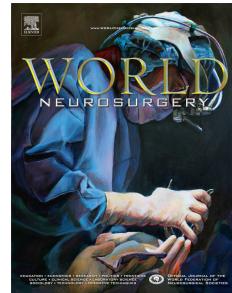


Accepted Manuscript

Recrudescence of symptoms of remote ischemic stroke after a cerebral angiogram:
Report of a case

Stephanie R. Falatko, DO, Philip G.R. Schmalz, MD, Mark Harrigan, MD



PII: S1878-8750(17)30296-6

DOI: [10.1016/j.wneu.2017.02.122](https://doi.org/10.1016/j.wneu.2017.02.122)

Reference: WNEU 5353

To appear in: *World Neurosurgery*

Received Date: 2 January 2017

Revised Date: 24 February 2017

Accepted Date: 27 February 2017

Please cite this article as: Falatko SR, Schmalz PGR, Harrigan M, Recrudescence of symptoms of remote ischemic stroke after a cerebral angiogram: Report of a case, *World Neurosurgery* (2017), doi: [10.1016/j.wneu.2017.02.122](https://doi.org/10.1016/j.wneu.2017.02.122).

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Recrudescence of symptoms of remote ischemic stroke after a cerebral angiogram: Report of a case

Stephanie R. Falatko DO¹, Philip G. R. Schmalz MD¹, and Mark Harrigan MD¹

¹Department of Neurosurgery, University of Alabama Birmingham, Birmingham, Alabama USA

The authors have no funding or industry affiliations to disclose.

Corresponding author: Stephanie R. Falatko, D.O.

Contact information: Faculty Office Tower 1005

510 20th Street South

Birmingham, AL 35294

Email: srfalatko@uabmc.edu

KEY WORDS: anamnestic recall, angiogram, recrudescence, stroke mimic, subarachnoid hemorrhage

ABBREVIATIONS: ADC Apparent diffusion coefficient; CT computed tomography; DFP dysarthria-facial paresis; FLAIR fluid- attenuated inversion recovery; IV intravenous; MRI magnetic resonance imaging; TIA transient ischemic attack; DWI diffusion weighted imaging

ABSTRACT

BACKGROUND: Recrudescence, or reappearance of previously resolved symptoms of ischemic stroke, may occur after physiological stress. Although this syndrome is generally thought to be uncommon, it may actually account for a significant proportion of stroke mimics.

CASE DESCRIPTION: A 67-year-old man was admitted with a Hunt-Hess grade II spontaneous subarachnoid hemorrhage. He underwent a digital subtraction cerebral angiogram as part of his imaging evaluation. About 30 minutes after the procedure, he developed dysarthria, right facial droop and a right pronator drift. The patient and family denied a history of similar symptoms or previous ischemic stroke. A brain MRI demonstrated a remote left lacunar infarction. The patient's symptoms resolved after 24 hours and were attributed to recrudescence of his previous lacunar infarction. It is likely that the physiological stress of the subarachnoid hemorrhage, combined with the cerebral angiogram, triggered the event.

CONCLUSION: Recrudescence of symptoms due to a previous stroke may be initiated by subarachnoid hemorrhage and/or a cerebral angiogram. The possibility of ischemic stroke recrudescence should be kept in mind as a possible stroke mimic.

KEY WORDS: anamnestic recall, angiogram, recrudescence, stroke mimic, subarachnoid hemorrhage

BACKGROUND

Reappearance of previously resolved symptoms of a remote ischemic stroke is termed *recrudescence of stroke symptoms* and has also been referred to as anamnestic syndrome. Recrudescence is typically triggered by physiological stressors, including metabolic derangements, infection, or fatigue.^{1,2} Recrudescence has also been associated with some medications and is an important stroke mimic.² Stroke mimics account for as many as 30% of stroke-related presentations; with 42% of these patients having a history of previous infarction.^{3,4} Here we report the only case of recrudescence represented in the neurosurgical literature that appeared in a patient after subarachnoid hemorrhage and a cerebral angiogram.

