

Haemodialysis in the treatment of lactic acidosis associated with acute hepatic and renal failure

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APPROXIMATELY one third of endogenous lactate is normally metabolized in the liver. In acute liver failure, accumulation of non-metabolized pyruvate and lactate results in high blood pyruvate and lactate levels (Thölen *et al.*, 1966). Often in this condition it is possible to control the arterial pH by the administration of sodium bicarbonate or THAM; if however there is also renal failure or marked secondary aldosteronism, the administration of large amounts of sodium and fluid is limited. Under these circumstances, haemodialysis offers a means of correcting the acidosis without increase of extracellular volume and with removal of lactate.

Case report

A. H. P., a 31-year-old cook was admitted to a district hospital with severe dyspnoea at rest. This had started suddenly after 8 days of abnormal fatigue. The patient was known to drink eight to ten bottles of beer daily. Hyperventilation and cyanosis of the lips were observed in the absence of pulmonary findings. The patient was jaundiced with a large liver 7 cm below the ribs. The blood pressure was 140/60 mmHg, pulse rate 96/min. Serum glutamin-pyruvate-transaminase (SGPT) was 870 WU (Wroblenski), prothrombin-time was 15% (Quick). The following day the blood pressure (BP) fell to 70/0 mmHg and could not be corrected by administration of Dextran, angiotensin II and 200 mg hydrocortisone i.v. The patient was then transferred to the Medical Department of the University of Basel.

On admission, the patient was drowsy and confused, but responded to simple commands. Hyperventilation, peripheral cyanosis and ankle oedema were present and at both pulmonary bases now there were crepitations. Central venous pressure (CVP) was 31.5 cm H₂O and arterial BP 85/40 mmHg. Serum sodium was 127 mEq/l and was considered to be low because of overhydration. The patient was anuric after an excretion of 300 ml during the previous 24 hr. Blood urea was 70 mg/

* II, V, VII-index, sum of blood clotting factors II, V and VII in percent. Normal value 260–300 (Colombi, 1969).

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100 ml, the II, V, VII-index* 77, blood ammonia 202 γ /100 ml (Conway), SGOT 2400 WU and serum bilirubin 3.2 mg/100 ml (Schellong & Wende) indicating a severe liver failure with impending hepatic coma. The blood pH of 6.94, Paco₂ of 15 mmHg with a base excess exceeding –25 mEq/l were explained by a serum lactate of 150 mg/100 ml. The electrocardiogram showed partial right bundle branch block (Fig. 1). On the chest X-ray, cardiomegaly, distension of the pulmonary veins and some patchy infiltrations were seen (Fig. 2).

A Scribner shunt was inserted into the right forearm and an 8-hr haemodialysis was performed with a Travenol RSP Dialyser using an ultraflow 100 coil. Further characteristics of this dialysis are given in Table 1.

TABLE 1. Dialysis characteristics

Dialyser type	Travenol RSP
Coil	Ultraflow 100
Blood flow	270–320 ml/min
Venous pressure	80–110 mmHg
Dialysate flow	600 ml/min
Duration	8 hr
Dialysate concentration:	
Na	132.0 mEq/l
K	1.3 mEq/l
Cl	106.0 mEq/l
Mg	1.0 mEq/l
Ca	3.5 mEq/l
Acetate	35.0 mEq/l
Glucose	200.0 mg/100 ml

Rapid improvement of respiration, increase of arterial BP to 145/75 mmHg, decrease of CVP to 22 cm H₂O, disappearance of mental confusion and a dramatic improvement in arterial blood gases, blood pH, serum lactate, serum sodium and urea were observed (Table 2) during dialysis. Because of persisting anuria, another 5-hr haemodialysis was done the next day. On the third day after admission, a diuresis started with 190 ml/24 hr (Fig. 3). However, the most promising features of improvement were a consistent increase of the II, V, VII-index and a blood lactate keeping within normal limits. SGPT decreased from 3000 WU to 36 WU within 10 days. When on the fifth day after

