SHORT ARTICLE

Metabolic Acidosis as a Complication of Bicarbonate Haemodialysis

Irshad Ahmad Sirwal, Bassam Bernieh, Abdulrahman Osman Mohamad, Mohamed Adnan Abbadi, Mossadeque Ahmed, Ahmad Abdelwahab Altabakh

Abstract

Twelve episodes of severe metabolic acidosis were observed among 10 maintenance dialysis patients using Bicarbonate Haemodialysis (HDB). Patients were stable at the start of haemodialysis (HD) and became sick during or following the procedure. The main clinical features observed were abdominal pain and vomiting, hypotension or shock, and CNS manifestations. Laboratory investigations revealed severe metabolic acidosis in all and hyperkalemia in 4 patients. On four occasions, dialysate fluid sample analysis revealed purely acidic dialysate being delivered to the patients. Patients were treated by sodium bicarbonate, redialysis on another machine and vasopressors when severely hypotensive. One patient died and the rest improved. This potentially lethal complication needs to be considered early in all patients who become sick during or following HDB.

Key words

Bicarbonate dialysis, Metabolic acidosis, Redialysis.

Introduction

Metabolic acidosis is a universal problem in patients with chronic renal failure. The use of bicarbonate buffer at dialysis is more physiological than acetate. It is better tolerated, provides better cardiovascular stability and electrolyte and acid-base homeostasis (1, 2). It also improves the platelet function (3). As a result, the trend is more towards the use of bicarbonate (HCO3) dialysate buffer (4).

The use of acetate and bicarbonate dialysis at the same dialysis centre results in the necessity of different dialysate concentrates. Hence, there is an increasing chance of mistake by using the wrong concentrate (4). Dialysis equipment can proportion the dialysis fluids using an acid concentrate as acetate and still obtain the proper conductivity without setting off alarms (5). To prepare bicarbonate buffer dialysate on-line, an acidic and a basic concentrate have to be mixed with water. Mixture of only the acidic component with water may not be detected by conductivity–meter and unless a pH–meter is included, an acid dialysate will be delivered to the patient inducing life threatening metabolic acidosis.

From the Department of Nephrology, King Fahad Hospital, Medinah Al Munawarah. K.S. A.
Correspondance to : Dr. Irshad Ahmad Sirwal, P. O. Box 2874, Medina Munawarah, K. S. A.

We report 12 episodes of such a complication which was fatal in one and caused significant morbidity in 9 patients.

MATERIAL

At our kidney unit, bicarbonate buffer is used for all except few patients. Acetate buffer is used rarely. In the period between February 1993 to March 1996, a total of 49860 sessions of dialysis were performed. We encountered 12 episodes of severe metabolic acidosis (0.024%) among 10 HD patients. The dialysis had been carried out on Fresenius A2008C on 11 and Cobe C2 machine on one occasion (none of these machines have on-line pH–meter). Clinical features and arterial blood gas analysis were recorded in all and blood biochemistry done on 4 occasions. The dialysate samples were analysed for pH, bicarbonate sodium and potassium during 4 episodes when the complication was recognised during dialysis procedure. All patients received intravenous sodium bicarbonate and redialysis on another machine was carried out on 11 occasions. In addition, vasopressors were initially used in 5 episodes.

OBSERVATIONS

The incidence of metabolic acidosis during or after HDB was 0.024% (12 episodes among 49860 dialysis sessions). The patients' characteristics and time interval between dialysis and diagnosis of metabolic acidosis are shown in Table I. Two patients had 2 episodes each and 9 machines were involved (one machine had 3 and another 2 episodes). Identifiable causes of acidosis when diagnosis was made during dialysis procedure were : (a) human error – failure to add sodium bicarbonate powder to bicarbonate concentrate in one, and (b) technical error – defective suction of bicarbon solution by machine in 3 episodes.

Table 1 : Patients' Characteristics

S. No. of patient	Episode No.	Time before diagnosis was made	Remarks
1.	1	10 hrs. after HD	Symptomatic end HI but left HD unit.
	2	During dialysis	(a)*
2.	3	10 hrs. after HD	Symptoms during HI (? Acute abdomen)
3.	4	5 hrs. after HD	Both dialysed on
4.	5	12 hrs. after HD	on same machine and day
5.	6	10 hrs. after HD	? Acute M.I. (d)
6.	7	During dialysis	HB+ve Patients
	8	End of HD (b)*	dialyzed on same
7.	9	During dialysis	machine.
8.	10	End of HD (b)*	–
9.	11	During HD (c)*	–
10.	12	4 Hrs. after HD	–

(a) Bicarbonate powder had not been added to water.
(b) Bicarbonate solution not consumed at all during dialysis.
(c) Bicarbonate solution partly consumed during dialysis.
(d) Acute MI – Acute Myocardial Infarction.