CASE DESCRIPTION

A 67-year-old left-handed man with a history of hypertension, hyperlipidemia, and type 1 diabetes mellitus presented with a severe thunderclap headache. Computed tomography (CT) demonstrated hyperattenuation within the prepontine and interpeduncular cisterns (Figure 1). A CT angiogram did not demonstrate an aneurysm or other vascular anomaly to explain the subarachnoid hemorrhage. A digital subtraction angiogram was performed on hospital day two. The patient received fentanyl, 25 mcg IV and midazolam, 1 mg IV for analgesia and sedation during the procedure. The cervical-cerebral angiogram was unremarkable for vascular abnormality or atherosclerotic disease. Serial neurological assessment was performed throughout the angiogram. No neurological changes were identified during the procedure and the patient was returned to the intensive care unit in stable condition.

Approximately 30 minutes after the procedure, the patient developed dysarthria, right lower facial paresis, and subtle right upper extremity drift (NIH Stroke Scale Score: 3). The medical history was reviewed in depth and both the patient and the family denied any knowledge of a previous stroke or stroke-like symptoms. An emergent brain MRI was obtained. Diffusion weighted imaging (DWI) did not show evidence of an acute ischemic stroke (Figure 2). However, a chronic lacunar infarction with surrounding gliosis and T2 FLAIR signal consistent with microangiopathic change was seen in the left centrum semiovale, corona radiata, and basal ganglia (Figure 3).

Additional work-up confirmed the absence of electrolyte derangement or underlying infection. All sedating medications were held and long term electroencephalogram showed no evidence of epileptiform discharges or focal features. A multidisciplinary case conference review corroborated the diagnosis of recrudescence of remote ischemic stroke in the setting of physiological stress. Within hours the patient's dysarthria and facial paresis resolved, followed by complete resolution of upper extremity weakness the following day. The patient was diagnosed with perimesencephalic nonaneurysmal subarachnoid hemorrhage, complicated by transient recrudescence of stroke symptoms. The patient recovered completely and was discharged home on hospital day three.

DISCUSSION

This patient's transient neurological symptoms were most likely an occurrence of recrudescence. Other causes of the witnessed neurological deficit, such as a new ischemic stroke, transient ischemic attack or ongoing seizures, were either excluded or determined to be unlikely. The dysarthria and facial symptoms resolved within several hours. The arm weakness, however, persisted beyond 24 hours and resolved by discharge. Gradual improvement with removal of triggers is characteristic of anamnestic syndrome as opposed to transient ischemic attack in which complete symptom resolution occurs within 24 hours.^{2,5}

The patient experienced several possible triggers for recrudescence, including sleep deprivation, physiological stress, central nervous system inflammation, and the administration of analgesia and sedation. The latter has been described elsewhere as a precipitant of recrudescence.⁶

This case highlights a presentation of the well-described but uncommonly observed phenomenon of transient recrudescence of a prior stroke deficit. In this case, recrudescence was preceded by subarachnoid hemorrhage and a cerebral angiogram. As is typical with recrudescence, the triggering of the event was likely multifactorial. First, the patient was admitted with a diagnosis of subarachnoid hemorrhage. He was sleep-deprived due to frequent neurologic assessments. Second, he received both benzodiazepines and opioids during the cerebral angiogram. Finally, the physiologic stress of intracranial hemorrhage with associated inflammation and pain contributed to the re-emergence of a previously silent lacunar infarction.

Several studies have attempted to explain the mechanism of recrudescence. The transient re-emergence of prior deficits has previously been described in patients receiving sedation, specifically with fentanyl and midazolam, the same regimen used for sedation and analgesia during this patient's cerebral angiogram.⁶ It has been proposed that opioid administration causes the functional suppression of re-organized compensatory cerebral networks that form after injury.² Infection and inflammation have been implicated as triggers in an experimental model. Laboratory rats underwent middle cerebral artery occlusion and then were given an immune stimulus. The animals experienced a reemergence of stroke-like symptoms when they were allowed to recover from the stroke and then later challenged with a different brain antigen.¹ Taken together, these studies support the notion that anamnestic recall may be triggered by a variety of clinical scenarios, from medications to inflammatory states.