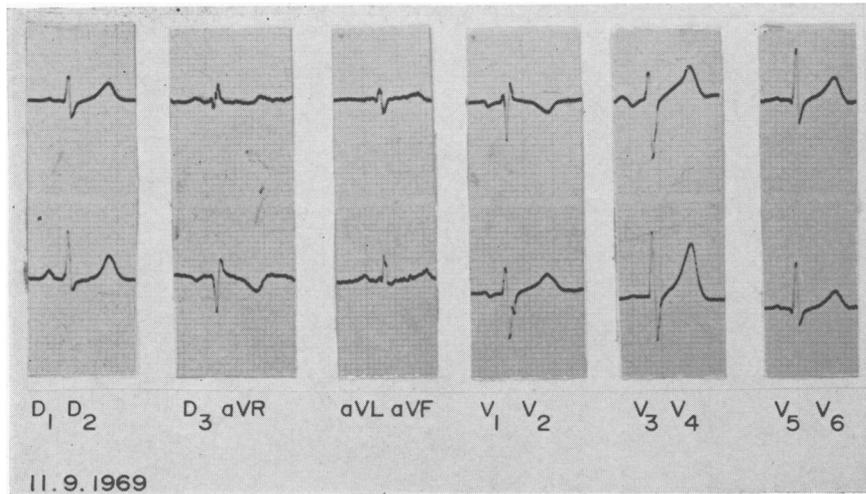


FIG. 1. Patient A. H. P., 31, electrocardiogram showing partial right bundle branch block.

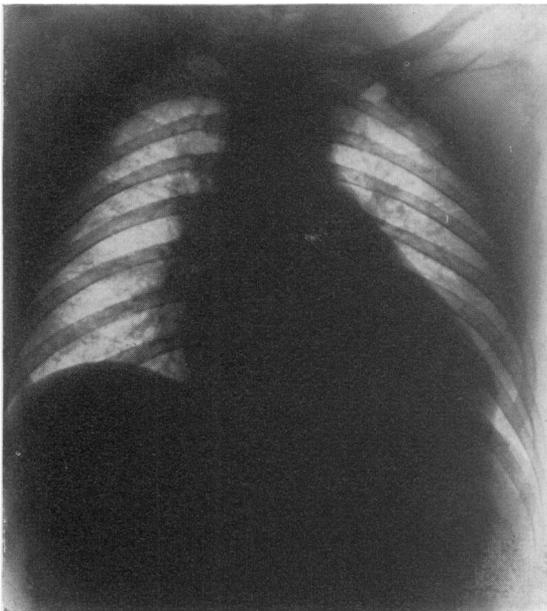


FIG. 2. Patient A. H. P., 31, chest X-ray showing cardiomegaly, distension of the pulmonary veins and some patchy infiltrations.

admission the prothrombin-time had reached 50%, laparoscopy and liver biopsy were carried out. Because of large adhesions the liver surface could not be seen. The biopsy specimen showed extensive centrilobular haemorrhagic necrosis, associated with mild fatty degeneration. A repeat biopsy 18 days later showed massive regeneration. At that time the prothrombin-time was 72%, SGPT 18 WU,

TABLE 2. Physical and chemical data before and after 8-hr haemodialysis for severe lactic acidosis

	Before dialysis	After dialysis
pH	6.94	7.50
Paco ₂ (mmHg)	15.0	30.2
HCO ₃ (mEq/l)	Not definable, low	
Base-excess (mEq/l)	> -25.0	+1.5
Lactate (mg/100 ml)	150.0	17.0
Urea (mg/100 ml)	75.0	44.0
Na (mEq/l)	127.0	130.0
K (mEq/l)	4.5	3.4
Blood pressure (mmHg)	95/60	145/75
Central venous pressure (cmH ₂ O)	+31.5	+22.0
Body weight (kg)	76.1	73.1

SGOT 17 WU, blood urea 17 mg/100 ml, creatinine 0.7 mg/100 ml, C_{Inulin} 106 ml/min, C_{PAH} 546 ml/min, filtration fraction 20% and T_m PAH 75 mg/min. Chest X-ray was normal and the patient was discharged 23 days after admission.

