good health, was admitted on 27th February, 1990 with severe pain in both limbs and inability to walk. Prior to his illness he was punished with 300 nonstop situps after which he developed severe pain and tenderness in lower limbs. He passed dark brown urine 8 hours later. Urine output of patient declined for 2 days but he continued self hydration and passed more than 2 litres of urine/24 hours. He also developed anorexia and vomiting. There was no past history of diabetes mellitus or hypertension. Physical examination revealed a pulse of 80/minute, blood pressure 110/70 mmHg. JPV was not raised, chest was clear and there was tenderness over abdomen and lower limbs. His hemoglobin was 11.8 G/dl, BUN 61 mg/dl, serum creatinine 6.2 mg/dl, serum sodium 132 mEq/L, serum potassium 4.9 mEq/L, total bilirubin 0.8 mg/dl, SGOT 18 IU/L, SGPT 25 IU/L, HBsAg was negative. Hemoglobin was present in urine. His kidneys were normal in size on ultrasonography and left renal biopsy done on 28/2/1990 was normal. Arteriovenous shunt in left leg was made on 3/3/90 and he was hemodialysed on four occasions. He remained normotensive during his stay at hospital and was discharged on 9/3/90 as his renal functions returned to normal.

CASE III

A 25 years male, vaccinator, was admitted on 29/10/90 with complaints of severe pain in the legs and less urine since 4 days. Prior to these complaints he had diarrhoea and was punished by 200 nonstop situps. After punishment he felt severe pain in quadriceps muscles which were painful to touch. He passed dark brown coloured urine and later became aneuric with swelling on face. There was no past history of diabetes mellitus or hypertension. Physical examination revealed generalized oedema of

body. The pulse was 80/minute and blood pressure 110/70 mmHg. JVP was not raised, there was no visceromegaly. Chest was clear, quadriceps were very tender and limb raising was impossible. His hemoglobin was 12.4 G/dl, BUN 50 mg/dl, serum creatinine 4.9 mg/dl, serum sodium 136 mEq/L, serum potassium 4.3 mEq/L, total bilirubin 0.5 mg/dl, SGPT 1000 IU/L, SCOT 607 IU/L, LDH 236 U/L, CPK 426 U/L, HBsAg was negative. Hemoglobin was present in urine. On ultrasonography his kidneys were normal in size. Left renal biopsy on 30/10/90 showed acute tubular necrosis. As his BUN was rising so ateriovenous shunt was made in left leg on 31/10/90 and he was hemodialysed on six occasions with 2 units of blood. He started passing urine on 31/10/90 which increased gradually. He was discharged on 25/11/90 when his kidney functions became normal.

CASE IV

A 37 years male, jobless, previously in good health was admitted on 6/11/90 with complaints of pain in both thighs. He was punished by nonstop situps for one hour, 13 days prior to admission. After exercise he passed dark brown urine and developed loose motions which stopped on its own. He was rehydrated immediately at home and started passing urine. There was no past history of diabetes mellitus or hypertension. His physical examination revealed a pulse of 82/minute and blood pressure 130/80 mmHg. JYP was not raised. Chest was clear and there was no visceromegaly. Quadriceps muscles were tense and tender. His hemoglobin was 13.3 G/dl, BUN 68 mg/dl, serum creatinine 7.7 mg/dl, serum sodium 14 (??) mEq/L, serum potassium 3.0 mEq/L, total bilirubin 0.4 mg/dl, SGOT 37 IU/L, SGPT 45 IU/L, LDH 315 U/L, CPK 398 U/L, HBsAg was negative. Hemoglobin was present in urine. On ultrasonography his kidneys were normal in size and left renal biopsy on 9/11/90 showed acute tubular necrosis. He was discharged on 16/i 1/90 as his kidney functions became normal.'

DISCUSSION

Since By water and Beall[5] have shown the association of myoglobinuria and oliguric renal failure with crush syndrome of World War Two, many investigators have worked on it and many, theories have been put forward for its explanation. Arce et al[6] concluded that oliguria associated with myoglobinuria acute renal failure is due to cessation of glomerular filtration. Heme pigment may be associated as potentiating factor in the development of renal failure[7]. Maroney[8] showed that the lumen of collecting tubules, distal tubules and ascending limbs of Henle are plugged by precipitated proteins and plugs are finally held in place by edema fluid. Ono in 1953[7] after studying 46 ice skaters and 10 marathon runners described that sporadic exertion induced skeletal muscle necrosis and represents an exaggeration of normal physiologic response to severe exertion as minute amounts of myoglobin in the urine appears by a normal phenomenon following strenuous exercise. Some workers[9-11] have described hypokalemia as the cause of muscle degeneration i.e., rhabdomyolysis and myoglobinuria, however, when acute renal failure develops, serum potassium level rises rapidly. Howenstine[12] described renal ischaemia and dehydration as predisposing factors to acute renal failure following myoglobinuria. Renal flow decrease in exercise by many fold during dehydration and increased atmospheric temperature has also the same effect. Heavy exercise, crush injuries, burns, electric shock, McArdles disease or myophosphorylasc deficiency glycogenosis, grand mall seizures and various thugs are known causes of myoglobinuria. Intravenous hemoglobin has been shown to have vasoconstricting properties[12]. Whether myoglobin similarly causes vasoconstriction is uncertain. ATP, aldolase, lactic acid and transaminases are released during rhabdomyolysis and one of these substances may have adverse cifect on kidneys. Acute renal failure occurs in those persons who are not accustomed to heavy exercise and severity depends upon the state of hydration prior to exercise and temperature[13]. The above reported 4 cases of exercise induced acute renal failure were treated during 1990 at Jinnah Postgraduate Medical Centre. In all cases there was no family history of muscle cramps, exercise in-

tolerance or dark urine so diagnosis of idiopathic paroxysmal myoglobinuria is unlikely. There was also no previous history suggestive of underlying kidney disease or burns, seizures, crush injuries and difficulty or pain on exertion. The predisposing factors and the onset of illness were very similar in each case which occurred after situps. All the patients were physically inactive before the heavy exercise . There was no history of hypertension, cardiac, renal or muscular disease nor there was any history of drug ingestion. Pulse and blood pressure were normal in 3 cases except case I at the time of examination who was also drowsy; all others were well oriented. There was no skin rash or G. I.T. haemorrhage. Quadriceps muscles of all the patients were tender, all sweated profusely during exercise and all passed dark coloured urine while case III became totally anuric because he was dehydrated due to loose motions prior to punishment. In all cases liver function tests were normal while SGPT and LDH of case I and III were increased. These two patients were seriously ill, case III was totally anuric. Kidney function tests were abnormal so dialysis was performed in three cases. Renal biopsies of case III and W showed acute tubular necrosis and that of case II was normal while it was not done in case I. In follow up their renal function became normal. Our case I and III were seriously ill which was reflected by raised level of CPK and LDH which shows necrosis of quadriceps muscles and myoglobin was released. Case III was already dehydrated due to loose motions and sweated profusely during situps so he became totally anuric after passing dark coloured urine while case IV was rehydrated immediately and he developed less severe symptoms. He was not oliguric and did not need hemodialysis. GFR decreases by 51% if dehydrated person performs exercise while in well hydrated person it decreases by only 19%[7], so by rehydrating person immediately after heavy unaccustomed exercise, acute renal failure can be prevented as shown by Bywater[14].

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