



SILENT STROKE - A CASE REPORT

Internal Medicine

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ABSTRACT

A 56-year-old male presented with subtle cognitive decline. Neuroimaging revealed a large infarct. Despite absence of overt symptoms, cognitive assessment indicated impairment. This case underscores the importance of recognizing silent strokes and their impact on cognitive function, warranting vigilance in clinical evaluation." **Case Report:** A 56-year-old male presented with 2 days duration of headache and giddiness on and off with no focal neurological deficits, MRI brain revealed acute infarction involving medial aspect of left temporal lobe, lateral aspect of Left occipital lobe and splenium of corpus callosum on left side. **Conclusion:** Silent strokes pose a diagnostic challenge due to their asymptomatic nature. This case emphasizes the importance of proactive management of underlying risk factors to prevent recurrent events and mitigate long-term cognitive impairment.

KEYWORDS

silent stroke, silent areas in brain

INTRODUCTION:

Silent strokes, also known as silent cerebral infarctions (SCIs), are ischemic events that occur in the brain without noticeable symptoms. These covert incidents can lead to long-term neurological deficits and contribute to cognitive decline. This case report examines a patient who experienced a silent stroke, focusing on the clinical presentation, diagnostic process, and management strategies.

Case Presentation:

A 56-year-old male who is a known case of Diabetes and hypertension since 4 years and 1 year respectively on regular medication presented to casualty with chief complaints of headache and giddiness of 2 day duration.

History of presenting illness: patient was apparently normal until 2 days when he suddenly developed headache along with giddiness back at work, that gradually progressed with pulsations in frontal area there was no photophobia, phonophobia and nausea, vomitings.

Giddiness was experienced on standing and while walking though patient denies any vertigo, clumpsiness patient therefore returned home @ 1pm and later went to a local clinic due to persistent headache where he received symptomatic treatment and returned home.

On next day later in the evening again he developed headache similar to what he had in the previous day it not relieved with NSAID's.

On day 2 he came to hospital with similar complaints around @ 5:00pm There was no history of trauma, loss of consciousness, seizure, fever No other systemic symptoms

Patient is an occasional alcohol consumer and non smoker, consumed alcohol 2 days back

On general examination: patient was conscious, irritable, oriented to time place and person

Patient was vitally stable with no focal neurological deficits or visual field defects

On Clinical Assessment:

Cardiovascular, respiratory and Per abdomen examination was within normal limits

Central nervous system: higher mental examination was normal , there were no motor or sensory deficits seen, cranial nerves intact, cerebellum examination was also normal

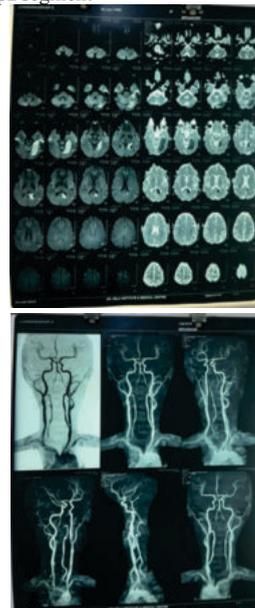
Cognitive Assessment: Mild impairment in memory was seen and on examination of visual fields right homonymous hemianopia was noted

Diagnostic Workup:

1. Imaging:

Magnetic Resonance Imaging (MRI) of the Brain: Revealed Acute infarct seen involving the medial aspect of left temporal lobe and lateral aspect of left occipital lobe and splenium of corpus callosum on left side

On MRA: Abrupt cut off noted in p2 segment of left posterior cerebral artery with visualized p3 segment and cortical branches , a small thrombus noted in p2 segment



2. Other Tests:

- Lipid Panel: Elevated cholesterol levels
- Hemoglobin A1c: Elevated (9.52%), indicating poor glycemic control
- Carotid Doppler Ultrasound: Detected mild atherosclerotic changes in the carotid arteries.

Diagnosis:

The patient was diagnosed with a silent stroke, likely due to underlying risk factors such as hypertension, hyperlipidemia, and poorly controlled diabetes. The absence of overt symptoms had delayed the recognition of the cerebrovascular event.

Management:

- Blood Pressure Control:**
 - Initiation of antihypertensive medication to maintain blood pressure within the recommended range.
- Glycemic Control:**
 - Intensification of diabetes management to achieve better blood glucose control.
- Lipid-lowering Therapy:**
 - Prescribing statins to manage hyperlipidemia and reduce the risk of future cardiovascular events.
- Lifestyle Modifications:**[2]
 - Dietary counseling and encouragement of regular exercise to promote overall cardiovascular health.
- Neurorehabilitation:**
 - Referral to a neurorehabilitation program for cognitive and functional training.

Follow-up:

The patient was scheduled for regular follow-up appointments to monitor blood pressure, glycemic control, and lipid levels. Repeat imaging was planned to assess the progression of the cerebral lesion. Rehabilitation efforts aimed at improving cognitive function were ongoing.

