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Non-reactive unilateral right mydriasis in a 70-year-old woman with acute gastro-enteritis secondary to cholera

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Abstract
Headaches, Dizziness and transient loss of consciousness are common early signs of neuropathology. In our settings, Hypoglycemia and Hypotension may present with either dizziness, loss of consciousness or both. With hypoglycemia causing decreased glucose supply to the brain and hypotension causing decreased blood supply to the brain. This case study looks at a case of a 70-year-old woman with acute gastro-enteritis 2^0 to cholera who presented unconscious to our facility with non-reactive unilateral mydriasis.
She had rapidly received 5.5L of normal saline to correct her dehydration at a primary care point, where the unilateral mydriasis was first noticed. However, on development of lateralizing signs pathognomic of stroke, she was referred to our facility. Where we continued the rapid fluid and oxygen therapy. Until she was diagnosed with fluid overload and later died.
The working explanation is that the non-reactive pupils were as a result of an increased intracranial pressure as a result of rapid rehydration causing neuronal deterioration leading to cerebral oedema. However, the lateralizing signs may also be linked with the unilateral mydriasis. Inability to perform an autopsy will elude us the chance of a 100% conclusive diagnosis.

Keywords: Unilateral mydriasis, cholera, acute gastro-enteritis, ICPs, increased intracranial pressure, dehydration, non-reactive pupils

Introduction
Although low blood sugar seems desirable and often is nonproblematic in some people; abnormally low blood pressure may be life threatening. The symptoms range from as harmless as fatigue, to as severe as fainting. Hypotension is often an indication of an underlying pathology. Dehydration is one of the common causes of hypotension. In this case, we aim to see and discuss the case of a hypertensive elderly woman who presented with symptoms of hypovolemic shock secondary to cholera, who had atypical pupillary signs of Increased intracranial Pressure (ICP).

Case Presentation
A 70-year-old female trader whose source of drinking water is well-water was brought into the accident and emergency on referral from a smaller clinic seeking expert care from where she had received 5.5L of 0.9% Normal saline fluid infusion. She presented with 18 hours history of 3 bouts of passage of loose stool, 4 bouts of vomiting and 8 hours of altered consciousness. She does not smoke, consume alcohol beverages and had no history of seizures nor past history of loss of consciousness.
On examination she was unconscious with a Glascow Coma Scale (GCS) of 5/15, loss of nasolabial fold on the right with mouth deviated to the left. she had dilated pupils on the right and constricted pupils on the left approximately 6mm and 2mm, respectively; both unresponsive to light. She was severely dehydrated but not pale, anicteric, no edema, afebrile (36.4C) and had a Random Blood Sugar (RBG) of 13.8mmol/L. she had a regular but rapid pulse rate of 120 beats-per-minute. Blood pressure was unreadable. She had first and second heart sounds with no added heart sounds. Her respiratory rate was 18 cycles-per-minute and an oxygen saturation of 52% in room air. Her abdomen was full, moved with respiration, soft, tender, bowel sounds were not present but had free flowing colorless rice water fluids

exuding from her anus. She had cold clammy extremities and pale looking skin.

While plans were made to do her laboratory investigations (full blood count; blood culture; and serum electrolytes, urea and creatinine) the next day; she was admitted and commenced on 0.9% normal saline fluid at 1Litre per hour.

With this the blood pressure became readable at 90/80mmHg after 1L of Intravenous (IV) fluid, then 120/70 after 4L of IV fluid. Intranasal oxygen was administered at 3L/min, improving the saturation to 92%. Intravenous ciprofloxacin 200mg antibiotics 12hrly was commenced. Urethral catheter was inserted, but there was no urine production for the first 2 hours.

A working diagnosis of Acute Gastro-enteritis secondary to Cholera with hypovolemic shock and acute kidney injury was made.

Intravenous fluid administration was stopped 4 hours and 4 litres after presentation, due to financial constraints. 9 hours after presentation patient was still unconscious and blood pressure was unreadable. She had a respiratory rate of 30 cycles-per-minute, noisy labored breathing and wide spread coarse crepitations on chest auscultation. An assessment of fluid overload was made and fluid corrected to 1L 4hrly and Furosemide 80mg stat was administered. 10 hours after presentation she regained consciousness, patient requested and signed that oxygen administration should also be stopped. This was accompanied by drops in oxygen saturation. 11 hours after presentation, patient began to gasp in-between respiratory; all resuscitative measures proved abortive and she was confirmed clinically dead. At this point, total fluid input was 9500mls and urine output was 3000mls.

