E. coli bacteraemia and multi-organ failure complicating classical heatstroke – a case report

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Introduction

We present the case of a gentleman who required ICU treatment for multi-organ failure after developing classical heatstroke (CHS) and subsequent E. coli bacteraemia. The gastrointestinal (GI) tract becomes more permeable in hyperthermia, and the bacteraemia was likely due to translocation through a more permeable intestinal membrane into the systemic circulation, representing an additional cause of organ injury. E. coli bacteraemia in heatstroke has only been reported very rarely before.

Case report

A 78-year-old gentleman with a history of hypertension, treated with atenolol, and psoriasis presented to our district general hospital with heatstroke and GI symptoms. On the previous day, he had taken a 2-hour car journey without air conditioning on a day with an ambient temperature of 37.8°C. He had been well prior to the car journey. Upon arrival back home, he felt hot and unwell but ate dinner with his family. He reported that the dinner contained sausages which although cooked, were past their expiry date. In the early hours of the following morning, he felt hot, developed colicky abdominal discomfort with loose stool, and had a pre-syncopal episode. There were no other symptoms apart from confusion. He was brought to the emergency department, around 18 hours after the initial car journey.

On admission, his peripheral temperature was 40.2°C, heart rate 120 /min, BP 120/78 mmHg, respiratory rate 24 /min and oxygen saturations of 95% on air with a GCS score of 13 (E4 V3 M6). His initial lactate was raised. He had acute kidney injury, liver derangement and a raised C-reactive protein (CRP) but normal white cell count and differential.

He was initially cooled with intravenous (IV) fluids and treated empirically with IV piperacillin/tazobactam and gentamicin for presumed sepsis. A CT scan revealed no evidence of cholecystitis, colitis or biliary or bowel obstruction, and no evidence of renal or urinary disease. The CT brain scan showed no acute pathology. He was admitted to a medical ward.

Blood cultures grew E. coli. A urine sample grew enterococcus, thought to be a contaminant. Stool samples did not show evidence of enteroinvasive or verotoxic E. coli, or other common gastroenteritis pathogens.

He was admitted to the ICU 12 hours after hospital admission. He had cardiovascular, renal and liver failure and coagulopathy (see Table 1). The cardiac marker troponin I was raised, but there was no evidence of an acute territorial ischaemic event or clinical symptoms.

On the ICU, antibiotics and supportive care were continued, and he improved (see Table 1 and Graphs 1 & 2). After admission to hospital, his temperature remained below 38°C throughout.

He was discharged from ICU after 6 days and discharged home 5 days later.

The source of the E. coli bacteraemia was unclear. Given the timing of the onset of symptoms, no unwell family members and negative stool tests, acute infective gastroenteritis was considered very unlikely. The symptoms of E. coli gastroenteritis occur after a minimum of 24 hours, and more usually after 3 to 4 days [1].

There was no signs or symptoms of a urinary, soft tissue or hepatobiliary source of infection. The E. coli bacteraemia was therefore attributed to gut translocation from heat illness.

Discussion

Heatstroke is the most serious disease in a spectrum of heat illnesses. Heatstroke is characterised by hyperthermia of over 40°C combined with acute dysfunction of the central nervous system, for example, delirium or a low GCS score. Heatstroke is classified by into exertional heatstroke (EHS) and classical (or non-exertional) heatstroke. The former is typically seen in young, able-bodied individuals performing rigorous physical activities, such as athletes, the military or workers in some physical occupations. The latter often occurs in elderly patients with co-morbidities, in the absence of strenuous activity.

England records 2000 heat-related deaths every year [2]; climate change is estimated to increase heat-related mortality by around 250% in 30 years' time [2]. CHS has a mortality rate of up to 64% [3].

Systemic multi-organ dysfunction (MODS), including renal failure and rhabdomyolysis, hepatic failure, cardiovascular collapse, neurological dysfunction and haemorrhagic complications, including disseminated intravascular coagulopathy, is well recognised in EHS, from which recovery may be delayed or be incomplete [4]. MODS with CHS in the temperate UK, as presented here, is less reported.