DISCUSSION:

The term "silent areas of the brain" can be interpreted in different ways, 1. **Silent Areas in Terms of Function:**

- The brain is divided into different regions, each responsible for specific functions such as motor control, language processing, sensory perception, etc. "Silent areas"[1] could refer to regions that don't typically produce overt signals or symptoms when they are active or when there's a disturbance.

- For example, the prefrontal cortex might be considered a "silent" area in the sense that dysfunction there may not manifest as obvious motor or sensory deficits but can still lead to complex cognitive and executive function issues.

2. **Silent Strokes and Silent Areas:**

- In the context of strokes, "silent strokes" occur when there is damage to the brain tissue without noticeable symptoms. These strokes can happen in regions of the brain responsible for cognition, and the term "silent" refers to the absence of traditional stroke symptoms like sudden weakness or speech difficulties.
- These silent strokes can occur in various brain regions, including those associated with memory, language, or executive functions.

3. **Silent Areas in Neuroimaging:**

- In neuroimaging studies, certain brain areas might appear "silent" if they do not show significant activation or if they are not easily identifiable based on the method being used (e.g., functional MRI or structural MRI).

4. **Silent Areas in Pathology:**

- In the context of neurological pathology, certain areas of the brain might be referred to as "silent" if they are not associated with typical symptoms even when damaged. This can be relevant in diseases like Alzheimer's, where certain areas of the brain can undergo degeneration without immediately causing noticeable symptoms.

It's crucial to note that the brain is highly interconnected, and even

regions traditionally associated with specific functions can have complex interactions. In different contexts, the term "silent areas" might refer to different aspects of brain function, structure, or pathology.

In terms of brain function, the concept of "silent areas" generally refers to regions of the brain that do not exhibit overt or easily observable activities or symptoms under normal circumstances. These areas may not be associated with specific sensory or motor functions, and their activities may not lead to noticeable behaviors. It's important to note that the term "silent" in this context doesn't imply that these areas are inactive or unimportant; rather, their functions may be less readily apparent or involve more subtle and complex processes. Here are a few examples:

1. **Prefrontal Cortex:**

- The prefrontal cortex, located at the front of the brain, is often considered a "silent" area in terms of outwardly observable functions. It is involved in executive functions such as decision-making, planning, and personality, and its dysfunction may not manifest as obvious motor or sensory deficits.

2. **Association Areas:**[4,5]

- Association areas, which integrate information from different sensory modalities and higher-order cognitive functions, are crucial for complex processes like memory and language. While damage to these areas can lead to significant cognitive impairments, their role may not be immediately apparent in everyday behaviors.

3. **Supplementary Motor Area (SMA):**

- The supplementary motor area, involved in planning and coordinating complex movements, might not produce observable symptoms when damaged, especially if other motor areas compensate for its functions.

Understanding these "silent areas" is crucial for a comprehensive understanding of brain function, as they contribute to higher-order cognitive processes and play a role in integrating information across different brain regions. Advances in neuroimaging[6,7] and neuroscience continue to shed light on the intricate functions of various brain regions, even those that may not have traditionally been associated with easily observable behaviors.

CONCLUSION:

Silent strokes pose a diagnostic challenge due to their asymptomatic nature. This case emphasizes the importance of proactive management of underlying risk factors to prevent recurrent events and mitigate long-term cognitive impairment. Healthcare providers should be vigilant in identifying and addressing silent strokes, especially in individuals with predisposing conditions such as hypertension, diabetes, and hyperlipidemia.

REFERENCES:

1. Vermeer SE, Longstreth WT Jr, Koudstaal PJ. Silent brain infarcts: a systematic review. *Lancet Neurol.* 2007;6(7):611-619. doi:10.1016/S1474-4422(07)70170-9
2. Smith EE, Saposnik G, Biessels GJ, et al. Prevention of stroke in patients with silent cerebrovascular disease: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2017;48(2):e44-e71. doi:10.1161/STR.000000000000116
3. Wardlaw JM, Smith EE, Biessels GJ, et al. Neuroimaging standards for research into small vessel disease and its contribution to ageing and neurodegeneration. *Lancet Neurol.* 2013;12(8):822-838. doi:10.1016/S1474-4422(13)70124-8
4. DeCarli C, Miller BL, Swan GE, et al. Predictors of brain morphology for the men of the NHLBI twin study. *Stroke.* 1999;30(3):529-536. doi:10.1161/01.STR.30.3.529
5. Kuller LH, Longstreth WT Jr, Arnold AM, et al. White matter hyperintensity on cranial magnetic resonance imaging: a predictor of stroke. *Stroke.* 2004;35(8):1821-1825. doi:10.1161/01.STR.0000132193.35955.95
6. Vermeer SE, Prins ND, den Heijer T, et al. Silent brain infarcts and the risk of dementia and cognitive decline. *N Engl J Med.* 2003;348(13):1215-1222. doi:10.1056/NEJMoa022066
7. Fisher CM. Lacunes: Small, Deep Cerebral Infarcts. *Neurology.* 1965;15(8):774-784. doi:10.1212/wnl.15.8.774