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CASE REPORT

PYREXIA OF UNKNOWN ORIGIN RESPONDING TO ANTIDEPRESSANT THERAPY: A CASE REPORT

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ABSTRACT

The subjective complaint of raised body temperature is a common observation in outpatient clinic, though prevalence data to support the fact is lacking. Some studies have reported this phenomenon to be related to psychological stress, but still it remains less recognized and poorly understood. These patients thus have to undergo several investigations and treatment with minimal improvement and at times being questioned on the veracity of such complaints. This case highlights the plight of such a patient who was thought to have developed pyrexia of unknown origin, but in fact had stress related variations in body temperature.

INTRODUCTION

Pyrexia of unknown origin (PUO) is a challenging medical condition both in terms of presentation of the illness and its management. Despite the use of several newer investigative techniques, the cause of fever might not be found in a substantial percentage of cases. Psychological stress can cause high core body temperature (Tc) which has been termed as psychogenic fever; (Falcon-Lesses *et al.,* 1930) however the fact is still under recognised. This may lead to delay in diagnosis and management and add to the distress of the patient. We report the case of a female with ten years duration of pyrexia of unknown origin which turned out to be psychogenic fever and responded to the use of anti depressants.

CASE REPORT

A 45 year old female presented with complaints of low grade fever, malaise, heaviness of head for the last five years. Her symptoms started with the patient experiencing off and on raised body temperaturethroughout the day with recorded temperature ranging from 98ºF- 100ºF. These episodes were not accompanied by chills or shivering, cough or sputum, sore throat, pain abdomen, bowel irregularity, increased urinary frequency, urgency or dysuria, seizures or loss of consciousness. The patient denied any history of tuberculosis, hepatitis or other infectious diseases, and had no family history of hypertension, diabetes or tumour. Over the past years she had consulted many physicians and was admitted as in-patient for some period of time. Several investigations were done like complete blood count, blood and urine culture, chest X-ray, ultrasonography of abdomen and imaging studies of head, para-nasal sinuses and abdominal cavity; all of which came out to be normal. She took a range of antibiotics and even completed a course of anti-tubercular therapy, but to no avail. Finally, seeing no improvement she was referred to the psychiatric department. Her physical examination did not reveal any rashes, lymphadenopathy or any abnormality. The chest as well as heart sounds were clear. There was no abdominal tenderness or rebound pain, no percussive pain in the liver or kidney area, and no tenderness in the gallbladder or appendix area. Neurological examination was also unremarkable. On detailed history taking, patient revealed other complaints like heaviness of head, lethargy, apprehension, irregular sleep, decreased interests in surroundings. These symptoms had started after the sudden demise of her husband five years ago. One particular observation by the patient was the fact that these 'febrile episodes' occurred more during periods of physical exhaustion and psychological stress, which were frequent as she had to work a lot in order to take care of her children. Mental status examination showed decreased psychomotor activity and speech, depressed affect and preoccupation with her ill health

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with intact insight and judgement. Hamilton rating scale for Depression (HAM-D) was applied and the patient scored 16, indicating a diagnosis of major depression. The scores for the sub-items: depressive mood, insomnia and somatic symptoms (general) were among the top 3 ratings (3, 2, and 2, respectively), with obvious corresponding somatic symptoms. She was diagnosed as a case of Dysthymia and started on SSRI Paroxetine. On subsequent follow up over the next five months, patient reported improvement in her presenting complaints and has been maintaining well till date.

DISCUSSION

Petersdorf and Beeson defined Pyrexia of unknown origin (PUO)as a state of febrile illness for more than three weeks, with a body temperature greater than 38.3 °C on several occasions and uncertain diagnosis after one week of study in hospital (Petersdor and Beeson, 1961). Knockaertand Vanderschueren updated the definition of PUO (Knockaert *et al.,* 2003). They proposed to change the quantitative criterion of three days' investigation after which no diagnosis has been made to a qualitative one, by claiming for an "appropriate intelligent standard inpatient or outpatient workup". However, despite the improvement of diagnostic technologies, especially imaging modalities, it is surprising that some studies still indicate an increased percentage of PUO cases which remain undiagnosed (Bleeker-Rovers *et al.,* 2007).

In 1930, Falcon-Lesses introduced the term 'Psychogenic fever' for stress-induced hyperthermic changes seen in their patient, who would exhibit high oral temperature at the clinic but normal temperature at home (Falcon-Lesses *et al.,* 1930). It is seen that there are patients who develop episodic or persistent high core body temperature (Tc) without any inflammatory cause. Among those, there are patients whose high Tc is associated with psychological stress. Regardless of the source of stress, there is a subset of patient who develop acute rise in Tc, while others show a persistentlow-grade fever (37–38ºC) lasting months and even years after repeated exposure to stress (Kaneda *et al.,* 2009). Rausch et al demonstrated in their study an increased daytime body temperature in cases with major depression and suggested hypothesis of an inflammatory component of depression (Rausch *et al.,* 2003). However, other studies have shown that psychological stress induced hyperthermia is not mediated by pyretic cytokine production but by emotional expression-associated sympathetic activation, which in turn, mediates non-shivering thermo genesis in brown adipose tissue (Oka, 2015). Therefore, systemic administration of cyclooxygenase inhibitors, such as indomethacin, do not inhibit this hyperthermia, while patients benefit from serotonergic tricyclic antidepressants such as amitriptylineand clomipramine, selective serotonin reuptake inhibitors(SSRIs) such as paroxetine and/or relaxation training (Oka, 2015). It has also been suggested that chronic stress exposure can decrease serotonin in the medial preoptic area of the hypothalamus (mPOA) to cause long-lasting thermoregulatory dysfunction (Oka *et al.,* 2001).

Overall although an exact mechanism of raised body temperature in patients with chronic stress/depression is not known, but the existence of such condition in these patients is being acknowledged of late. In the index case the patient likely developed stress induced psychogenic fever, which was mistakenly being treated on the lines of pyrexia of unknown origin for these many years without any success. This condition was bothersome for both the physicians and the patient, as though patient was distressed by the raised body temperature, there was no abnormal finding on any investigation to account for the same and fever did not subside by any antipyretic drug. Based on certain evidences, the patient was started on SSRI Paroxetine to which she responded gradually.[8]This case highlights the range of effects psychological stress can have on a person's body and the fact that still physicians hesitate to acknowledge the manifestations of psychological stress and hence the delay in appropriate patient management.

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