Case Report

Isolated Medial PICA Territorial Ischemic Infarction with an NIHSS of 0 — Emphasis on the Neurological Exam to Guide Urgent Recanalization with tPA

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Abstract

Posterior circulation stroke is a potentially life threatening and functionally disabling stroke. Nevertheless, patients with posterior circulation stroke may initially present with a constellation of nonspecific symptoms mimicking other pathologies, causing delay in diagnosis. With the improvement of acute stroke management, patients who have a timely diagnosis of acute ischemic stroke can benefit tremendously from intravenous thrombolysis or mechanical thrombectomy. We present a patient with acute posterior circulation ischemia who presented with dizziness and acute vestibular syndrome. Although his NIHSS was 0, he had significant truncal ataxia, being unable to even ambulate. He was treated successful with intravenous tPA. Further workup revealed a medial PICA territorial infarction from large vessel atherosclerosis. Medial PICA infarction is a common cause of isolated truncal ataxia and NIHSS 0 strokes. We reviewed the common presentation of posterior circulation ischemia, challenges and pitfalls in diagnoses and the limitation of the current stroke scale in quantifying posterior circulation stroke severity. A clinical approach tailored to accurate detection of posterior circulation ischemia is outlined in this case report.

ABBREVIATIONS

PICA: Posterior Inferior Cerebellar Artery; NIHSS: National Institutes of Health Stroke Scale; tPA: tissue Plasminogen Activator

INTRODUCTION

Patients presenting to the Emergency Department with acute onset dizziness can be a diagnostic challenge. In patients presenting with the vague complaint of dizziness, 3.2%-4.1% are in fact having a cerebrovascular accident [1]. In patient suffering from acute posterior circulation strokes, a recent study comprising 407 patients did demonstrate that the most common patient presentation was dizziness [2]. Other than dizziness, clinical symptoms commonly associated with posterior circulation ischemia include occipital headache, gait unsteadiness, nausea, vomiting, and vertigo [3]. It is not surprising that an acute posterior circulation infarct can be missed by even experienced physicians as these symptoms are sometimes attributed to more common pathologies such as migraine, gastroenteritis, and presyncope [4].

We report a patient who presented to the ED with new-onset dizziness with persistent nausea and vomiting (acute vestibular syndrome). The diagnostic approach and pitfalls to diagnosis will be discussed in this case report.

CASE PRESENTATION

This is a 64-year-old man presenting to the emergency department complaining of vomiting. He states that about 5 pm, he began having persistent dizziness, headache, nausea and vomiting. The dizziness he describes as if the room is moving or spinning. He describes the headache as throbbing and pounding in nature starting from his left temporal region and radiating to the left suboccipital region. He denied weakness or blurry vision. His initial vital signs showed blood pressure of 170/90 mmHg and heart rate of 76. The ED physician noticed an ataxic gait and activated the stroke pager. Upon initially stroke assessment, his NIHSS was 0. However, his stance was wide based with an inability to ambulate. A non-contrast CT scan of the head showed no acute intracranial process. Although there was a strong suspicion for an acute ischemic infarction, NIHSS was 0 and tPA was initially held. However, CT perfusion did reveal an area of penumbra in the left cerebellar region (Figure 3). Intravenous thrombolysis was administered after the results of the CT perfusion. Afterwards, he was admitted for a stroke workup. An echocardiogram was unremarkable, and telemetry revealed a normal sinus rhythm. His CTA of the neck was significant for occlusion at the level of the V1 segment of the left vertebral artery with absent of the V2 segment but reconstitution of the V3 and V4 segment s MRI of the brain revealed diffusion restriction in the inferior vermis and the medial posterior inferior cerebellar hemisphere (Figure 1). Neither the superior vermis nor the more lateral posterior...
inferior cerebellar hemisphere showed any area of diffusion restriction. By HOD3, he was able to walk with a wide based gait and smaller stride lengths. Atherosclerotic disease of the ipsilateral vertebral artery was thought to be the etiology of his PICA infarction and he was started on dual antiplatelet therapy. He had an uncomplicated hospital stay and he was discharged home on HOD10 with improved condition.

