A RARE CASE OF AUTONOMIC DYSREGULATION WITH HYPERTHERMIA IN A TRAUMATIC BRAIN INJURY PATIENT

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INTRODUCTION

Autonomic dysregulation (AD) with hyperthermia is a rare medical diagnosis that can be found in traumatic brain injury (TBI) and non-traumatic brain injury (NTBI) patients. Symptoms include sudden onset hypertension, fever, tachycardia, pupil dilation, and agitation after TBI, hydrocephalus, brain hemorrhage, or central neoplasm [1]. Uncontrolled fevers in these patients require careful consideration to rule out infection, hormone imbalances and inflammatory sources. When extensive workups may fail to reveal a source, treatment with antibiotics, antipyretics or steroids may be tried for treating an undetermined infection or a cancer fever. This case demonstrates the importance of considering central origin for consistently recurring fevers in brain injury patients.

CASE REPORT

27 year old male presented to the ED after a traumatic motorcycle accident. After undergoing a left craniotomy, he developed multiple fevers of unknown origin with negative fever workups. The patient was discharged to acute rehab after completing a one week course of vancomycin and cefepime.

Past medical and surgical history was significant for alcohol abuse disorder.

Family history was non-contributory. The patient's sister indicated daily consumption of alcohol but did not specify the type nor quantity.

On physical exam, the patient was a thin appearing, cachectic male, in no acute distress, with significant findings of depression of the left cranium at the surgical site with a protective helmet in place and PEG tube. Full passive ROM in both upper and lower extremities, with fair strength in the LUE and LLE but poor strength with flaccidity in the RUE and RLE was noted. The sensation assessment was unreliable due to nonverbal status, and muscle stretch reflexes were 2+, with negative Hoffman and Babinski signs.

Vitals: Patient spiked temperatures of 103˚F with negative fever work-ups.

Laboratory studies were significant for euvolemic hyponatremia.

Imaging studies: included head CT (see images).

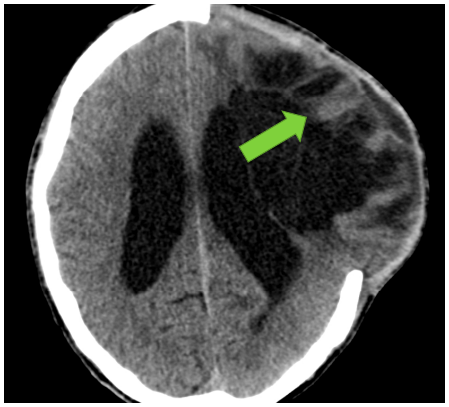
Diagnostic and treatment considerations: Upon initial evaluation and based on the labs and patient's history of brain injury, SIADH was diagnosed. Patient had completed his course of vancomycin and cefepime prior to the rehab admission, yet still demonstrated fevers along with episodes of intermittent tachycardia and diaphoresis. Repeat fever work ups were negative.

After careful assessment, the decision was made to trial the patient off antibiotics and start the patient on amantadine to improve his cognition. Fluids were restricted for SIADH. Amantadine was started on the second day of his hospitalization, and PRN Tylenol was given via PEG tube for the fevers.

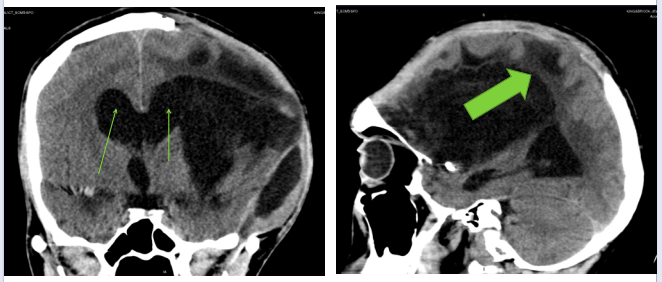
The patient was started on physical and occupational therapy programs. As his cognition improved, his fevers also subsided. The amantadine dose was titrated up and the Tylenol dosing was titrated off. Irregular episodes of fevers, diaphoresis, and tachycardia resolved during this period.

Despite showing moderate improvement in ambulation and cognition, the patient still required significant assistance with ADLs. Subsequently, the patient was discharged to a nursing home.

CT IMAGING



CT scan of the head without contrast shows left frontotemporal craniectomy, encephalomalacia, with herniation of a large portion of the left frontal lobe through the craniectomy defect (arrow).



Coronal view (left image) shows chronic ventriculomegaly (small arrows). Sagittal view (right image) shows encephalomalacia, edema, ongoing ischemia (large arrow) without acute intracranial hemorrhage.

DISCUSSION

- Our patient presented with intractable fevers, secondary to dysregulation of his autonomic nervous system precipitated by his accident. Although concern for systemic infection or inflammation may lead the clinician to initiate empiric antibiotics or steroids, careful evaluation of the patient's history is paramount to come to the right diagnosis.

- A multidisciplinary approach was required to weigh the risks and benefits prior to discontinuing empiric antibiotic therapy and initiating treatment with amantadine. Amantadine appeared to stabilize the fevers while improving his cognition during therapy.

- AD is associated with closed head injuries and should be viewed as a possible cause of uncontrolled fevers. Clinicians should look for episodic fever, elevated HR, shortness of breath, elevated blood pressure, increased extensor tone, pupil dilation and sweating [1].

- One proposed mechanism is the initial release of cytokines secondary to trauma, inflammatory stimulation, and the increased intracranial pressure after acute brain injury via the activation of COX-2 pathways in the periventricular cells and the production of PG E2 [2,3].

- A second proposed mechanism is the creation of heat shock proteins in a coordinated response to tissue injury. Research has shown hyperthermia can be associated with glutamate and nitric oxide release caused by AD of the brain stem [4-6].

- The current documented effective drugs to treat are propranolol (ß-blocker), opioids, clonidine (α2-agonist), bromocriptine (dopamine agonist), chlorpromazine (phenothiazine), and dantrolene (muscle relaxant) [1].

CONCLUSIONS

This case demonstrates the difficulty associated with diagnosing the source of uncontrolled fevers in patients with brain injury. Due to the increased risk of infection often associated with trauma patients, the most common first step is to rule out infectious sources of fever.

Paroxysmal hyperthermic AD should be considered in all patients with brain injuries and intermittent fevers. Pharmacologic treatment that can control hypothalamic thermoregulatory dysfunction should be considered.

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