The patient in this report presented with anamnestic recall of a variant stroke syndrome known as dysarthria-facial paresis (DFP) with clumsy hand. Considered to be a variant of dysarthria-clumsy hand syndrome, which is face-sparing, DFP results from lacunar infarction within the corona radiata and internal capsule.⁷ This injury preferentially involves the corticobulbar tract for language articulation, which is typically in the dominant hemisphere.⁸ This patient's clinical exam and radiographic findings seemed to be a prototypical presentation of dysarthria-facial-paresis (Figure 2), with additional involvement of the corticospinal fibers in the corona radiata producing upper extremity weakness and a mild pronator drift. As described in previous reports, the facial paresis was subtle, brief, and preceded the improvement in dysarthric speech.⁹ The fact that the patient was left-handed suggests that he may have been right-hemisphere-dominant. This may explain why the patient had no recollection of a prior stroke. The recovery process after a stroke is, in a way, an "amnestic process," in that the neurological deficit is gradually "forgotten" as the deficit resolves.

CONCLUSIONS

Recrudescence of prior stroke symptoms is an important stroke mimic and has not been previously reported after subarachnoid hemorrhage. In any given patient with stroke symptoms, recrudescence is a diagnosis of exclusion, and is supported by objective evidence of a previous

neurological injury that would be expected to produce the observed symptoms. The above criteria were met in this case. This description of recrudescence may be of interest to neurosurgeons, particularly those with neurovascular expertise in cerebral angiography and the treatment of subarachnoid hemorrhage.

ACKNOWLEDGEMENTS

We are grateful to Dr. Karen Albright for providing her expert opinion. Dr. Albright's involvement provided clarity and improved the quality of this manuscript significantly.