DISCUSSION

The National Institutes of Health Stroke Scale (NIHSS) is a standardized and easily reproducible quick exam that is most often used to evaluate a patient suspected of having an acute ischemic stroke [4]. Typically, a NIHSS of 3 or 4 represents minor deficits [5] caused by small vessel occlusion, whereas a larger NIHSS usually represents major deficits caused by large artery occlusion [6]. The parameters assessed on the NIHSS is primarily focused on neurological deficits typically seen in anterior circulation ischemia meaning that the average NIHSS representing major deficits tends to be lower for posterior circulation ischemia. This is exemplified by a 2014 study assessing 372 patients presenting with acute posterior circulation stroke demonstrating an average NIHSS of 2 [7].

Furthermore, an NIHSS of 0 does not invariably rule out an acute cerebrovascular event. A recent study evaluated 2,618 patients presenting with acute ischemic stroke showed that 0.76% of presenting patients had an NIHSS of 0. In these patients, truncal ataxia from a midline cerebellar infarction was the most common presentation [4]. Since the NIHSS does not assess for this type of ataxia [4], an NIHSS of 0 may in fact mislead the treating physician, resulting in a missed diagnosis. Savitz et al. examined 15 patients over a period of 5 years who had a missed diagnosis of cerebellar infarction. At least 9 of these patients did not have their gait assessed [3]. An assessment of these patients' gait may have revealed a wide-based gait indicative of truncal ataxia.

A lesion of the cerebellar vermis can cause truncal ataxia and postural instability without associated limb ataxia [8]. Occlusion of one of the vessels perfusing the vermis can produce such a lesion. The inferior cerebellar vermis gets its blood supply from the posterior inferior cerebellar artery (PICA) [8]. The MRI of the patient in this case report showed infarction in the inferior vermis, consistent with an infarction along the PICA distribution [8]. The triangular pattern of medial cerebellar hemispheric involvement also fits the pattern seen in infarction along the medial PICA territory [8].

Perhaps the most salient point in this case report is that the NIHSS is insufficient in evaluating posterior circulation infarction and in some cases, an NIHSS of 0 can be seen in an isolated cerebellar infarction. Although there was some cerebellar hemisphere involvement seen on DWI in this patient, limb ataxia has been shown to be less frequent when the medial cerebellar hemispheric region is solely involved because the dentate nucleus is typically spared [9]. In patients presenting with significant limb paresis, the NIHSS will be high and thus gait assessment may be less relevant. However, for a patient with only posterior circulation symptoms, it is paramount for gait to be assessed - an impaired gait may be the only positive neurological finding. In a patient with profound truncal ataxic, the gait is expected to be wide based. In addition to gait assessment, careful attention to the eye movements is important because the flocculonodular lobe is also supplied by the medial PICA and thus lesion in this region will cause various eye movement abnormalities such as nystagmus and skewed deviation [10].

In this case report, further image assessment with CT perfusion did reveal posterior circulation ischemia (Figure 3). Caution should however be made in putting more emphasis on CT perfusion than a targeted neurological exam for diagnosis of acute posterior circulation ischemia; a recent 2016 retrospective
series only revealed a 76.6% sensitivity of CT perfusion in the
diagnosis of posterior circulation ischemia in the acute setting
[11,12]. An alternative approach to the management of suspected
posterior circulation ischemic in a patient presenting with
dizziness and acute vestibular syndrome, is to do a stat MRI of the
brain. However, this approach has several shortcomings. Firstly,
itis less time efficient than a more focused neurological exam
thus making this approach ill-advised if reperfusion therapy with
thrombolytics is being considered. Secondly, it is more costly
and less readily available than a neurological exam. Lastly, the
HINTS exam in combination with the NIHSS is significantly more
sensitive than an MRI in detecting acute posterior circulation
strokes. The sensitivity of MRI in the first 24 hours of a posterior
circulation infarction is 80% [11] whereas the sensitivity and
specificity of the HINTS exam plus NIHSS was found to be 96.8%
and 98.5% in a recent prospective of 190 patients [11]. The
addition of gait assessment further reduces false negative rates.
Profound truncal ataxia (wide based stance with the inability
to walk) is very specific for a central lesion and is not seen in
peripheral causes of dizziness with acute vestibular syndrome

CONCLUSION

Finally, the morbidity and mortality of a missed cerebellar
stroke is high with many patients having long-term gait
impairment [3]. A focused neurological exam incorporating
HINTS exam and gait assessment will improve the accuracy of
early detection of posterior circulation ischemia. This enables
earlier reperfusion therapy thus reducing morbidity and

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