A Case of Factitious Fever and ‘Epilepsy’

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SUMMARY: We describe the case of a 23 year old male who presented with a history of intermittent pyrexia associated with apparent episodic loss of consciousness. During these events, thermometers placed in the rectum and axillae supported the elevated oral temperature readings. After numerous investigations including electroencephalography had excluded organic disease, the patient was observed applying a hot douche to his rectum prior to his temperature being recorded.

Introduction
Prolonged pyrexia of unknown origin (PUO) is generally an atypical presentation of a common disease (1, 2). Organic causes of a pyrexia must first be excluded before factitious fever is considered a diagnostic possibility. However, when episodes of loss of consciousness are associated with the development of a fever, the situation becomes more difficult to resolve and can lead to an even more prolonged period of investigations prior to solving the diagnostic dilemma.

We report the case of a 23 year old male who presented for neurological opinion following a series of episodes of loss of consciousness associated with pyrexia. However, subsequent to prolonged neuroelectrophysiological investigations, a diagnosis of factitious fever was proved.

Case Report
This 23 year old soldier was admitted to the Queen Elizabeth Military Hospital for investigations of PUO and recurrent episodes of loss of consciousness. Six months prior to this admission, while on a military exercise, the patient had apparently lost consciousness for approximately one hour and had been noted to be pyrexial. He was treated as a case of heat illness. After a few days of observation in a local hospital, his temperature readings had returned to normal and he was discharged.

Four months later, the patient had fallen off a lorry and a few minutes after this event he had become unrousable but reacted to painful stimuli. Shortly after admission to a local civilian hospital, the patient regained consciousness and neurological examination revealed no abnormal clinical findings apart from minimal drowsiness.

The patient was observed in an intensive care unit setting for a period of 48 hours during which time he was under constant nursing supervision and no pyrexia was documented. Computerised Tomography (CT Scan) of the skull was unremarkable. Subsequent to being transferred to a general ward, the patient became more alert and, on one occasion, was observed to bleed from the inner canthus of the right eye for which no obvious cause was elicited. A few days later, the patient was noted to have an intermittent pyrexia (oral — 39.6°C, axilla — 39.4°C and rectal — 39.0°C) which was not associated with an increase in heart rate. Apart from lethargy, the patient did not complain of any symptoms.

Repeated clinical examinations failed to detect any possible cause for the fever. All haematological (including blood film examination during pyrexia), biochemical radiological and microbiological investigations (including several blood cultures, as well as Brucella, Cytomegalovirus, Toxoplasma and Epstein Barr virus antibody titres) did not suggest any diagnosis.

On the twenty-third day following his head injury, the patient was observed by medical and nursing staff to suddenly fall to the ground with subsequent shaking of all four limbs accompanied by twitching of his facial muscles. Subsequently, the patient appeared to be unrousable for several hours but responded to painful stimuli. However, there was no tongue biting and no urinary incontinence associated with this event. Clinical examination revealed a pyrexia of 40°C and there were no abnormal neurological findings.

The patient was transferred to the Queen Elizabeth Military Hospital for neurological assessment. Repeat CT Scan and magnetic resonance imaging of brain were both normal. Examination of cerebrospinal fluid obtained on three separate occasions failed to reveal any evidence of pathology.
The patient continued to exhibit episodes of shaking of limbs accompanied by apparent loss of consciousness and intermittent pyrexia (as measured by thermometers placed in the axillae and rectum). The patient’s temperature readings in relation to his episodes of altered consciousness over a typical three day period are detailed in Figure 1. It was observed that the patient’s heart rate did not increase during pyrexia and, despite rapid reductions (within minutes) in recorded temperature, the patient never exhibited perspiration. In addition, during these three days, the patient was not permitted to receive diaphoretics or any other type of medication.

At no stage were these alterations in consciousness associated with clinically detectable abnormal neurological signs. Electroencephalography (EEG) recorded during these episodes revealed suppression of alpha wave rhythm on eye opening and no epileptiform activity, thereby suggesting that the patient was not unconscious.

