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A Comparative Analysis of Mid-Brain Lesions and Basal Ganglia in Subcortical Stroke-Related Acute Psychosis.

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Abstract

An abrupt onset of psychosis in an older or elderly individual with no prior history of psychiatric disorders should trigger a comprehensive evaluation for neurological causes of psychiatric symptoms. This study examines and compares the clinical features of new-onset psychotic symptoms in two patients, one with an acute basal ganglia hemorrhagic stroke and the other with an acute mid-brain ischemic stroke. Delusions and hallucinations resulting from basal ganglia lesions are believed to arise from frontal lobe dysfunction, leading to a disruption in reality checking pathways in the brain. On the other hand, visual hallucinations stemming from mid-brain lesions are thought to occur due to the dysregulation of inhibitory control in the ponto-geniculate-occipital system. Psychotic symptoms following a stroke exhibit diverse clinical characteristics that are influenced by the stroke's location in the brain. The use of antipsychotic medications may offer relief from symptoms.

Keywords:

Comprehensive evaluation Neurological causes Psychiatric symptoms New-onset psychotic symptoms Basal ganglia hemorrhagic stroke Acute mid-brain ischemic stroke Delusion Hallucinations Frontal lobe dysfunction Reality checking pathways Visual hallucinations Inhibitory control Ponto-geniculate-occipital system Clinical characteristics Stroke location Antipsychotic medications Elderly psychosis Psychiatric disorders Stroke symptoms Relief from symptoms

Introduction: Acute psychosis following subcortical stroke represents a multifaceted clinical entity that poses significant challenges in both diagnosis and management. While psychosis associated with stroke has been recognized for decades, the specific impact of lesion location within subcortical structures, such as the mid-brain and basal

ganglia, remains an area of ongoing investigation. Understanding the nuances of psychosis in relation to these distinct neuroanatomical regions is essential for tailoring effective therapeutic strategies and optimizing patient outcomes.

Subcortical strokes, affecting the deep brain structures beneath the cerebral cortex, can manifest with a spectrum of neuropsychiatric symptoms, including cognitive impairment, mood disturbances, and psychosis. Among these manifestations, acute psychosis represents a particularly complex and poorly understood phenomenon, characterized by hallucinations, delusions, and thought disturbances. The interplay between vascular insults to subcortical regions and subsequent psychiatric manifestations underscores the intricate relationship between brain anatomy and functional neurobiology.

The mid-brain, comprising the mesencephalon, and the basal ganglia, including structures such as the striatum, globus pallidus, and substantia nigra, serve critical roles in motor control, cognition, and emotion regulation. Lesions within these subcortical areas have been implicated in various neuropsychiatric disorders, including schizophrenia, mood disorders, and psychosis following stroke. However, the specific contributions of mid-brain versus basal ganglia lesions to the development and clinical expression of acute psychosis remain a subject of debate and inquiry.

This comparative analysis seeks to elucidate the distinct clinical profiles and neuroimaging correlates of acute psychosis associated with mid-brain lesions versus basal ganglia involvement following subcortical stroke. By examining differences in symptomatology, lesion localization, and treatment responses between these two groups, we aim to advance our understanding of the pathophysiological mechanisms underpinning post-stroke psychosis. Such insights hold the potential to inform personalized approaches to patient care and guide future research endeavors aimed at unraveling the complexities of neuropsychiatric sequelae following cerebrovascular events.

- **Acute Psychosis**: Sudden onset of hallucinations and delusions in older or elderly individuals without a known history of previous psychiatric disorders.
- **Neurologic Causes**: When encountering new-onset psychotic symptoms, it’s crucial to consider underlying neurologic causes.
- **Brain Lesions**: Previous reports describe acute psychosis resulting from various brain lesions, including strokes affecting different brain areas such as the prefrontal and occipital cortices, as well as subcortical locations like the basal ganglia, thalamus, mid-brain, and brainstem.

Comparison of Cases:

Patient 1 (Basal Ganglia Hemorrhagic Stroke):

Clinical Features: Developed delusions and hallucinations.

