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A Case Report of a 51 year old woman presenting with seizures

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ABSTRACT :

A 51-year-old lady who was having seizures arrived at the emergency room via ambulance. She lost consciousness while she was at work. There have been no recent episodes of fever, chills, rigors, coughing, chest discomfort, dyspnoea, palpitations, headaches, vision issues, syncope, weakness, paraesthesia's, ataxia, head injuries, rash, or incontinence of the bowels or bladder in the patient. Her weight hasn't changed recently, and she has a decent appetite. She routinely works out and does not use alcohol or smoke. Blood pressure is 122/66 mm Hg, the temperature is 36.6°C (98°F), and the heart rate is 90 beats per minute. Axial and sagittal maximum-intensity-projection images from CT angiography of the head (Panels B and C, respectively) show that the major intracranial arteries are all unremarkable, with no stenosis, irregularities, or occlusion (Figure 2). A chest radiograph was normal. Overnight observation and cardiac function monitoring were conducted on our patient. Intravenous normal saline, acetaminophen for a low-grade fever, and intravenous lorazepam at a modest dose (0.5–1 mg) were administered to her. The patient fared well; she did not experience any more seizures overnight, and her tremor lessened along with her blood pressure and heart rate returning to normal. With instructions to follow up with her primary care physician, the patient was released.

Keywords: Seizures, unconscious, neurology emergency.

Case Presentation

A 51-year-old lady who was having seizures arrived at the emergency room via ambulance. She lost consciousness while she was at work. When they heard a scream, a colleague moved fast to help. The patient's arms were flexed against his chest, causing the co-worker to witness a stiffening and elongation of his torso. The patient then jerked slightly before becoming unresponsive for four to five minutes. There was no urine incontinence during the incident, but there was hard breathing and excessive salivation. After being lethargic and disoriented for fifteen to thirty minutes, the patient became amnestic regarding the incident. The patient had coronavirus disease 2019 (Covid-19) 14 weeks earlier and had returned to work after 2 weeks. The patient denied any history of increasing despair, suicidal thoughts, or suicide attempts. There was also no family history of seizures. Blood pressure is 122/66 mm Hg, the temperature is 36.6°C (98°F), and the heart rate is 90 beats per minute. When inhaling room air, her respiration rate and oxygen saturation were within normal limits. Having a Glasgow Coma Scale score of 3, she is profoundly unconscious and unresponsive to painful stimuli. She doesn't have ankle oedema and is in good hydration. Everything looks normal on the head, ears, eyes, nose, and throat. Her rashes and tattoos are absent, and there is no indication of anaemia, cyanosis, or adenopathy. She has a flexible neck and no discernible thyroid gland. An palpable peak in the fifth lumbar intercostal space indicates appropriate jugular vein pressure. Lung clarity and heart sounds are both normal. Lacking organomegaly, the abdomen is supple.

Pupils are tiny and nonreactive; corneal, gag, and oculocephalic reflexes are absent; the skull and spine are normal; and the carotid arteries are equally palpable and free of bruits. Her cranial nerves, motor, and sensory systems are hard to evaluate due to obtundation. Her pupils were both symmetrically receptive to light, dilated to 6 mm, and her skin was somewhat flushed and diaphoretic. The patient had neither asterixis nor myoclonus, but a minor bilateral 10-Hz hand tremor. Her general medical and neurologic examination results were otherwise unremarkable and showed no meningeal symptoms.

Investigations

Forty minutes after the patients arrival, computed tomography (CT) angiography of the head and neck performed after the administration of intravenous contrast material (Figure 1). No evidence of infraction, haemorrhage, or intracranial or cervical arterial stenosis or occlusion. The dural venous sinuses and deep cerebral veins were present. Axial and sagittal maximum-intensity-projection images from CT angiography of the head (Panels B and C, respectively) show that the major intracranial arteries are all unremarkable, with no stenosis, irregularities, or occlusion (Figure 2). A chest radiograph was normal. Approximately 1 hour after the patients arrival, initial laboratory test results were received (Table 1). The blood ethanol level was undetectable and urine toxicologic screening was negative, as was screening for human chronic gonadotropin. A blood thyrotropin level was normal. Within 2 hours after the patients arrival, her aphasia abated; her speech was slow but fluent. On a repeat review of symptoms, she noted that she had left warm earlier in the day, but she reported no other symptoms.