Discussion

Correction of lactic acidosis by haemodialysis has been reported in patients with phenformin-induced lactic acidosis (Ewy *et al.*, 1963; Maher & Schreiner, 1967; Westervelt, 1966). Haemodialysis has not yet been recommended in the treatment of lactic acidosis occurring in acute hepatic failure. This treatment however has to be considered for the control of the severe lactic acidosis that often occurs in severe hepatic failure.

Haemodialysis as a treatment for endogenous hepatic coma has not been very successful (Trey *et al.*, 1966). This, however, does not mean that this procedure is useless in the treatment of severe

Case reports

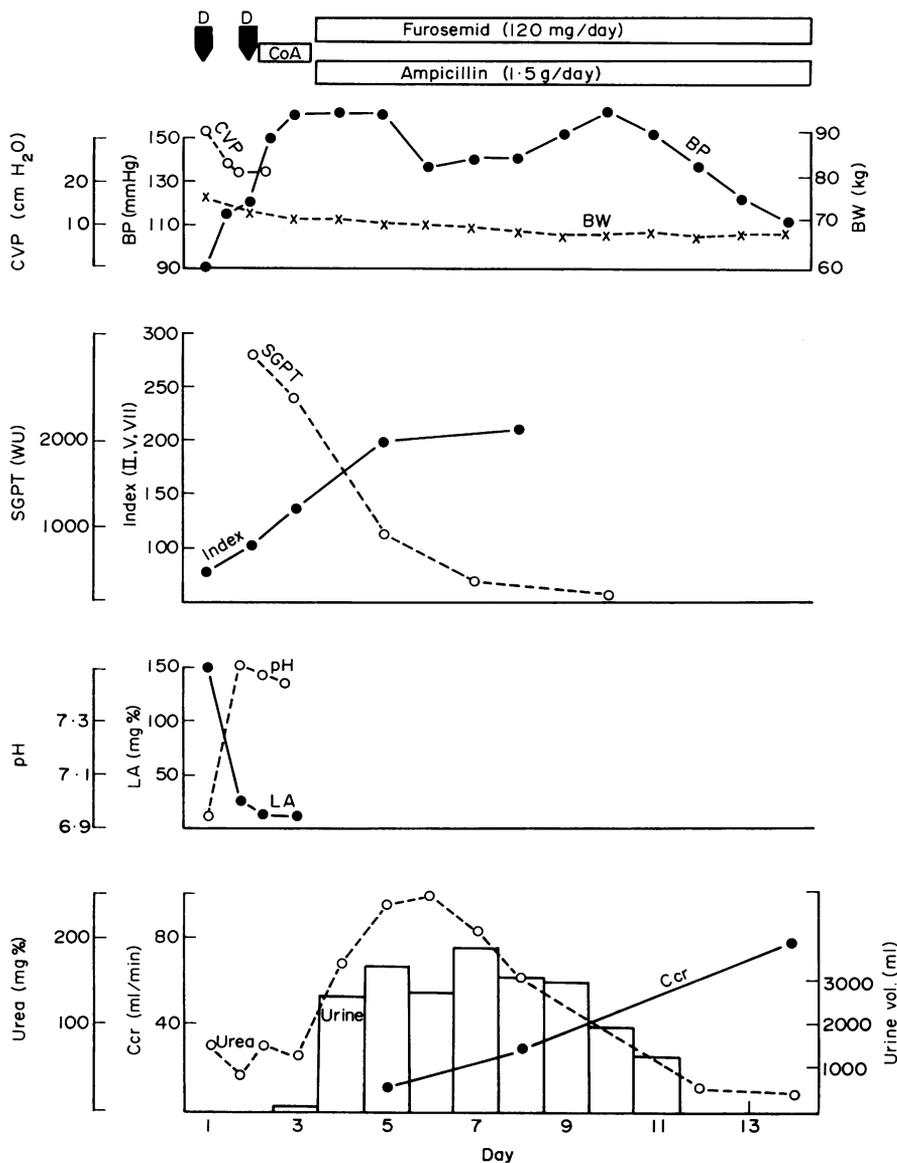


FIG. 3. Patient A. H. P., body weight, circulatory, hepatic and renal features during and after haemodialysis (D) and administration of cofactor (CoA). CVP, central venous pressure; BP, systolic blood pressure; BW, body weight; LA, lactic acid; Ccr, creatinine clearance.

hepatic failure, for it is capable of correcting severe metabolic disorders arising from hepatic insufficiency.

An increase in blood lactate is a regular feature of severe acute hepatic failure, and sodium retention is also commonly present in this condition (Colombi, 1969), due to secondary aldosteronism and/or renal failure. Lactic acidosis can be corrected by the

administration of sodium bicarbonate or THAM, unless sodium retention precludes further expansion of the extracellular volume. In these circumstances haemodialysis offers a method of correcting the lactic acidosis without the danger of sodium or volume overload, both by buffering the H⁺ ions and also by the removal of lactate.

The underlying cause of hepatic and renal

insufficiency in the case described here is not clear. Hepatic insufficiency and right heart failure preceded both decrease of BP and renal insufficiency. Moreover histology of the liver biopsy specimen was consistent with a cardiovascular origin and seemed to exclude viral or drug-induced hepatitis. Furthermore Weil's disease, mushroom poisoning or CCl_4 poisoning and pancreatitis were all excluded either by the history or laboratory investigations. Vitamin B complex was given initially before thiamine deficiency could be proved.

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Pneumatic rupture of colon