Theorized Mechanism: Dysfunction of reality-checking pathways due to frontal lobe impairment caused by basal ganglia lesions.

Certainly! Let's delve into the fascinating world of brain function and how basal ganglia lesions can impact reality-checking pathways.

1. **Basal Ganglia**:
    - The basal ganglia are a group of interconnected nuclei located deep within the cerebral hemispheres.
    - They play a crucial role in motor control, cognition, and emotion.
    - Key components include the **caudate nucleus**, **putamen**, **globus pallidus**, and **subthalamic nucleus**.

2. **Reality-Checking Pathways**:
    - These pathways involve the frontal lobe and are responsible for assessing the accuracy of sensory information and distinguishing between reality and imagination.
    - When you see something or hear something, your brain evaluates whether it's real or a product of your mind.
    - The frontal lobe, especially the **prefrontal cortex**, is involved in this process.

3. **Impairment Due to Basal Ganglia Lesions**:
    - When there's damage to the basal ganglia, it can disrupt the normal functioning of the frontal lobe.
    - Specifically, lesions in the basal ganglia can affect the prefrontal cortex's ability to perform reality checks.

    - This impairment may lead to altered perceptions, hallucinations, and delusions.

Mechanism:

- The exact mechanism isn't fully understood, but several theories exist:

- **Dopamine Dysregulation**: The basal ganglia are involved in dopamine regulation. Dysregulation of dopamine levels due to lesions could impact frontal lobe function.

- **Circuit Disruption**: The basal ganglia and prefrontal cortex communicate via neural circuits. Lesions disrupt these circuits, affecting information flow.
- **Frontal-Subcortical Loops**: These loops involve the basal ganglia and prefrontal cortex. Lesions may disrupt these loops, leading to cognitive and perceptual changes.

Clinical Implications:

- Patients with basal ganglia lesions may experience:
- **Psychosis**: Hallucinations (seeing or hearing things that aren't there) and delusions (false beliefs).
- **Reality Distortion**: Their perception of reality may be altered.
- **Cognitive Changes**: Executive function deficits, attention problems, and memory issues.

Patient 2 (Mid-Brain Ischemic Stroke):

Clinical Features: Presented with visual hallucinations.

Theorized Mechanism: Dysregulation of inhibitory control in the ponto-geniculate-occipital system.

Location Matters: Psychotic symptoms vary based on the lesion's location within the brain.

Certainly! Let's explore the fascinating concept of inhibitory control within the ponto-geniculate-occipital system.

Ponto-Geniculate-Occipital System:

- This system involves interconnected brain regions: the **pontine nuclei**, the **geniculate nuclei**, and the **occipital cortex**.
- It plays a crucial role in visual processing, particularly transmitting visual information from the eyes to the brain.

Inhibitory Control:

- Inhibitory control refers to the ability of neural circuits to suppress or modulate the activity of other neurons.

- Within the ponto-geniculate-occipital system, inhibitory neurons play a critical role in shaping visual responses.

- These inhibitory mechanisms help filter out irrelevant information, enhance signal-to-noise ratios, and maintain visual stability.

Mechanisms:

- **GABAergic Interneurons**: Gamma-aminobutyric acid (GABA)-releasing interneurons are abundant in this system.

- They provide inhibitory input to both the geniculate nuclei and the occipital cortex.

- By dampening excitatory signals, they prevent excessive neural firing.

- **Lateral Inhibition**: Neurons in the geniculate nuclei and occipital cortex engage in lateral inhibition.

- When one neuron is active, it inhibits neighboring neurons, sharpening the contrast between visual stimuli.

- **Feedback Loops**: Reciprocal connections between the occipital cortex and the geniculate nuclei allow for feedback modulation.

- This feedback helps refine visual representations and adapt to changing environmental conditions.

Clinical Implications:

- Dysregulation of inhibitory control within this system can lead to visual disturbances:

- **Visual Hallucinations**: When inhibitory mechanisms fail, aberrant neural firing may create false perceptions.