While under neurological investigations, the patient was observed to be bleeding from the left ear and examination revealed blood on the floor of the external auditory canal with evidence of a recent abrasion. The patient denied the possibility that this injury had been self induced.

A blood glucose profile performed over a 14 hour period produced unusual results with plasma levels ranging between 31 mmol/l (fasting) to 5.76 mmol/l (postprandial) (normal range 4.4 - 6.7 mmol/l). However, the patient had received advance notice that his blood glucose levels would be monitored. Therefore, without warning, fasting and random blood glucose measurements were repeated and were found to be within the normal range.

A glucose tolerance test (with warning) was performed and the unusual results obtained from this investigation are compared with typical normal and diabetic curves in Figure 2.

Eventually, the nursing staff were instructed to obtain a second temperature recording 15 minutes after a documented abnormal reading. During this 15 minute period, the patient was confined to his bed and was under constant nursing observation and, without exception, the second temperature readings which were obtained in this manner were normal. On two occasions, a glass of hot water was found on the patient’s bedside locker. It was also noted that, prior to recording an elevation in rectal temperature, the patient would insist on a visit to the bathroom. However, after several minutes of nursing supervision, a second rectal thermometer reading would be normal. On one of these visits to the bathroom, the patient was observed to apply a hot douche to his rectum.

Following the discovery of a hand laceration which required suturing, a search of the patient’s bedside locker revealed the presence of five hospital type thermometers.

The patient was finally referred for a psychiatric opinion ten months after his first presentation with loss of consciousness. When confronted with descriptions of his behaviour, the patient denied that he had manipulated his temperature recordings.

After a period of psychiatric assessment and supportive therapy, the patient was diagnosed as suffering from a post-concussional syndrome which had been compounded by a personality exhibiting a degree of stress vulnerability. It was considered that the patient had not been consciously attempting to deceive medical and nursing staff.

Formal psychiatric opinion stated that post-concussional syndrome in combination with his personality rendered this soldier unfit for further military service and the patient was medically downgraded to M2 S8 PES = None (Indefinitely).
Discussion

This case of factitious fever presenting with apparent epilepsy is most unusual. In addition, because measured rectal temperatures appeared to support the oral thermometer readings the patient was considered to be manifesting a true pyrexia. Pathological conditions which can manifest as pyrexia and seizures include intracranial abscess, infective encephalomeningitis, dural sinus thrombosis, intracranial haemorrhage and neuroleptic malignant syndrome. However, numerous radiological investigations and repeated examinations of cerebrospinal fluid failed to detect any neuro-pathological cause for this soldier’s fever. Furthermore, electroencephalograms which were recorded during the patient’s periods of altered consciousness did not reveal any evidence of a cerebral dysrhythmia and demonstrated that consciousness and vision were normal during these episodes.

In one study of 343 patients presenting with PUO, 32 (9%) were diagnosed as cases of factitious fever (3). The authors of that study suggested signs which would alert physicians to the possibility that they were dealing with factitious pyrexia: absence of tachycardia with abrupt temperature spikes, lack of diurnal variation in temperature, lack of perspiration associated with rapid reductions in temperature, absence of fever in the prolonged presence of medical or nursing attendant, other factitious disease manifestations, marked discrepancy between oral and rectal temperatures when taken simultaneously.

Our patient demonstrated all but the latter of these signs. Switching thermometers is the most common method of inducing factitious fever (3) and our patient was found to be in possession of five thermometers.

It is possible to produce as fraudulent rectal temperature reading by rapidly repeated contractions of the anal sphincter muscles (4). However, our patient appears to have resorted to applying hot douches to his rectum. Presumably, the frequent visits to the bathroom also entailed taking a shower or hot bath and this would account for the observed elevations in axillary temperature. Such activities are recognised to raise body temperature for up to 45 minutes (5).

Similarly, after ingestion of a hot drink, a time lapse of six minutes is required before an accurate measurement of oral temperature can be obtained (5).

This case illustrates that patients with this syndrome can easily confuse experienced medical and nursing staff. Only by persistent and assiduous clinical observation can factitious fever be recognised and appropriate treatment initiated.

REFERENCES