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PNEUMATIC rupture of colon is a rare type of industrial accident, first reported by Stone in 1904. Since then there have been a few reports. The classical paper of Wyllys Andrews (1911) gives a vivid description of this injury. Invariably, this accident is the result of a practical joke, when the nozzle of a compressed-air hose is directed at the anal region of the victim. The rarity of the injury has prompted us to report the following case.

Case Report

S.L., 40-year-old male, working in the blast furnace of Bhilai Steel Plant, was admitted as an emergency on the afternoon of 18 June, 1970, complaining of severe abdominal pain and difficulty in breathing. About an hour earlier, while at work in the plant, two of his co-workers held him firmly and directed the outflow nozzle from a compressed-air pipeline towards his anal region and opened the release valve. He suddenly felt a sensation of 'blowing up' with acute abdominal pain and difficulty in breathing.

On examination, he was conscious but had agonizing abdominal pain. The respiratory rate

was 45/min, with very shallow, thoracic type of breathing. Pulse was 120/min and B.P. 90/60 mm Hg. The most striking feature was a grossly distended abdomen. On palpation there was acute generalized tenderness with board-like rigidity of abdominal wall. The percussion note was tympanitic and liver dullness obliterated. No free fluid was detected. Bowel sounds were absent. Examination of the perineum did not show any external injury. Rectal examination did not reveal any laceration or perforation in the anal canal or rectum. Some blood was noticed on the examining finger.

A clinical diagnosis of pneumatic rupture of colon was made.

Straight X-ray abdomen in erect position showed extensive pneumoperitoneum with compression and clear visualization of liver, spleen and gall bladder.

Operation. After initial resuscitation, a laparotomy was carried out through a lower left paramedian incision. As soon as a small opening was made in the peritoneum, the air whistled out and the distended abdomen collapsed like a pricked balloon. Concomitant with this deflation, the anaesthetist reported sudden improvement in the respiration and