- **Migraine Auras**: Altered inhibitory processes contribute to the visual auras experienced by some migraine sufferers.

- **Epileptic Seizures**: Imbalance between excitation and inhibition can trigger seizures.

- **Treatment**:

- **Antipsychotic Medications**: Symptomatic relief can be achieved through antipsychotic medications.

Types of Antipsychotics:

Typical (First-Generation) Antipsychotics:

Examples: Haloperidol, chlorpromazine, fluphenazine.
Mechanism: Block dopamine receptors (especially D2 receptors) in the brain.
Effectiveness: Effective in treating positive symptoms (hallucinations, delusions) but may have more side effects.

Atypical (Second-Generation) Antipsychotics:

- Examples: Risperidone, olanzapine, quetiapine, aripiprazole.
- Mechanism: Affect multiple neurotransmitter systems (dopamine, serotonin, glutamate).
- Effectiveness: Address both positive and negative symptoms with potentially fewer side effects.

Symptomatic Relief:

Positive Symptoms:
Hallucinations: Antipsychotics can reduce the intensity and frequency of hallucinations.
Delusions: They help manage false beliefs and paranoid thoughts.
Negative Symptoms:Social withdrawal, reduced motivation, and flattened affect may also improve with treatment.
Cognitive Symptoms:
Some atypical antipsychotics may enhance cognitive function.

Side Effects:
Extrapyramidal Symptoms (EPS):
Common with typical antipsychotics.
Includes parkinsonism (rigidity, tremors), dystonia (muscle spasms), and akathisia (restlessness).
Metabolic Side Effects:
Weight gain, dyslipidemia, and increased risk of diabetes.

Atypical Advantages:

Lower risk of EPS and potentially better tolerability.

Still, they may cause weight gain and metabolic changes.

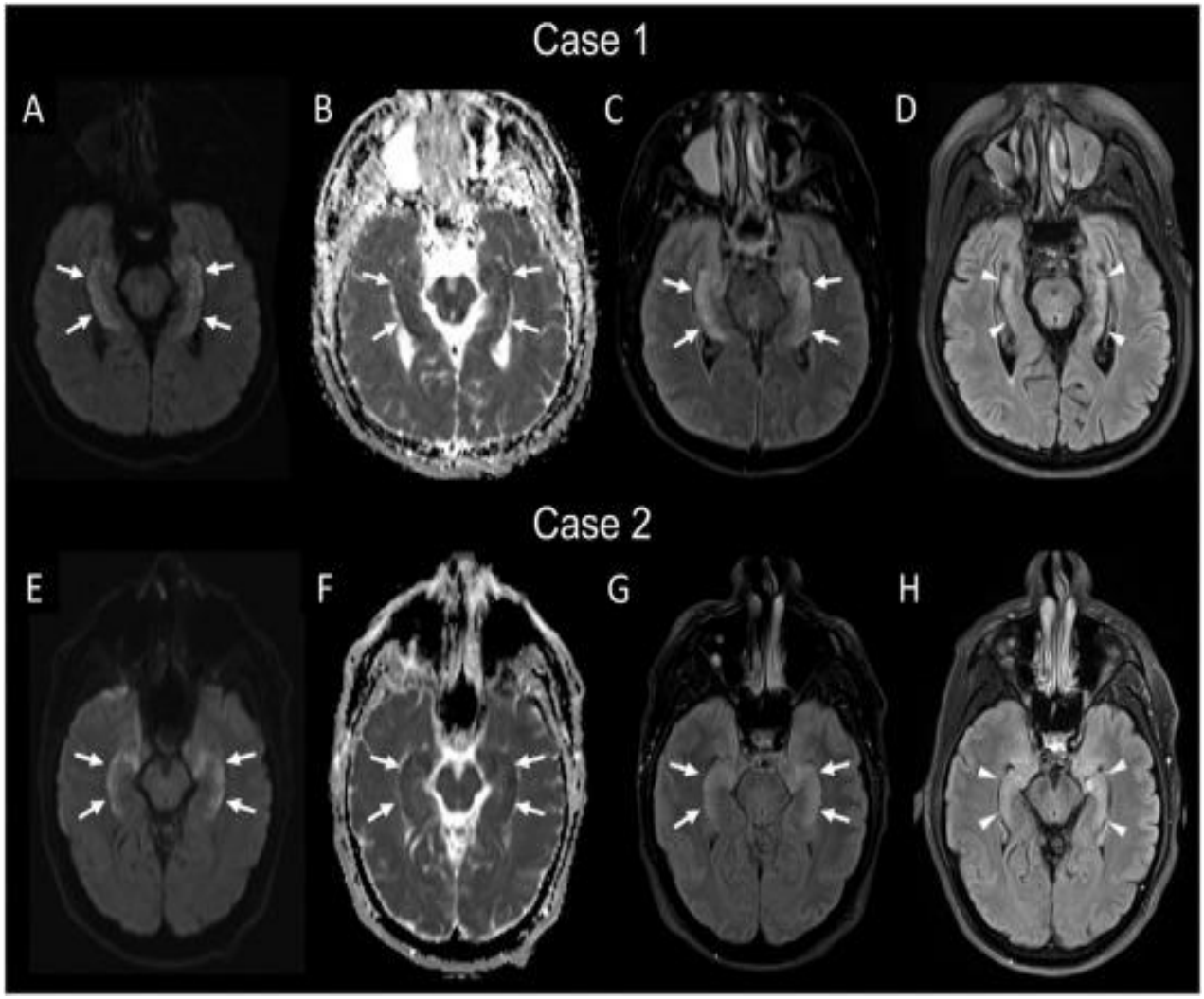
Individualized Treatment:

