ACUTE RENAL FAILURE TREATED WITH PERITONEAL DIALYSIS

(A CASE REPORT)

by

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Summary

A young man of 28 yrs. developed rigor, hyperpyrexia, hypotension and acute renal failure following reaction to Dextrose saline transfusion. He required peritoneal dialysis. While he was recovering from acute renal failure he suddenly died of pulmonary embolism.

Reaction to saline transfusion has not been uncommon recently at Bir Hospital and it requires a through investigation as to its cause. This case is being reported to bring to notice this serious reaction to saline transfusion and to put to record the first use in Bir Hospital of Peritoneal Dialysis in the management of Acute Renal Failure.

Case Report

Mr. P.C., aged 28 yrs. M, (In Pt. No. 469) was admitted to Bir Hospital, ENT department on 23.1.028 with Lt. side Acute Mastoiditis. Modified radical mastoidectomy was done under GA on 17.2.028. After operation his general condition remained good, PR: 90/mt, and BP was 115/80. Preoperative urine test showed a trace of albumin. About ten hours after operation following transfusion of 5% Dextrose saline he suddenly developed rigor and rapidly became hyperpyrexia (temp: 105.4 F) and hypotensive (systolic BP of 30 mm Hg). Subsequently he became oliguric. Temperature dropped to 102.6 F after four hours of cold sponging and then to 99 F after 8 hours. During that period an attempt was made to raise the blood pressure by administration of Hydrocortisone and continuous Binodrenal infusion. He also received one litre of Sod. lactate solution, 540 ml of blood and 100 ml of Peristone N. He was referred to our "Intensive Care Team" on 19.2.028. By this time he had been hypotensive for thirty eight hours and was anuric. However his periphery was warm and dry. PR was 180/mt, BP: 80/60. Blood urea 158 mg per cent, Serum electrolytes: Na+ 134, K+ 5.2, and Cl- 92 mEq/L respectively; Urine: S.G. 1030 and showed slight albuminuria and microscopic haematuria.

Management

After clinical assesment of the patient a nylon catheter (Portex: Intravenous set) was introduced to Superior ven cava via left Median cubital vein percutaneously and it was connected to a saline Central Venous Pressure manometer. At that time the CVP was 17 cm. of water.

It was thought that the patient was not hypovolaemic and the shock state was due to reaction to the saline transfusion and he was now in Acute Renal Failure following prolonged hypotension. It was decided to boost up the cardiac efficiency by digitalisation and to treat acute renal failure with restriction of protein and the fluid to 500 ml plus any visible loss of fluid in the form of urine, stool, vomiting or sweating. And it was aimed to supply over 1000 calories to prevent breakdown of patients' own protein by carbohydrate only in the form of 30 per cent and 50 per cent Dextrose solution infused into the Superior vena cava. Antibiotics (Penicillin and Streptomycin) which the patient was receiving before was continued at the reduced doses.

His progress in the following days is shown briefly in the chart. On the third day (20.2.028) he was noted to be icteric. His serum bilirubin was 18mg per cent and both SGPT and SGOT was very much raised. The icterus was most probably due to hepatic damage (centrilobular necrosis) caused by prolonged shock state (Sherlock, S. 1963). But the high level of the serum bilirubin and the fall of haemoglobin from 10 to 8 G per cent in a few days' time suggested to us the possibility of the haemolytic process and the Prednisolone was prescribed. (Berlyne, G.M., 1970).

He remained anuric, icterus was increasing and the blood urea rapidly increased to 412 mg per cent on the fourth day. At this stage it was decided to undertake peritoneal dialysis. 1.36 per cent Dextrose solution with the following composition was used : Na + 141, Ca + + 3.6, K + 3, Cl - 101 and lactate 44.6 mEq/L respectively. To each litre of the above dialysis fluid was added Heparin 1.5mg and Tetracycline 12.5mg. Baxter laboratory nylon catheter was introduced to the peritoneal cavity through a stilet by a small incision in the midline below the umbilicus and its tip was positioned to oneside in the pelvic cavity. Plexitron Y-type 'dianeal' solution administration set (Baxter) was used and dialysis was conducted as described by Maxwell et al (1959) and Thomson et al (1964). Total of twenty litre was dialysed in two sessions of twelve and eight hours each. After two dialysis, on the sixth day of the renal failure his blood urea dropped to 250 mg per cent and his icterus diminished significantly. At this stage he went into diuretic phase. The urine volume rapidly increased from 540 ml to about three litre and the blood urea dropped to normal level on the eleventh day. During this diuretic phase the excess of

fluid was given orally in the form of electrolyte solution with the following composition: Sodium chloride 3 G, Potassium chloride 2 G and Sodium bicarbonate 1 G per every litre of water. Vitamin supplement was given and the increase food intake was encouraged.

