Psychogenic Stroke: A Case Report

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Abstract—Stroke mimics complicate stroke diagnosis in the emergency department. Here, we present a case, diagnosed as psychogenic stroke, in which the patient presented with right-sided facial deviation, slurred speech, and right-sided weakness, after receiving bad news. Her imaging findings were unremarkable, and her symptoms resolved entirely after two hours. We conclude with several remarks about the relationship between emotions and stroke and the challenges of diagnosing psychogenic stroke.

Index Terms—Psychogenic stroke, pseudostroke, stroke mimics

I. INTRODUCTION

Stroke mimics make stroke diagnosis challenging in the emergency department setting. As many as one out of four suspected stroke cases is found to be a stroke mimic [1]. As the healthcare system works to reduce the door-to-needle time for thrombolysis administration, the incidence of stroke mimic cases receiving thrombolytics has drastically increased, with an upward trend of 3.5% to 4.1% [2-5].

While there is minimal information available about psychogenic pseudostroke (PS), the condition is far more widespread than the medical community acknowledges. However, there is no epidemiological evidence of PS. It is thought that the prevalence of functional paralysis is comparable to that of multiple sclerosis (5 per 100,000); as the majority of PS cases present with some degree of motor impairment, this percentage should be considered a close estimate [6]. The reason for the scarcity of data on PS is unknown, but two aspects should be considered as contributors: under-reporting of PS or, more significantly, over-reporting of PS as a genuine stroke. Both result from a lack of confidence, or a reluctance, to reveal the actual diagnosis [6,7].

When acute symptoms are indicative of a stroke but are of psychogenic origin, this is referred to as PS [6]. The term ‘psychogenic PS’ encompasses a range of nonorganic acute stroke-like presentations, including malingering. There are many indicators of PS, including as a history of other unresolved conditions that have previously been investigated to no apparent benefit; a lengthy history of repetitive ‘transient ischaemic attacks’; a background of mental disorders; the existence of emotional or situational triggers; and symptoms provoked or relieved by placebo [6,8]. The inconsistency of the diagnostic tests and the absence of objective indicators of disease are essential aspects to be scrutinized with regard to this condition. PS is difficult to diagnose and manage, and even in instances where the neurologist knows the accurate diagnosis is PS, there is frequently a reluctance to disclose the diagnosis [6].

The most challenging problem in the diagnosis and treatment of PS is the administration of intravenous (IV) recombinant tissue plasminogen activator (rt-PA) [6] — when the diagnosis is ambiguous but leans toward PS, the neurologist faces the danger of symptomatic intracerebral haemorrhage (sICH) [9].

As examination approaches and clinical signs for determining functional weakness, speech disorder, and numbness become more widely recognised, it is critical to consider psychogenic stroke, among other mimickers. Here, we present a case of psychogenic stroke that resolved after a period of observation.

CASE PRESENTATION

A 38-year-old female presented to the emergency department (ED) complaining of right-sided facial deviation, slurred speech and right-sided weakness, commencing two hours prior, shortly after she had woken up and received bad news. She had no fever or headache and had not sustained head trauma.

The patient had no medical or surgical history,
either past or present. She denied ever using any medication, including oral contraceptives. She also denied smoking, drinking, or using illegal drugs.

Clinically, she presented with a normal level of consciousness, but appeared anxious. Neurological examination revealed facial asymmetry with right-sided mouth deviation and absence of the nasolabial fold (lower face). She also presented with dysarthria with right arm and leg drift. The initial NIH scale was 6 points. All her vital signs were within normal range, including her blood glucose level (4.6 mmol/L).

A stroke protocol was activated. A computed tomography (CT) scan revealed no acute territorial infarction or intracranial haemorrhage. In addition, computed tomography angiography (CTA) revealed the absence of occlusion, significant flow-limiting stenosis, or aneurysmal dilatation of the carotids and the circle of Willis.

Laboratory analysis revealed a white blood cell (WBC) count of 12.19x10³/μl, haemoglobin level of 12.5 g/dl, and a platelet count of 212,000 per microlitre of blood. The serum sodium level was 141 mmol/l, the potassium level was 3.89 mmol/l, the chloride level was 108 mmol/l, and the corrected calcium was 2.39 mmol/l. Other laboratory parameters, including urea, creatinine, anion gap, hepatic function panel, coagulation profile, and troponin, were all within the normal limits.

After two hours of observation in the ED, the patient recovered completely, became more comfortable, and was discharged. After two months, a follow-up phone call was made: The patient complained of intermittent headaches and right-sided numbness (upper and lower); the facial deviation and slurred speech had not recurred.