Several risk factors increase the risk of HS. In this case, psoriasis [5] and beta-blockade in the treatment for cardiovascular disease [6] are both recognised risk factors.

The tissue damage in heatstroke is probably due to both direct thermal damage and inflammation. Current treatment focusses on rapidly reducing the core temperature [7]. Administration of steroids may also improve outcome [8] but is not currently advocated. However, translocation of gastrointestinal pathogens into the systemic circulation through a GI wall made more permeable by the high temperatures may also occur. Antibiotics against GI bacteria may improve outcome [9] but are also not currently recommended.

Bacteraemia after heatstroke is reported. In one series, 28% of patients with CHS admitted to ICU after a heat wave had concomitant bacterial infection in blood cultures, but in none was E. coli found [10]. Furthermore, procalcitonin, which has a high sensitivity and specificity for detecting bacteraemia, was elevated in 58% of patients with CHS and was associated with mortality [11].

E. coli bacteraemia occurring at the onset of CHS has seldom been reported. Only one other similar report was found, in a 45-year-old man with schizophrenia [12].

Conclusions

Heatstroke may be complicated by changes in GI permeability, which may be associated with GI bacterial translocation and multi-organ failure.

Acknowledgements

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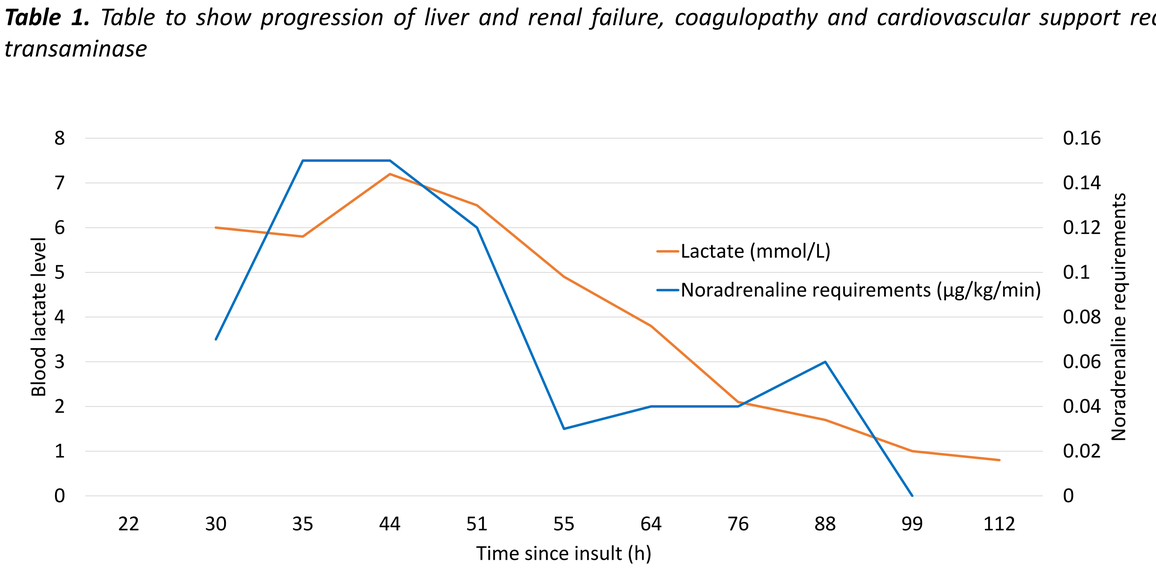
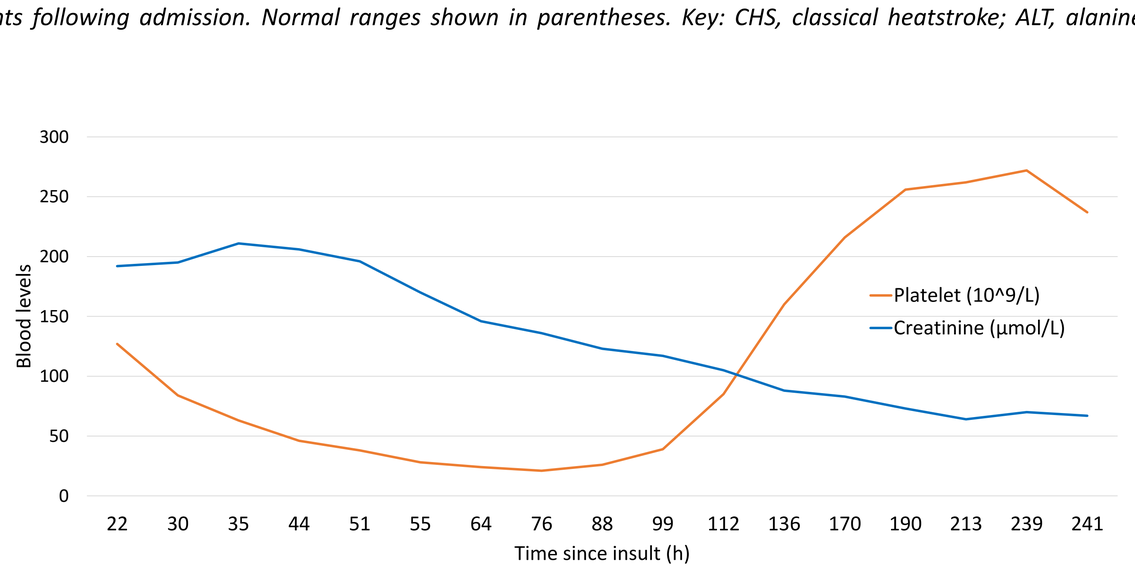
Results

