

CASE REPORT

Transient dense flaccid left-sided hemiparesis in a case of heat injury

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ABSTRACT

A left flaccid hemiparesis resolving completely over 44 hours occurring in a soldier of 19 years of age following a fitness test is described. This was not associated with any abnormality on magnetic resonance imaging of the brain and was probably an exceptionally rare manifestation of heat-induced neuropraxia.

sided hemiplegia with slow spontaneous recovery over a period of 44 hours in a case of heat injury prompts this report.

Report of case

A 19 yrs old, right-handed, Craftsman REME attached to the first battalion Worcestershire and Sherwood Foresters Regiment (1 WFR) at Banja Luka was one of 8 heat casualties arising when a 150-strong company-sized formation undertook a combat fitness test (CFT) at about 0800hrs on 170399. He was the worst of the 3 casualties admitted to UK Armoured Field Ambulance Group, Sipovo. He had been well and had taken no alcohol the previous night. The weather was cold with snow lingering. He took water throughout the CFT. He was dressed in thermal T-shirt, combat kit with socks, boots and helmet, was wearing webbing with day sack and carrying an SA80 assault rifle. Two kilometres before the end of the CFT, he felt drowsy and tired but, encouraged to complete the CFT, he drank a lot of water and continued and was actually able to finish, including the concluding test of carrying a man for 100 metres. The next thing immediately after this that he could remember was difficulty keeping his eyes open, sitting down and then being helped into a Landrover which transported him to the medical centre at Banja Luka where he arrived at 1030hrs. Clinical progress and treatment related to time are shown in Table 1.

Table 1. Early clinical course of this patient

Time	Pulse (b/m)	BP (mmHg)	GCS			Pupils	Observations	Fluids	UVol (mlds)
			E	V	M				
1110	111	116/51	2	1	2	=	O2 by mask	11.5DW 11.NS	
1125			3	1	6			11.NS	0
1140	96		3	1	6	=		11.NS	20
1200		140/57	1	1	4	=			
1255	103	145/77	3	4	5	=	L hemiparesis noted. Moving R side. Head turned to R	11.5DW	1100
1315	102	168/69	4	5	5		Starting to move L leg BM=4.5mM		
1330							Ignoring L side & left arm BM=2.4mM	50ml 50% DW	1900
1400							Aware of L arm & 50% turning head to L and talking but not moving L arm G=2.2mM	50ml DW 11. NS	
1500			4	5	5		Not moving L arm still L facial palsy G=7.1mM		
1600							Moving L arm. Weak grip. L facial weakness but improving		

b/m=beats per minute, BM-BM Sticks® blood glucose, DW=dextrose solution, G=glucose, GCS=Glasgow Coma Score (E=eye score, V=verbal score, M=motor score), NS=normal saline, UVol=urine volume

Introduction

Focal neurological signs in cases of heat illness are exceedingly rare. None have been mentioned in a review of previous reports of cases of heat illness (1,2) and none have been seen by me in 15 years of military medical service during which time many cases of heat illness of injury have been examined. However, one report of fatal cases of heat injury mentioned that hemiplegia had been observed in an unspecified number (3). The usual clinical picture in relation to military heat casualties is that a generalized cerebral disturbance consisting of deteriorating consciousness and worsening ataxia occurring on an organized run leads to loss of consciousness and collapse with fairly swift return to normal on appropriate treatment with no persisting focal neurological abnormality on clinical examination. The occurrence of a dense left-

Neurological Findings

With recovery of level of consciousness and speech coincident with a rise in blood pressure about 3hrs after onset of heat illness, hemiparesis was noted on the left. The head was turned to the right and movement was apparent on the right. After a further 20 minutes, movement returned to the left leg but the patient ignored the left side and left arm. Four hours after onset of disturbance, there was still no movement in the left arm although the patient was aware of his arm and was now turning his head to the left. After another 2 hours, movement returned to the left arm although left facial weakness, which was improving, persisted. By 24 hours after the onset of illness, higher mental functions were normal and there was no visual or sensory inattention. The only neurological sign was an extensor plantar

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Table 2. Cumulative laboratory results