Choosing the right antipsychotic depends on the patient's specific symptoms, medical history, and potential side effects.

Regular follow-up with a psychiatrist is essential to monitor efficacy and address any adverse effects.

Case Presentation 1: Left Basal Ganglia Hemorrhagic Stroke A fifty-nine-year-old right-handed male with no known past medical history was brought in by emergency medical services to the emergency room after acute onset at home of right sided weakness and visual and auditory hallucinations that started approximately eight hours prior to arrival. The patient was alert and oriented to self, location, and date. Examination revealed a right lower facial droop and right hemiparesis associated with pronator drift. Sensation for light touch and pinprick was normal in the upper and lower extremities bilaterally. Deep tendon reflexes were brisk on the right side compared to the left side. The patient reported content specific delusions that the right side of his body was "rotting," that he had a tooth that was decaying int he right side of his mouth and that the nurses had injured the right side of his body when transporting him. Despite repeated reassurance by his treating physicians that none of these were true, he continued to display these fixed false beliefs. His visual hallucinations consisted of seeing colors and lights and hearing voices telling him that the right side of this body was "dead. "Hewes treated with low dose risperidone and his hallucinations steadily decreased in frequency over the course of the next two weeks. Initial laboratory assessment showed a normal serum chemistry panel, normal complete blood cell count, normal urinalysis, and negative urine toxicology screen for illicit substances. Serum HIV testing was negative and the thyroid stimulating hormone level was within normal limits. A 1.5 Tesla magnetic resonance imaging scan of the brain without contrast showed a 3.8cm (about 1.5 in) by 2.2cm (about 0.87 in) intraparenchymal hematoma located in the left basal ganglia with adjacent edema likely affecting the corona radiate and possibly extending to the optic radiations. There was no midline shift. Gray-white differentiation was preserved, and the ventricles, sulci, and cisterns were normal. Additionally, no extra-axial fluid collections or significant atrophy was present, and there was no evidence of acute or subacute ischemic change. Small periventricular hyperintensities were present in the white matter on a fluid attenuated inversion recovery (FLAIR) sequence, consistent with chronic small vessel vascular disease.