Discussion

In lay terms, mydriasis means physiologic or non-physiologic dilation of the pupils [1-2]. This usually causes bilateral dilation. Unilateral mydriasis is atypical and may denote the presence of an underlying pathology. Although, it can also be as a result of a drug response, often sympathomimetics or drugs with anticholinergic properties on the eyes. However, unilateral mydriasis is seen rather as a pathological phenomenon and has been associated with migraines in some studies but, these are rather benign and episodic [3]. Fixed unilateral mydriasis may be as a result of an optic nerve compression or Increased intracranial Pressure (ICP) as seen in Hutchinson Pupil [4].

Now, this patient presented with a non-reactive unilateral mydriasis. Non-reactive mydriasis is in clinical correlation with ICP. Although the most common cause of ICP are head traumas, it is also likely to be caused by a brain swelling [5]. Which in non-traumatic cases are commonly caused by dehydration and rapid rehydration of severe dehydration. As a result of neurologic deterioration following rapid correction [6-7]. However, this patient presented after receiving 5.5L of normal saline infusion from another facility. Giving credence to both possibilities.

Furthermore, she also presented with (signs of) stroke. Studies have shown that strokr may cause ICPs and also stroke may be caused by an ICP [5]. In her case, based on the history obtained, there was no clear history as to when the oral deviation occurred. Although it was noticed while receiving fluids at the point of primary care, necessitating her referral to our health facility. However, in our patient, who showed signs of dizziness, altered consciousness and unilaterally dilated pupils before the loss of nasolabial fold and mouth deviation, the stroke was most likely a complication of the raised ICP.

Notwithstanding, both health facilities appropriately tackled the severe dehydration as an emergency, but not the ICP. With knowledge that intractable increased intracranial pressure can cause death or devastating brain damage, it is important to recognize and control it early [8]. Now, there is a directly proportional relationship between decreasing pupillary reactivity and Increasing Intracranial Pressure (ICP), with the patients who have non-reactive pupils being at highest peaks of Increased Intracranial pressure [9].

Common failures to recognize and tackle ICPs in Nigeria may be as a result of the knowledge gap that exists in Neurological managements and maneuvers from her medical training. This gap is detrimental to the global surgery and non-surgical neurological interventions in Nigeria. As earlier mentioned, the most common cause of ICP lesions are head traumas and according to the World Health Organization, Nigeria has the highest rates of Road traffic Accidents (RTA) in Africa [5-10]. Thus, one will wonder if this gap in knowledge contributes to the fact that, RTA related deaths occurring in Nigeria are the highest proportion of deaths that occurring in the African continent [11]. Especially those with neurological involvements. However, the correlation between knowledge gaps in management of ICP and RTA related morbidity and mortality in Nigeria is beyond the scope of this paper and is open for future researches, medical training modifications and policy implementations.

Currently, treatments for raised ICP as commonly practiced in this region are either medically (Mannitol) or Surgically (Drainage of Cerebrospinal fluid or bleeds around brain or craniotomy to ease the swelling).

Conclusion

The Unilateral mydriasis observed in this patient was as a result of an Increased intracranial pressure (ICP). Increased Intracranial pressure (ICP) is a medical emergency. Early diagnosis and prompt treatment is very crucial to ensuring a good prognosis. Pathologies such as severe dehydration that can lead to ICP should be diagnosed early and treated promptly with the possibility of an ICP being kept in mind. ICP may cause devastating complications such as stroke, and even death. As seen in this patient.

Prevention and prompt treatment will need the efforts of improved health seeking behavior from the patients, sensitization of healthcare staff and trainees to spot the signs and act early.

Additional Note

It is paramount to note that, though this was an atypical presentation, more details on the pathology could have been ascertained if an autopsy was done. A neurosurgeon did suggest that the Unilateral mydriasis and mouth deviation could have been lateralizing manifesting. signs Hence, working with the theory that the stroke or an undiagnosed intracranial pathology resulted in these lateralizing signs and was the cause of the unilateral mydriasis and Increased intracranial pressure. However, poor history of onset of symptom also left him speculating. This though was a postmortem (informal) conversation that we were unable to backup, as there was no autopsy done. Thus, a major shortcoming to the advancement of neurology and

neuropathology in Nigeria is the perceived taboo associated with autopsy.

Recommendation

1. Patients with severe dehydration should be evaluated for signs of ICP.
2. Medical management should be commenced in time in patients showing signs of ICP to prevent further damage.
3. Cerebral oedema is a complication of dehydration it is also a complication of rehydration with devastating neurological damages. Thus, though rehydration should be commenced in due time, strict fluid load and rates should be calculated and adhered to so as to prevent fluid overload and rehydration neuropathology.

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Conflict of Interest

Authors declare no conflict of interest

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