Psychogenic Stroke: A Case Report

Ghadah Ayad AlEnizi and Mohamad Bakir

***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,

Ghadah Ayad AlEnizi is with Emergency department, King Fahd Medical City, Saudi Arabia, e-mail: Ghada_alonazy@hotmail.com

Mohamad Bakir is with College of Medicine, Alfaisal University, Riyadh, Saudi Arabia, e-mail: Mo7ammedbakir@gmail.com (Corresponding author).

DOI.10.52609/jmlph.v2i3.51

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 $12.19x10^3$/Ul, 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 TIAs, 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

[1] Jones AT, O'Connell NK, David AS. Epidemiology of functional stroke mimic patients: a systematic review and meta-analysis. Eur J Neurol. 2020; 27:18–26. doi: 10.1111/ene.14069

[2] Ali-Ahmed F, Federspiel JJ, Liang L, Xu H, Sevilis T, Hernandez AF, et al. Intravenous tissue plasminogen activator in stroke mimics. Circ Cardiovasc Qual Outcomes. 2019; 12:e005609. doi: 10.1161/CIRCOUTCOMES.119.00560

[3] Liberman AL, Liotta EM, Caprio FZ, Ruff I, Maas MB, Bernstein RA, et al. Do efforts to decrease door-to-needle time risk increasing stroke mimic treatment rates? Neurol Clin Pract. 2015; 5:247–252. doi: 10.1212/CPJ.0000000000000122

[4] Burton TM, Luby M, Nadareishvili Z, Benson RT, Lynch JK, Latour LL, et al. Effects of increasing IV tPA-treated stroke mimic rates at CT-based centers on clinical outcomes. Neurology. 2017; 89:343–348. doi: 10.1212/WNL.0000000000004149

[5] Keselman B, Cooray C, Vanhooren G, Bassi P, Consoli D, Nichelli P, et al. Intravenous thrombolysis in stroke mimics: results from the SITS International Stroke Thrombolysis Register. Eur J Neurol. 2019; 26:1091–1097. doi: 10.1111/ene.13944

[6] Behrouz R, Benbadis SR. Psychogenic pseudostroke. J Stroke Cerebrovasc Dis. 2014 Apr;23(4):e243-8. doi: 10.1016/j.jstrokecerebrovasdis.2013.11.010. Epub 2014 Jan 16. PMID: 24439129.

[7] Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. J Neurol Neurosurg Psychiatry. 2005 Mar;76 Suppl 1(Suppl 1):i2-12. doi: 10.1136/jnnp.2004.061655. PMID: 15718217; PMCID: PMC1765681.

[8]. Lim EC, Seet RC. What is the place for placebo in the management of psychogenic dis-

ease? J R Soc Med. 2007 Feb;100(2):60-1. doi: 10.1177/014107680710000202. PMID: 17277261; PMCID: PMC1790983.

[9]. Chernyshev OY, Martin-Schild S, Albright KC, Barreto A, Misra V, Acosta I, Grotta JC, Savitz SI. Safety of tPA in stroke mimics and neuroimaging-negative cerebral ischemia. Neurology. 2010 Apr 27;74(17):1340-5. doi: 10.1212/WNL.0b013e3181dad5a6. Epub 2010 Mar 24. PMID: 20335564; PMCID: PMC2875935.

[10] Smyth A, O'Donnell M, Hankey GJ, Rangarajan S, Lopez-Jaramillo P, Xavier D, Zhang H, Canavan M, Damasceno A, Langhorne P, Avezum A, Pogosova N, Oguz A, Yusuf S; INTERSTROKE investigators. Anger or emotional upset and heavy physical exertion as triggers of stroke: the INTERSTROKE study. Eur Heart J. 2022 Jan 25;43(3):202-209. doi: 10.1093/eurheartj/ehab738. PMID: 34850877.

[11] Prasad M, Khanna P, Katyal VK, Verma R. Acute Psychological Stress is a Trigger for Stroke: A Case-Crossover Study. J Stroke Cerebrovasc Dis. 2020 Jun;29(6):104799. doi: 10.1016/j.jstrokecerebrovasdis.2020.104799. Epub 2020 Apr 2. PMID: 32249204.

[12] Sharma A, Prasad K, Padma MV, Tripathi M, Bhatia R, Singh MB, Sharma A. Prevalence of triggering factors in acute stroke: hospital-based observational cross-sectional study. J Stroke Cerebrovasc Dis. 2015 Feb;24(2):337-47. doi:10.1016/j.jstrokecerebrovasdis.2014.08.033. Epub 2014 Nov 22. PMID: 25444031.

[13] Koton S, Tanne D, Bornstein NM, Green MS. Triggering risk factors for ischemic stroke: a case-crossover study. Neurology. 2004 Dec 14;63(11):2006-10. doi: 10.1212/01.wnl.0000145842.25520.a2. PMID: 15596741.

[14] Guiraud V, Amor MB, Mas JL, Touzé E. Triggers of ischemic stroke: a systematic review. Stroke. 2010 Nov;41(11):2669-77. doi: 10.1161/STROKEAHA.110.597443. Epub 2010 Oct 14. PMID: 20947837.

[15] Sidorov EV, Feng W, Selim M. Cost-Minimization Analysis of Computed Tomography versus Magnetic Resonance Imaging in the Evaluation of Patients with Transient Ischemic Attacks at a Large Academic Center. Cerebrovasc Dis Extra. 2014 Mar 28;4(1):69-76. doi: 10.1159/000360521. PMID: 24803915; PMCID: PMC3995378.

[16] Easton JD, Saver JL, Albers GW, Alberts MJ, Chaturvedi S, Feldmann E, Hatsukami TS, Higashida RT, Johnston SC, Kidwell CS, Lutsep HL, Miller E, Sacco RL; American Heart Association; American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; Interdisciplinary Council on Peripheral Vascular Disease. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. Stroke. 2009 Jun;40(6):2276-93. doi: 10.1161/STROKEAHA.108.192218. Epub 2009 May 7. PMID: 19423857.

[17] Wintermark M, Sanelli PC, Albers GW,Bello J, Derdeyn C, Hetts SW, Johnson MH, Kidwell C, Lev MH, Liebeskind DS, Rowley H, Schaefer PW, Sunshine JL, Zaharchuk G, Meltzer CC. Imaging recommendations for acute stroke and transient ischemic attack patients: A joint statement by the American Society of Neuroradiology, the American College of Radiology, and the Society of NeuroInterventional Surgery. AJNR Am J Neuroradiol. 2013 Nov-Dec;34(11):E117-27. doi: 10.3174/ajnr.A3690. Epub 2013 Aug 1. PMID: 23907247; PMCID: PMC4072500.