Case Presentation 2: Peduncular Hallucinosis due to Ischemic Stroke A fifty-two-year-old right-handed female with past medical history significant for type two diabetes, hypertension, and hyperlipidemia was brought to the emergency room by a friend with new onset of dizziness and unsteadiness when walking that had developed suddenly approximately one month earlier. She also reported a new onset of double vision and an occipital headache both of which had developed acutely three days prior to presentation and visual and auditory hallucinations that developed one day prior to presentation. On presentation to the emergency room, the patient was obtunded but able to be aroused and she could answer questions and follow simple commands when aro used. The patient was noted to be grabbing at unseen objects by the nursing staff. The patient was alert and oriented to self, location, and date. Cranial nerve examination showed fixed dilated pupils bilaterally that were not reactive to light, bilateral exotropia of the eyes at rest, and complete paresis of ocular movements. The corneal and gag reflexes were present. The patient had purposeful movement in all extremities but was noted to have more spontaneous movement of the left side limbs than the right side. Sensation for light touch and pinprick were normal in the upper and lower extremities bilaterally. Deep tendon reflexes were also normal and symmetrical throughout. The patient's visual hallucinations were formed and consisted of seeing a deceased uncle. The patient's auditory hallucinations consisted of intermittently hearing the deceased uncle's voice saying indistinct words and sentences. The patient demonstrated preserved insight: she was aware that the hallucinations were not real and that her uncle was deceased and therefore could not be present and talk to her. The patient received a single dose of haloperidol in the emergency room due to agitation, which temporarily resolved the auditory and visual hallucinations for the remainder of that night. The patient hallucinations were initially worse at night but then gradually decreased on scheduled haloperidol. The initial laboratory assessment for this patient showed a normal serum chemistry panel, normal complete blood cell count, normal urinalysis, and negative urine toxicology screen for illicit substances. A 1.5 Tesla magnetic resonance imaging of the brain without contrast showed areas of restricted diffusion on DWI sequences located bilaterally in the thalami, left cerebral peduncle, the mid-brain, and right external capsule consistent with acute infarcts. Additional small, scattered white matter hyperintensities were present in the periventricular regions bilaterally on the FLAIR sequence, consistent with small vessel vascular disease. The ventricles, cisterns, and sulci were normal in appearance and there was no significant atrophy. There were no intra-axial or extra-axial fluid collections, no mass, and no midline shift.



Discussion:

The pathophysiology and clinical presentation of peduncular hallucinosis, a neurological condition characterized by vivid visual hallucinations, often with a dreamlike quality. The exact cause of peduncular hallucinosis remains unclear, but it is commonly associated with lesions in the brainstem, cerebral peduncles, and other midbrain structures. The document also discusses two case reports of patients who developed psychotic symptoms, including delusions and hallucinations, following strokes or lesions in the basal ganglia. These cases suggest that disruption of frontal-subcortical circuits and impairment of reality monitoring can contribute to the development of psychotic symptoms in the context of neurological conditions.

What are the common causes of peduncular hallucinosis besides stroke?

The common causes of peduncular hallucinosis, besides stroke, include tumors (such as meningiomas, primary cerebellar tumors, and metastases), subarachnoid hemorrhage, and iatrogenic injury from surgery. These causes have been reported to result in peduncular hallucinosis, indicating that it can arise from disruptions in various brain structures and mechanisms beyond stroke-related lesions.

How do the auditory hallucinations described in the case report compare to those reported in a previous study by Benke?

The auditory hallucinations described in the case report include indistinct voices from deceased relatives. In contrast, the previous study by Benke reported auditory hallucinations of both distinct and indistinct voices, sounds made by animals, and in one case, a train. While both sources describe auditory hallucinations, the specific content and nature of the auditory experiences differ between the case report and Benke's study.

What is the proposed mechanism by which unilateral basal ganglia lesions can produce psychotic symptoms?