The main clinical features observed are shown Table 2. One patient (Episode 3) developed seve abdominal pain and hypotension during dialysis insp of fluids and vasopressors. She was admitted to hospi with provisional diagnosis of acute abdomen or seps The diagnosis of severe metabolic acidosis w delayed because arterial blood gas analysis w carried out 10 hours after admission. One inhospi patient (Episode 5) scheduled for vascular access surge the following morning was retrospectively discover to be sick and severely acidotic after another patie (Episode 4) dialyzed same day on the same machi presented 5 hours later to emergency room in critica ill condition. Another in-hospital patient (Episode was initially considered to have suffered acu myocardial infarction or acute mesenteric vascul occlusion.

Various laboratory investigations are given in Table 3. Blood chemistries (Episode 1, 7, 8 & 11) revealed that patients had received adequate dialysis. Hyperkalemia was observed on 4 occasions (Episodes 1, 4, 6 & 12). In all these four patients diagnosis of acidosis was made 5–10 hours after dialysis.

During 4 episodes (2, 7, 9 & 11), when diagnosis of severe metabolic acidosis was made during dialysis, dialysate fluid was analyzed, the results are shown in Table 4.

Nine patients (Episodes 1–11) improved and one patient (Episode 12) died. This patient stabilized her blood pressure after redialysis but continued to have atypical tachyarrhythmia and died 4 hours after redialysis.

Table 2 : CLINICAL FEATURES (N = 12)

		No. of cases	(%)
1.	Hypotension (BP < 90/60 mmHG.)	9	75
2.	Neurologic Manifestations (Altered Behaviour/Sensorium Confusion, Abusive/Delerious)	9	75
3.	Abdominal Pain	6	50
4.	Vomiting	5	42
5.	General Symptoms (Fatiguability, feeling unwell, etc.)	6	50

Table 4. Dialysate Analysis

	Episode No.			
	2	7	9	11
pH	6.67	4.63	4.32	6.5
HCO3 mmol/L	1.2	zero	zero	2.8
Na mmol/L	128	121	122	126
K mmol/L	2.7	2.5	2.6	2.4
Conductivity	14.3	14.3	13.8	14.0

Table 3. *ABG and Biochemistry Results at the time of Diagnosis

	Episode No.											
	1	2	3	4	5	6	7	8	9	10	11	12
pH	7.08	6.98	6.91	6.8	7.18	7.1	7.2	7.00	6.96	7.14	7.22	7.05
pCO2 mmHg	14.6	18.9	15	–	27	22	19.9	21	30	27	25	10
PO2 mmHg	120	118	–	–	120	120	140	108	110	120	102	154
HCO3 mmol/L	4.2	4.2	2.9	5.2	8	8	7.7	4.3	5.5	9.1	10.1	2.9
K meq/L	9.2	–	–	10.1	5.3	8.0	3.5	–	–	–	–	6.7
Creat Umol/L	618	–	–	–	–	–	517	303	–	–	411	–

* ABG : Arterial Blood Gases

DISCUSSION

Severe metabolic acidosis following or occuring during HDB in stable dialysis patients with two consecutive episodes on the same machine and finding purely acidic dialysate being delivered to patients leaves hardly any doubt about the diagnosis of dialysis related metabolic acidosis described in this report. The problems arising during these episodes of rapidly occuring metabolic acidosis and its management are discussed.

Firstly, the signs and symptoms are variable and non-specific and hence another diagnosis may be entertained in such cases leading to considerable delay unless an arterial blood gas analysis is included in the initial work-up. Delay in diagnosis is likely to affect the outcome adversely. Secondly, metabolic acidosis may cause hypotension by cardiac depression (6) and vasodilatation. This hypotension may be resistant to vasopressors, making redialysis difficult to carry out. Thirdly, hyperkalemia primarily occuring as a result of shift from intracellular compartment is another potentially lethal consequence as observed by others (7). Fourthly, cardiac arrhythmias particularly ventricular fibrillation (6, 8) and pulmonary oedema (9) may occur due to severe acidosis. Furthermore, rapid correction of acidosis will shift potassium into cells and added to this the loss of potassium during redialysis can cause serious hypokalemia, predisposing to life threatening cardiac arrhythmias (7, 10). Hence, these patients need meticulous monitoring.

Various safeguard measures such as colour-coding the concentrate container, the delivery line and connectors, keying the connector online to the connector on the concentrate container, etc., have been advocated to prevent such a lethal complication (4, 5). Although on-line pH monitoring is an ideal safeguard, this is currently not practised at many dialysis centres. When on-line ph–meter is not used, physicians should keep in mind the possibility of existence of such a problem to prevent morbidity and mortality. Human and technical errors are the causes of this very low incidence of metabolic acidosis during HDB. As it is potentially lethal, the possibility of its occurrence and potential adverse results should be explained to the dialysis nurses repeatedly and especially to the new recruits.

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