REFERENCES

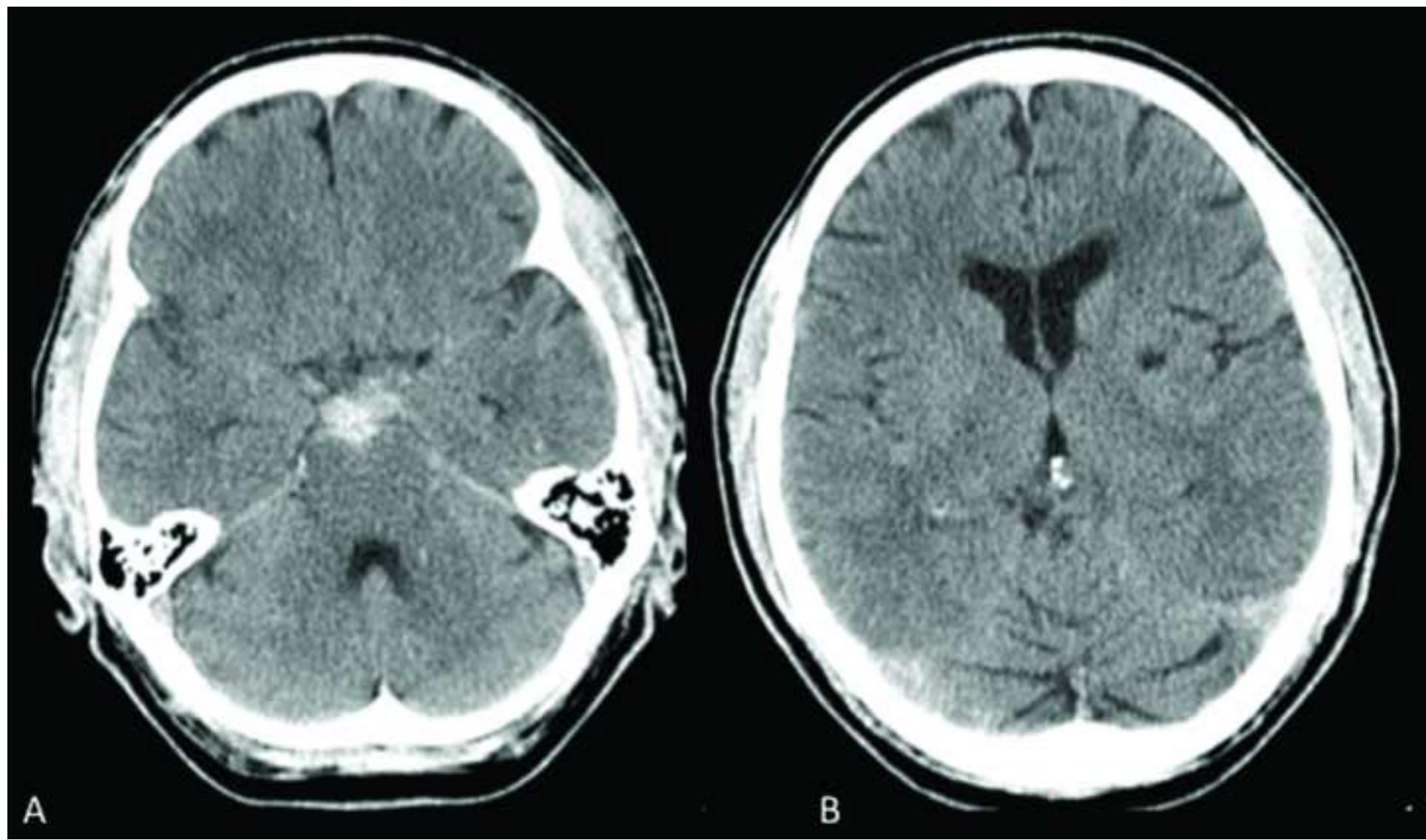
1. Zierath D, Thullberg M, Hadwin J, et al. CNS immune responses following experimental stroke. *Neurocrit Care*. 2010;12(2):274-284.
2. Bernstock JD, Budinich CS, Cohen LG, Awosika OO. Recrudescence of Focal Stroke Symptoms during Pain Management with Hydromorphone. *Front Neurol*. 2016;7:50.
3. Fernandes PM, Whiteley WN, Hart SR, Al-Shahi Salman R. Strokes: mimics and chameleons. *Pract Neurol*. 2013;13(1):21-28.
4. Hand PJ, Kwan J, Lindley RI, Dennis MS, Wardlaw JM. Distinguishing between stroke and mimic at the bedside: the brain attack study. *Stroke*. 2006;37(3):769-775.
5. Levy DE. How transient are transient ischemic attacks? *Neurology*. 1988;38(5):674-677.
6. Thal GD, Szabo MD, Lopez-Bresnahan M, Crosby G. Exacerbation or unmasking of focal neurologic deficits by sedatives. *Anesthesiology*. 1996;85(1):21-25; discussion 29A-30A.
7. Kim JS. Pure dysarthria, isolated facial paresis, or dysarthria-facial paresis syndrome. *Stroke*. 1994;25(10):1994-1998.
8. Urban PP, Rolke R, Wicht S, et al. Left-hemispheric dominance for articulation: a prospective study on acute ischaemic dysarthria at different localizations. *Brain*. 2006;129(Pt 3):767-777.
9. Edlow JA, Selim MH. Atypical presentations of acute cerebrovascular syndromes. *Lancet Neurol*. 2011;10(6):550-560.