On the fourth day he however developed gastro intestinal bleeding as manifested by malaena. This is not an unknown complication of acute renal failure (Cecil and Loeb 1967). Steroid was stopped. It was possible that the steroid therapy has aggravated the gastro-intestinal bleed. The G.I. bleeding and the macroscopic haematuria continued in diminishing rate and he was given a pint of fresh blood on the eleventh day.

On the eleventh night he developed a big pulmonary embolus. He suddenly became restless, orthopneic; the movement of the rt. side of the chest was restricted and there were coarse rales in this side. Next day he developed haemoptysis. X-ray chest showed the Rt. hemidiaphragm was raised and the Rt. lung field was translucent (ground glass appearance) compared to the left and there was prominence of the main pulmonary vasculature in the same side. ECG however did not show any evidence of RV strain. Because of the gastro-intestinal bleed and haematuria anti-coagulant therapy was deferred. On the twelfth day however, he suffered a second and fatal bout of pulmonary embolus.

Discussion:

Acute renal failure may occur in primary renal diseases like acute glomerulonephritis, acute pyelonephritis, acute polyarteritis and acute renal lupus; or following obstructive lesion of the urinary tract like enlarged prostate or sulphonamide crystalluria; or due to nephrotoxic drugs like sodium chlorate. But the great bulk of cases are due to acute conditions which are believed to be initiated by a spell of reduced blood flow (leading to acute tubular necrosis). This includes trauma, blood loss, burns, water and salt depletion, incompatible blood transfusion and severe systemic infection. It also includes other medical causes of shock, vascular calamities to renal circulation like dissecting aneurysm, and aortic surgery. In civilian hospital, the leading causes of the acute renal failure are most probably the obstetric cases like septic abortion, concealed haemorrhage, pre-eclampsia etc. It also occurs in leptospirosis and may occur in severe falciparum malaria (Sitaparija, V. et al, 1967) and after administration of low molecular-weight dextran in patients with advanced atherosclerotic vascular disease (Mailloux, L. et al, 1967).

The diagnosis of acute renal failure depends upon being aware of the conditions that can lead to it and the measurement of daily urine volume and serial blood urea. "The demonstration of a progressive rise in the concentration of blood urea by serial determinations is the most simple and reliable method and is of greater value than the measurement of the daily urine volume or its composition. Significant progressive urea retention does not occur without a decrease in the renal power of elimination." (Robson, J.S., 1965). In places where there is no laboratory facilities measurement of daily urine volume and the rough estimation of blood urea with Azostix (Ames Co.) should be used.

A daily urine volume of less than 400 ml and the rise in blood urea indicates acute renal failure.

In health, homeostasis is a dynamic balance between diet, catabolism and renal function. In oliguric phase the renal function is virtually eliminated and there is progressive retention of water, non-protein nitrogen, potassium and phosphates which are produced by the breakdown of the tissue protein.

Once established the clinical course of acute renal failure is remarkably constant regardless of the etiology. Two distinct phases can be recognised: 1) Phase of renal insufficiency - oliguric phase, and 2) Recovery or diuretic phase.

Meticulous attention to the metabolic control in acute renal failure and the judicious use of peritoneal dialysis or haemodialysis will enable the majority of the patients to survive. Even recovery from acute renal failure due to "Irreversible" glomerular disease have been reported (Richard, PP, et al, 1968).

Though, peritoneal dialysis is not as efficient a method of solute removal as haemodialysis it is simple, reliable and effective in treating severe renal failure and this method can be undertaken in any hospital and could largely be managed by the nursing staff (Maxwell et al, 1959 and Thomson et al, 1964).

Peritoneal dialysis was first used for treatment of renal failure by Cantor (1923). However it became popular only after the publication of the experience of Maxwell et al, in 1959. They described the method and procedure of intermittent peritoneal dialysis with technique using nylon catheter and commercially prepared solution. The detail procedure, complications, indications and contraindications have been described by Maxwell et al and Thomson et al.

Peritoneal dialysis has also been used successfully over prolonged period in patients awaiting renal transplantation (Cohen et al, 1968).

Acute renal failure is not uncommon at Bir Hospital. So far they have been either referred to better centres outside the country for treatment or have ended fatally. Judicious treatment with frequent use of peritoneal dialysis in future should save these lives.

"In less highly developed countries where the provision of resources for maintenance haemodialysis is an unjustifiable extravagance, acute renal failure deserves more attention (BMJ, 2, 1971)."

Unfortunately this patient died of massive pulmonary embolism, which is not a complication of dialysis. Early mobilisation and perhaps use of less concentrated dextrose solution for I.V. use in future may help us to prevent this complication.

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