An unusual cause of lactic acidosis following spinal surgery

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Accepted 23 August 2022

DESCRIPTION

A man in his 60s was admitted to the high dependency unit (HDU) following an elective instrumental fixation of T12-L5 for symptomatic spinal stenosis. He had a medical history of hypertension and sickle cell trait. His surgery was performed in the prone position and lasted 7 hours. He had approximately 1.5 L of blood loss. Brief periods of intraoperative and postoperative hypotension were managed with a combination of intravenous fluids, vasopressors and blood products. There was no period where the mean arterial blood pressure was less than 60 mm Hg. Postoperatively, he was successfully extubated and weaned off of vasopressors.

On day 0 on HDU, he began to develop an unexplained and progressive lactic acidosis with a raised anion gap of 24 mmol/L. Clinical examination was unremarkable. His haemodynamic parameters were within normal range, his thromboelastogram was normal, his urine output was between 1 mL/kg/hour and 2 mL/kg/hour and bedside echocardiography demonstrated that he was fluid replete with no features of tamponade. His lactate however continued to rise and peaked at 9.10 mmol/L (table 1).

Concerns were raised about the possibility of a retroperitoneal haematoma as a likely differential in view of the surgical approach that was taken. A CT scan was therefore performed of his abdomen and pelvis. This showed ischaemia in segments VI and VIII of the liver (figure 1).

Subsequent blood tests corroborated these findings with aspartate aminotransferase (AST) of 436 U/L, alanine aminotransferase (ALT) of 479 U/L, international normalised ratio of 1.5 and normal bilirubin (table 2). A liver screen for other possible aetiologies was negative.

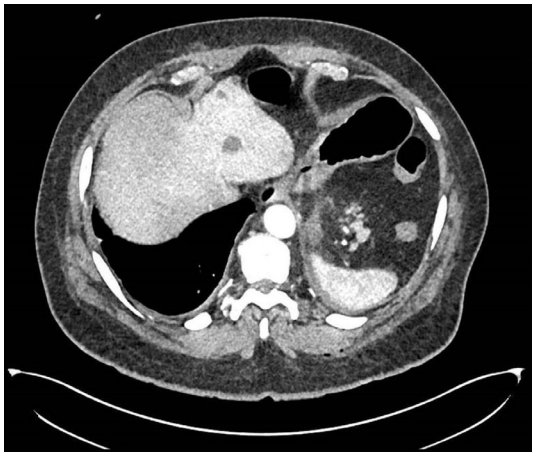

Figure 1 CT scan of the abdomen showing ischaemia of segments VI and VIII of the liver.

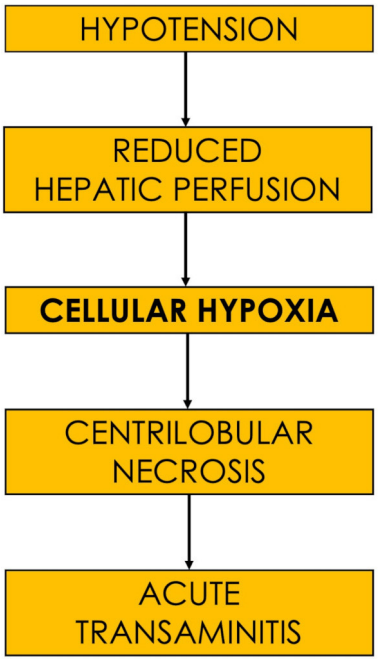

Figure 2 The pathophysiology of ischaemic hepatitis as a result of hypoperfusion (created by Dr Muzzammil Ali).

He was managed conservatively with intravenous fluids, N-acetylcysteine and empirical antimicrobials, following input from the liver specialist team. Within 24 hours, his lactic acidosis resolved and after 5 days his liver function tests normalised. He was subsequently discharged from the hospital with no concerns raised at his outpatient follow-up clinics.

This case highlights the importance of broad thinking when approaching lactic acidosis.[1] There are many ways of classifying this but one simple framework for the postoperative surgical patient can be seen in table 3.

In this particular case, the transient periods of intraoperative and postoperative hypotension were most likely responsible for the development of hepatic centrilobular necrosis and ischaemic hepatitis. This resulted in reduced lactate clearance and lactic acidosis.

The pathogenesis of this condition occurs as a result of a 'two-hit' mechanism where a vulnerable liver is exposed to systemic hypoperfusion and ischaemia.[2] This leads to cellular hypoxia

To cite: Kondratowicz J, Najeeb H, Ali M, *et al*. *BMJ Case Rep* 2022;**15**:e252352. doi:10.1136/bcr-2022-252352