Day of hospital admission	Baseline	1		2				3		4		5	6	7	8	9	10	10	
Day of ICU admission				1				2		3		4	5						
Time since CHS insult (h)		22	30	35	44	51	55	64	76	88	99	112	136	170	190	213	239	241	624
Platelet (150–450 x 10^9/L)	279	165	127	84	63	46	38	28	24	21	26	39	85	160	216	256	262	272	237
INR (0.8–1.2)		1.11	1.25	1.46	1.72	2.16	2.53	2.25	1.44	1.16	1.01	1.01	1.00						
White cell count (4–11 x 10^9/L)	7.9	7.2	20.9	22.2	19.3	17.7	15.2	16.3	20.4	24.8	32.0	28.7	28.6	36.5	30.7	21.5	14.9	13.8	5.6
Urea (2.9–7.5 mmol/L)	6.3	12.3	15.1	16.0	17.0	17.1	17.7	17.0	17.3	17.6	17.4	18.6	19.5	16.5	13.3	11.2	9.3	9.2	5.8
Creatinine (64–104 µmol/L)	62	151	192	195	211	206	196	170	146	136	123	117	105	88	83	73	64	70	67
Bilirubin (0–23 µmol/L)		50	56	46	56	57	57	55	47	39	41	29	20	27			18	16	
Alkaline phosphatase (30–130 U/L)		95	79	56	57	52	56	54	71	90	128	131	141	210	162		162	155	
ALT (10–49 U/L)		748	1216	1262	1656	1661	1799	1661	1344	1073	956	722	518	344			137	128	
Lactate (0.5–1.6 mmol/L)		6.6		6.0	5.8	7.2	6.5	4.9	3.8	2.1	1.7	1.0	0.8						
Noradrenaline requirements (µg/kg/min)				0.07	0.15	0.15	0.12	0.03	0.04	0.04	0.06	0							

Table 1. *Table to show progression of liver and renal failure, coagulopathy and cardiovascular support requirements following admission. Normal ranges shown in parentheses. Key: CHS, classical heatstroke; ALT, alanine transaminase*



Graph 1. *Progression of lactate levels and noradrenaline requirements during admission*



Graph 2. *Progression of platelet and creatinine levels during admission*

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