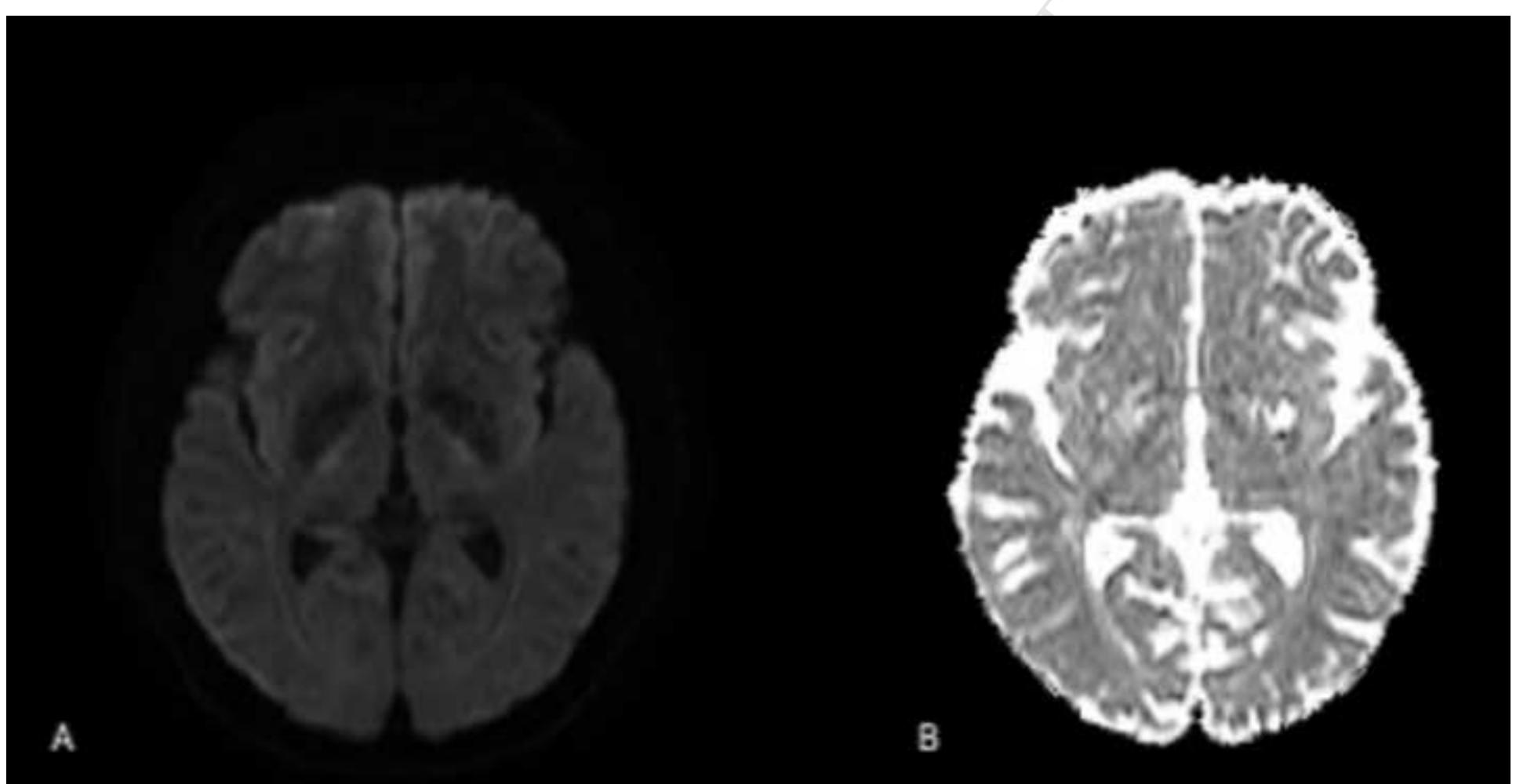
FIGURE LEGENDS

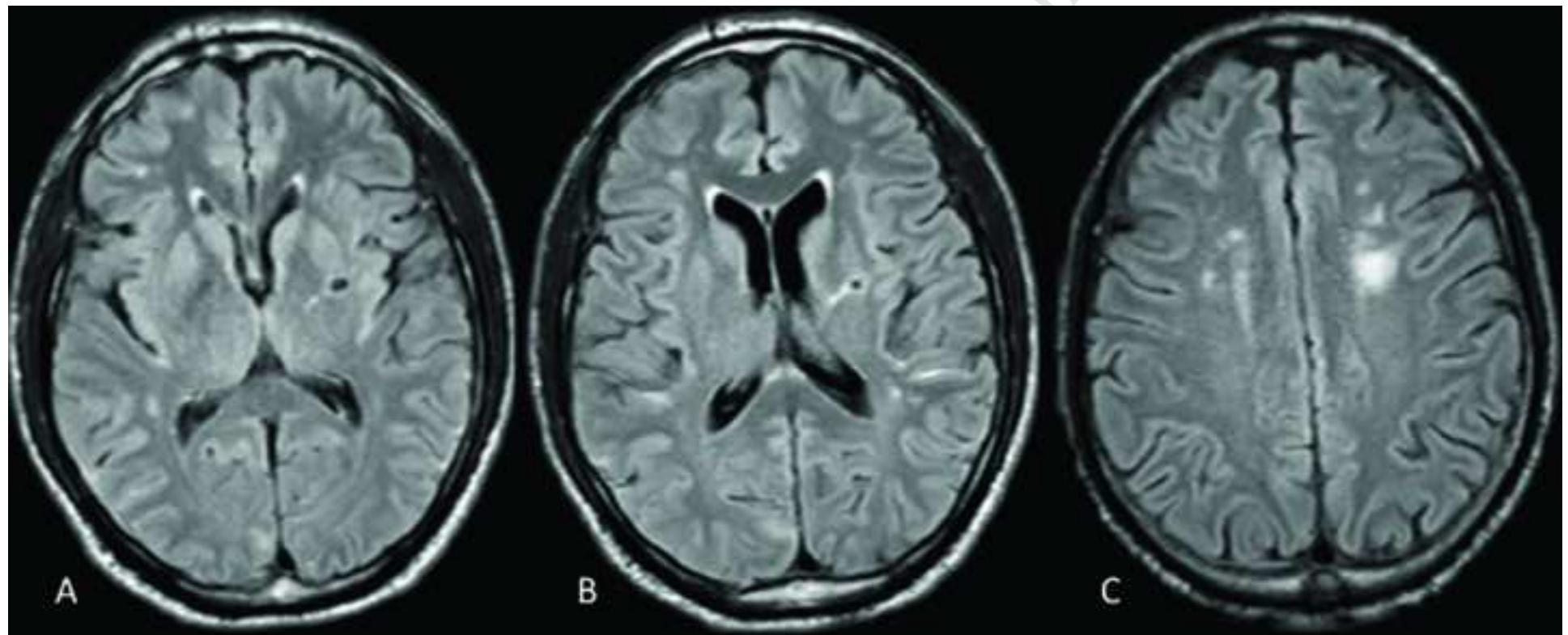
Figure 1. A. Noncontrast computed tomography scan demonstrating subarachnoid hemorrhage within the prepontine and interpeduncular cisterns. B. Previous left lacunar infarction involving the internal capsule and basal ganglia.

Figure 2. A. Diffusion weighted imaging shows no evidence of acute ischemic stroke. B. Apparent diffusion coefficient (ADC) demonstrates bright signal within the left basal ganglia and corona radiata correlating with a chronic lacunar infarction. No area of new ischemia is seen.

Figure 3. Serial FLAIR MR images demonstrating prior ischemic stroke and microangiopathic change involving the left posterior limb of the internal capsule (A), corona radiata (B), and centrum semiovale (C)







ABBREVIATIONS

ADC Apparent diffusion coefficient; CT computed tomography; DFP dysarthria-facial paresis; FLAIR fluid- attenuated inversion recovery; IV intravenous; MRI magnetic resonance imaging; TIA transient ischemic attack; DWI diffusion weighted imaging

HIGHLIGHTS

1. Recrudescence of an ischemic stroke can occur after subarachnoid hemorrhage.
2. Identify and address triggers promptly when recrudescence is suspected.
3. Knowledge of recrudescence as a stroke mimic will assist in patient counseling.
4. Recovery from stroke results in amnesia to the deficit once it resolves.