Differential Diagnosis, Treatment Outcome & Follow-up

Based on the patient's history of non-responsiveness, observed convulsions, and postictal disorientation, a seizure is the most likely diagnosis. A generalized tonic-clonic seizure was consistent with the witness descriptions. While the clonic phase is characterized by cyclic jerking of the extremities, the tonic phase manifests as generalized rigidity. The scream came from a contraction of the diaphragmatic and chest wall muscles during the patient's seizure, forcing air through a constricted glottis. Given that the patient is quite young and did not exhibit any neurological abnormalities throughout her neurologic evaluation, a stroke is unlikely to have occurred. People who have a unilateral stroke usually do not appear to be unconscious because one hemisphere is enough to maintain consciousness, even in cases of chemia or bleeding. Although brainstem-related vertebrobasilar strokes might cause unconsciousness, a neurologic examination should show signs of brainstem dysfunction, such as anomalies in pupil or ocular motor function or weakness in the extremities.

Since our patient did not have a history of head trauma or show any evidence of head trauma on initial examination, it is unlikely that head trauma or a concussion following a fall caused our patient's seizure. The main goal should be to make appropriate steps to rule out syncope, seizures, and other causes of momentary unconsciousness.

Over the course of the following thirty minutes in the emergency room, the patient's bewilderment subsided, and she did not experience any further seizures. Serum glucose, electrolytes, and the total blood cell count were all within normal ranges. Electrocardiography with 12 leads and chest radiography were both normal. Without contrast enhancement, a head computed tomography (CT) image revealed normal brain parenchyma and no signs of a tumour, bleeding, or stroke. The patient was monitored in the emergency room while receiving oxygen through a nasal cannula.

Diarrhea and flushing are possible symptoms of carcinoid syndrome, which is brought on by neuroendocrine tumours that release vasoactive chemicals. The patient did not have any of the common gastrointestinal (GI) symptoms associated with this diagnosis, like diarrhea. Serotonin and other vasoactive molecules are suddenly released during a carcinoid crisis, which can result in pro-found flushes, hemodynamic instability, bronchoconstriction, GI symptoms, and protracted lethargy that lasts for hours or even days. In addition to postictal disorientation, tremor, flushing, and a single generalized tonic-clonic seizure, our patient did not exhibit any other signs of carcinoid illness or crisis. [1]

A peculiar and sometimes deadly reaction to dopamine antagonists, neuroleptic malignant syndrome is typified by altered consciousness, heat, stiffness, and autonomic dysfunction. [2] When levodopa or dopaminergic agonists are abruptly stopped in patients with Parkinson's disease, neuroleptic malignant syndrome may also result. This diagnosis is highly doubtful because the patient has not been exposed to dopamine antagonists that are known to exist.

Confusion, agitation, hyperthermia, diaphoresis, tachycardia, and tremor are the hallmarks of serotonin syndrome.3. Patients using one or more drugs that raise serotonin levels, such as tricyclic antidepressants, monoamine oxidase inhibitors, or selective serotonin reuptake inhibitors (SSRIs), are generally considered to be part of the "at-risk" population. It is crucial to stress that serotonin syndrome can result from consuming even one antidepressant. Serotonin syndrome was suggested by the patient's symptoms of tremor, diaphoresis, tachycardia, hypertension, and heat. In her instance, the SSRI effects of both the tramadol and the mirtazapine exacerbated her serotonin syndrome.

Overnight observation and cardiac function monitoring were conducted on our patient. Intravenous normal saline, acetaminophen for a low-grade fever, and intravenous lorazepam at a modest dose (0.5–1 mg) were administered to her. The patient fared well; she did not experience any more seizures overnight, and her tremor lessened along with her blood pressure and heart rate returning to normal. With instructions to follow up with her primary care physician, the patient was released.

Figures:

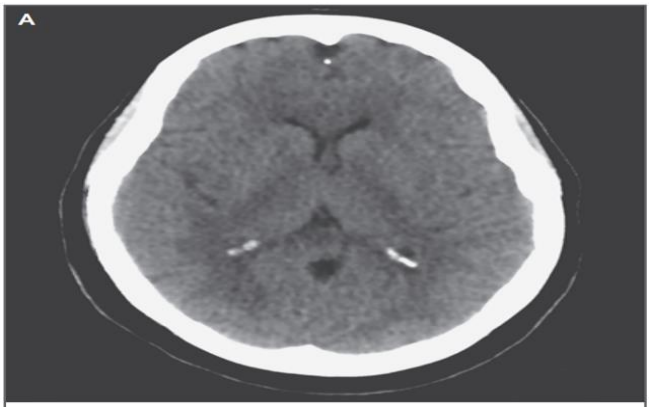

Figure 1A : CT angiography of the head

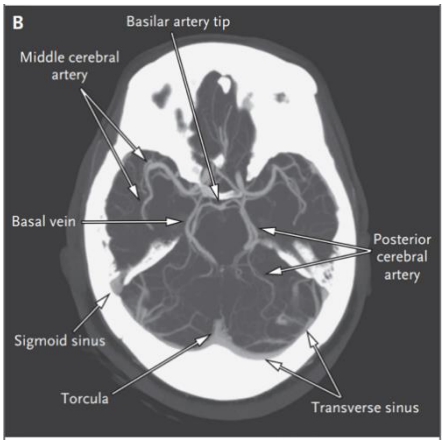

Figure 1B : CT angiography of the head

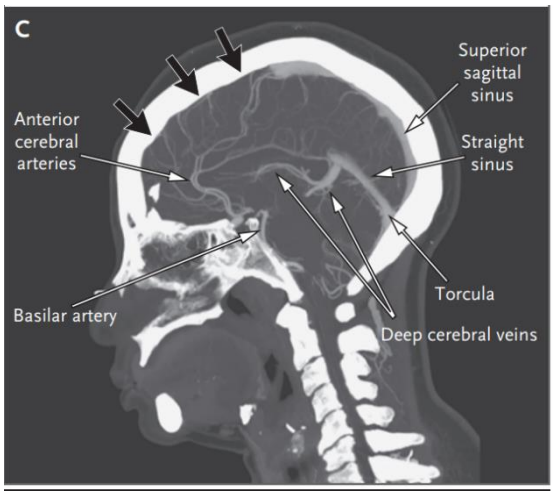

Figure 2: Can axial image from CT of the Head (Panel A).

Variable	Reference Range, Adults†	1 Hr after Arrival	3 Hr after Arrival
Hemoglobin (g/dl)	12.0–16.0	8.5	7.6
Hematocrit (%)	36.0–46.0	25.4	22.1
White-cell count (per μl)	4500–11,000	16,200	19,570
Platelet count (per μl)	150,000–400,000	5000	7000
High-sensitivity troponin T (ng/liter)	0–9	72	105
Erythrocyte sedimentation rate (mm/hr)	0–20	50	—
Lactate (mmol/liter)	0.5–2.0	2.4	—
C-reactive protein (mg/liter)	<8.0	26.7	—
Sodium (mmol/liter)	135–145	138	—
Potassium (mmol/liter)	3.4–5.0	2.5	—
Chloride (mmol/liter)	98–108	97	—
Carbon dioxide (mmol/liter)	23–32	27	—
Urea nitrogen (mg/dl)	8–25	21	—
Creatinine (mg/dl)	0.60–1.50	1.07	—
Glucose (mg/dl)	70–110	130	—
Alanine aminotransferase (U/liter)	7–33	18	—
Aspartate aminotransferase (U/liter)	9–32	50	—
Alkaline phosphatase (U/liter)	45–115	50	—
Total bilirubin (mg/dl)	0.0–1.0	2.6	—
Direct bilirubin (mg/dl)	0.0–0.4	0.3	—
Prothrombin time (sec)	11.5–14.5	14.8	15.2
International normalized ratio	0.9–1.1	1.2	1.2
Partial-thromboplastin time (sec)	22.0–36.0	—	28.8
D-dimer (ng/ml)	<500	—	3857
Fibrinogen (mg/dl)	150–400	—	291
Haptoglobin (mg/dl)	30–200	—	<10
Lactate dehydrogenase (U/liter)	110–210	—	1495
Reticulocytes (%)	0.7–2.5	—	5.8

Table 1: Lab Findings

Discussion

Clinical diagnosis of serotonin syndrome can be established following the identification of the symptoms and indicators and a thorough assessment of the patient's drug regimen. Serotonin syndrome [3–8] is a triad of altered mental status, autonomic dysfunction, and neuro-muscular abnormalities caused by excessive serotonergic activity in the central and peripheral nervous systems. The diagnostic criteria for serotonin syndrome were updated by Radomski et al. [8]. Now, a serotonergic medicine must be used in addition to or at an enhanced dosage, and there must be at least four significant symptoms or three major plus two mild symptoms. Furthermore, the symptoms cannot be the result of a recent neuroleptic agent treatment; an infectious, toxic-metabolic, endocrine, or previous mental illness.

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