Date	170399	180399	190399	200399	240399
Time (hr)	1400	0800	0800	0800	0800
Time since onset of illness	3.5hrs	21.5hrs	2 days	3 days	7 days
Hb (g/dl)	12.2 (Normal indices)	13.8	-	-	14.2
WBC (x 10 ⁹ /l)	23.8	8.6	-	-	4.24
Platelets (x 10 ⁹ /l)	163	132	-	-	179
INR	1.2	1.0	-	-	-
Urea (mM/l)	6.1	3.2	3.6	4.4	7.0
Creatine (μM/l)	122	114	123	111	113
Na (mM/l)	148	137	138	139	143
K (mM/l)	3.5	3.8	4.1	4.5	4.2
Cl (mM/l)	109	102	100	101	-
HCO ₃ (mM/l)	22	25	27	27	29
AsT (iU/l) [5-40]	121	113	79	50	20 [17-59]
AIT (iU/l) [5-40]	80	97	84	79	36 [21-72]
CK (iU/l) [55-170]	656	2820	2790	1830	98 [30-170]
LDH (iU/l) [313-618]	1128	908	735	652	137 [90-230]

AsT = aspartate aminotransferase, AIT = alanine aminotransferase, CK = creatine phosphokinase, LDH = lactate dehydrogenase. Reference ranges for enzymes are shown in square brackets. Results were from Sipovo, Bosnia, except on 240399 when they were from Royal Hospital Haslar where different reference ranges are shown.

response on the left and this had resolved after a further 20 hours. At no time during the admission was neck stiffness apparent nor did the patient report sudden onset of headache.

Other clinical findings

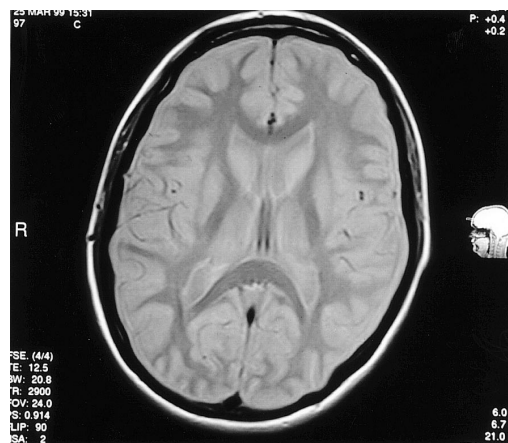
Both heels bore full thickness blisters which had burst and there was some minor bleeding. Otherwise, there were no other abnormalities. In particular, pigmenturia was not detected at any time and urine output was always well maintained.

Investigations

Results of investigations are shown in Table 2. These had all returned to normal by the time of admission to the Royal Hospital Haslar on 240399 where neurological examination remained normal, although heel injury was still apparent, and MR brain scan showed no apparent abnormality apart from some skull asymmetry (see Figure).

Discussion

The sequence of events was compatible with heat injury although no core temperature reading was obtained. This previously fit soldier with no past history of heat illness showed neurological deterioration following a fitness test. There was laboratory evidence of tissue injury with a leucocytosis, elevated transaminases, elevated creatine



Proton density, magnetic resonance axial image of the brain obtained with 1.5 T Picker Eclipse MR scanner on 25 March 1999 which showed no abnormality following onset of a transient dense flaccid left-sided hemiparesis, suspected to be an unusual manifestation of heat injury, after a combat fitness test on 17 March 1999.

phosphokinase, and a relative metabolic acidosis (bicarbonate at the lower limit of normal) probably betokening enduring lactic acidosis. Some of these laboratory abnormalities took more than three days to resolve although all were normal by one week. It is unlikely that the injury to the heels contributed to the laboratory abnormalities as the injuries persisted when admitted to the Royal Hospital Haslar at a time when all laboratory test results were normal. It is also unlikely that hypoglycaemia contributed to the clinical picture. Although a low blood glucose was identified, 5% dextrose had been given at the earliest opportunity and intravenous injection of 50% dextrose did not have any dramatic beneficial effect on any aspect of the neurological deficit which continued to improve slowly. The normal MR brain scan at one week suggested that the neurological deficit was due to the right (non-dominant) cerebral hemisphere having a lower threshold to develop a neuropraxia in response to elevated core temperature than the left.

Conclusion

Apparently, this is the first description of a non-fatal, heat injury-related transient left flaccid hemiparesis attributed to a neuropraxia affecting the non-dominant cerebral hemisphere.

References

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