II. DISCUSSION

There are two points to consider in this case report. First, that a neurological deficit was triggered by an emotional event. Second, the complete resolution of symptoms which might hint towards a transient ischaemic attack (TIA). It is prudent to assert that anger and emotional distress shortly before a stroke are relatively common and relate to all strokes, particularly intracranial haemorrhage [10]. In one study, anger or emotional distress were recorded by 9.2% (n = 1233) of individuals during the case period and 8.3% (n = 1111) in the control period. Those who expressed anger or emotional disturbance throughout the case period were more likely to have a history of diabetes, angina, hypertension, depression, or myocardial infarction and less likely to use cardiovascular preventive drugs [10]. In another study, in which 151 stroke patients were interviewed, acute psychological stress was linked to a 3.4-fold increase in stroke risk over the following two hours, compared with no exposure to these triggers (95% confidence interval 1.55 - 7.50) [11].

Although chronic risk factors for stroke are fairly well documented, acute precipitants, or triggers, of stroke are relatively unexplored. Therefore, a hospital-based observational cross-sectional study was conducted in which 290 patients took part. Psychological stress was present in 51 (17.6%) patients, with stressful life events accounting for 34 (11.7%), negative affect accounting for 17 (5.9%), and rage accounting for 12 (4.1%). In hazard periods, none of the patients reported exposure to recreational drug abuse, frightening events, or very vigorous physical effort [12]. Another study examined the relationship between exposure to various possible triggers – including anger, sudden postural changes in response to a startling experience, negative and positive emotions, excessive eating, intense physical exercise, and abrupt changes in temperature during waking hours – and the onset of acute ischaemic stroke. In their case-crossover research, a validated questionnaire was used to interview 200 stroke patients 1-4 days following the incident. 76 patients (38%) reported exposure to at least one of the study triggers, with the odds ratio for negative emotions being 14.0 (CI 95% 4.4 - 89.7), anger 14.0 (CI 95% 2.8 - 253.6), and abrupt changes in body posture in reaction to a startling experience 24.0 (CI 95% 5.1 - 428.9). The study concluded, therefore, that negative emotions, anger, and abrupt postural changes in reaction to a startling incident appear to be independent triggers for ischaemic stroke [13]. A systematic review of possible ischaemic stroke triggers revealed anger, negative or positive emotions, binge eating, psychological suffering, and a rapid postural shift in response to a frightening incident, all to be possible triggers [14].

Furthermore, the possibility that our patient’s
symptoms resemble a TIA is also debatable. In 2014, a retrospective chart study on TIA patients revealed no acute abnormalities in the group tested with CT, but minor ischaemic infarcts were observed in 44% of the participants tested by MRI [15]. Multimodal MR imaging is preferred in patients with TIA, and non-contrast computed tomography (NCCT) should be acquired only if MR imaging is unavailable, as NCCT has little utility in individuals whose symptoms have resolved [16]. Diffusion-weighted imaging (DWI) can detect lesions in around 40% of TIA patients, and DWI positivity in TIA patients is associated with an increased risk of recurrent ischaemic episodes [17]. In cases of psychogenic stroke, MRI is recommended to detect any minor infarction. Given that our patient later developed some neurological deficit in the form of numbness, an MRI would be recommended for cases of possible psychogenic stroke even if symptoms had resolved. In our case, the patient did not have an MRI before being discharged, highlighting the importance of follow-up.

Nevertheless, whether to withhold treatment based on a specific presentation remains unanswered. Improving clinical pathways might not be sufficient to resolve the misdiagnosis of stroke mimic cases; however, improving clinical skills and knowledge about mimics can better the approach to such cases.

PS management requires an interdisciplinary approach, involving teamwork between neurologists, psychologists, psychiatrists, and social workers [6]. Even if the patient has received IV rt-PA, efforts must be made to reduce their length of stay after the hyperacute stage and once the affirmative diagnosis of PS has been obtained. In PS patients, unnecessary stroke prevention drugs such as clopidogrel should be deferred as much as feasible [6]. While it is not a grave mistake to give IV rt-PA to a PS patient whose diagnosis is uncertain and whose risk of haemorrhage is low, failure to communicate the facts and leaving the patient with the impression that they have experienced an actual stroke is an error of judgment. Moreover, the recurrence rate of PS due to psychopathological or benefit-seeking situations is undetermined [6].

III. Conclusion

Psychogenic strokes must be evaluated as if they were actual strokes. Moreover, we emphasise the importance of follow-up examinations, as they are frequently used to arrive at a final diagnosis. Patients who present with neurological deficits triggered by emotional upset should be treated as if they had an ischaemic stroke; investigation and treatment should not be delayed while a psychogenic stroke is considered.

IV. References

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