Table 1 Trend in arterial blood gases

	Day 0	Day 0	Day 0	Day 0	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 2
Time	15:57	18:48	20:24	23:29	00:21	01:37	02:39	04:50	06:52	07:59	10:34	12:51	15:02	17:43	20:53	23:41	02:10
pH	7.32	7.33	7.34	7.30	7.27	7.27	7.26	7.28	7.31	7.31	7.32	7.32	7.33	7.38	7.41	7.42	7.390
PO_2 (kPa)	32.5	22.7	15.5	13.8	14.6	12.8	16.3	11.7	11.5	12.4	10.6	10.6	12.3	10.6	10.0	9.8	13.7
Pco_2 (kPa)	5.4	5.2	4.7	4.2	4.5	4.1	4.1	4.3	4.2	4.2	4.2	4.0	4.0	4.3	4.3	4.4	5.2
Std Bicarb (mmol/L)	20.4	20.0	19.2	16.5	16.2	15.3	14.9	16.1	16.2	16.9	17.0	16.6	17.0	19.5	21.1	22.1	22.8
Act Bicarb (mmol/L)	20.8	20.0	18.6	15.2	15.4	14.1	13.6	15.2	15.7	15.8	15.8	15.3	15.7	18.5	20.0	21.2	22.9
Base excess (mmol/L)	−4.9	−5.4	−6.4	−10.0	−10.4	−11.6	−12.1	−10.4	−9.4	−9.4	−9.3	−9.7	−9.2	−6.0	−4.1	−2.8	−1.8
Anion gap (mmol/L)	15.9	17.3	18.9	22.6	21.1	24.0	23.6	22.2	21.8	23.9	22.3	26.0	20.1	21.3	17.9	16.7	15.2
Fio_2	0.21	0.28	0.21	0.21	0.25	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21
Sodium (mmol/L)	137.3	136.8	136.9	137.2	136.8	137.6	136.4	136.9	138.9	139.0	137.6	138.7	133.9	136.1	134.9	135.2	137.0
Potassium (mmol/L)	4.99	5.52	5.56	5.29	5.31	5.57	5.07	5.24	5.70	5.15	4.99	4.57	4.61	4.41	4.26	4.33	3.95
Chloride (mmol/L)	105.6	105.0	105.0	104.7	105.6	105.1	104.3	104.8	107.1	104.4	104.5	102.1	102.7	100.7	101.3	101.6	102.8
Total Hb (g/L)	102.2	101.3	104.4	107.7	94.8	91.6	93.4	81.8	81.6	88.4	74.9	77.6	74.1	73.7	71.4	69.8	76.5
Haematocrit	31.5	31.5	32.2	34.0	29.9	28.6	31.0	26.2	28.0	28.0	24.2	25.6	25.0	24.3	23.8	23.0	24.3
Glucose (mmol/L)	7.76	9.53	10.77	11.69	10.50	10.12	10.66	9.65	9.38	9.45	10.64	11.21	13.17	10.42	9.02	7.18	6.30
Lactate (mmol/L)	1.96	1.83	2.56	5.34	6.71	8.02	9.10	7.49	6.45	6.20	6.37	5.71	5.45	3.85	3.33	2.35	1.32

The yellow colour reflected the trends in the parameters.

Table 2 Trend in biochemical blood tests

	Day 0	Day 1	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Urea (mmol/L)	6.7	7.2	8.6	7.0	5.4	5.4	4.0	4.8	4.4	3.5
Creatinine (µmol/L)	117	125	130	109	88	82	80	74	80	77
Estimated glomerular filtration rate (mL/min/1.73 m^2)	52	48	46	57	74	80	83	86	83	84
Sodium (mmol/L)	140	137	132	137	136	139	138	135	140	138
Potassium (mmol/L)	5.4	5.2	4.9	4.5	4.5	4.6	4.8	5.4	4.3	4.5
Aspartate aminotransferase (U/L)		317	436	564						
Alanine aminotransferase (U/L)	128	342	479	522	457	301	194	162		81
Bilirubin (µmol/L)	18	19	15	60	15	11	10	13		6
Alkaline phosphate (U/L)	55	61	55	25	81	104	99	99		88
Albumin (g/L)	21	25	23	7.0	25	26	26	28		25

The yellow colour reflected the trends in the parameters.

Table 3 Causes of lactic acidosis in the postoperative surgical patient

Increased lactate production	Reduced lactate clearance	1. Exogenous administration
► Increased anaerobic metabolism ► Accelerated glycolysis ► Increased pyruvate concentrations	► Liver disease ► Impaired gluconeogenesis ► Renal failure ► Decreased mitochondrial metabolism	► Lactate containing fluids

Learning points

- Lactate can be elevated for many reasons. While tissue hypoperfusion may be the most common cause of elevation, many other aetiologies exist. Clinicians need to be aware of the many potential causes of lactate elevation as the clinical and prognostic importance of an elevated lactate level varies widely by disease state. Moreover, specific therapy may need to be tailored to the underlying cause of the elevation.
- Ischaemic hepatitis is a clinical syndrome frequently encountered in critically ill patients that represents a complication of underlying cardiac, circulatory or respiratory failure. The pathogenesis of ischaemic hepatitis appears to occur as a result of a 'two-hit' mechanism where cellular hypoxia plays a central role. The mortality is high and is dependent largely on the underlying cause of hypotension. Treatment is directed at the underlying cause of the haemodynamic disturbance.

and a marked but transient elevation in AST and ALT (figure 2). The mortality is high and is dependent largely on the underlying cause of hypotension. Treatment is directed at the underlying cause of haemodynamic disturbance.

Contributors HN and MA gained consent, gathered the clinical information for the case, were involved in the diagnostic process and assisted with the write up of the case. TT critically reviewed the article for intellectual content and assisted with the write up of the case. MA and JK were involved with the conception and design of the manuscript, image creation and review of the literature.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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