The proposed mechanism by which unilateral basal ganglia lesions can produce psychotic symptoms is thought to involve the disruption of normal self-corrective functions that prevent the development of odd beliefs. Additionally, the impairment of the sense of familiarity, which may contribute to the development of paranoid ideation, is also considered to be a factor. The disruption of normal frontal-subcortical circuits, which are essential for normal behavior, is theorized to contribute to the development of neuropsychiatric symptoms, including psychotic symptoms. Specifically, unilateral basal ganglia lesions are believed to affect prefrontal functioning, leading to alterations that are theorized to be necessary for the generation of delusions and other psychotic symptoms.

What is the relationship between prefrontal dysfunction and the development of psychotic symptoms?

The proposed relationship between prefrontal dysfunction and the development of psychotic symptoms is that disruption of normal frontal lobe functioning can contribute to the generation of delusions and other psychotic symptoms.The prefrontal cortex is thought to play a crucial role in reality monitoring and checking systems that help prevent the development of odd beliefs. When these frontal lobe functions are impaired, it can lead to a breakdown in the ability to accurately assess and correct one's own thoughts and perceptions.Specifically, unilateral basal ganglia lesions have been shown to cause prefrontal lobe hypometabolism, suggesting decreased or altered frontal lobe functioning as a direct result of the basal ganglia damage. This disruption of normal frontal-subcortical circuits is theorized to be a key mechanism by which basal ganglia lesions can facilitate the development of psychotic symptoms.Additionally, impairment of the sense of familiarity, which may arise from basal ganglia dysfunction, is also proposed to contribute to the generation of paranoid ideation and delusional beliefs. The combination of disrupted reality monitoring and altered familiarity perception are thought to be important factors in the pathogenesis of psychosis in the context of basal ganglia lesions.In summary, the prefrontal cortex plays a critical role in reality testing and self-corrective functions, and dysfunction in this region, often resulting from disruption of frontal-subcortical circuits, is considered a key mechanism underlying the development of psychotic symptoms in the setting of basal ganglia lesions.

What additional research techniques could be useful for further understanding the pathways involved in acquired psychotic phenomena?

According to the context provided, the authors suggest that additional research utilizing advanced neuroimaging techniques such as diffusion tensor imaging could be useful for further identifying and defining the specific brain pathways and connections involved in the generation of acquired psychotic phenomena. The passage notes that the exact pathophysiology of conditions like peduncular hallucinosis remains unclear, and that further research employing techniques like diffusion tensor imaging to visually assess disruptions in brain pathways and connectivity

could help elucidate the underlying neural mechanisms. The context indicates that while lesions in various brain regions like the basal ganglia, brainstem, and prefrontal cortex have been associated with the development of psychotic symptoms, the specific pathways and connections mediating these acquired psychotic phenomena are not fully characterized. Advanced neuroimaging approaches that can probe white matter integrity and connectivity could provide valuable insights to further define the neural circuitry involved. So, in summary, the passage suggests that diffusion tensor imaging and other advanced neuroimaging methods could be useful complementary research techniques to help better understand the specific brain pathways and network disruptions that contribute to the generation of psychotic symptoms in the context of acquired brain lesions or dysfunction.

Based on the provided context, here is a summary of the key points regarding acute psychosis linked to subcortical stroke, comparing mid-brain lesions and basal ganglia lesions:The context discusses two case reports that illustrate how lesions in different subcortical regions can lead to the development of psychotic symptoms, including delusions and hallucinations, following a stroke. Basal Ganglia Lesions: - Lesions in the basal ganglia can disrupt normal self-corrective functions and the sense of familiarity, leading to the development of odd beliefs and paranoid ideation.- A previous case report showed that a unilateral basal ganglia (caudate) lesion was sufficient to produce content-specific delusions, likely due to bilateral alterations in prefrontal functioning.- The case presented in the current report had a more extensive left basal ganglia lesion, which likely disrupted multiple frontal-subcortical circuits, contributing to the patient's delusions and hallucinations-Brain/Peduncular Lesions:- Peduncular hallucinosis is thought to result from disruptions along the pathway from the ascending reticular activating system to the intralaminar thalamic nuclei, leading to a "release phenomenon" and visual hallucinations.- Peduncular hallucinosis is characterized by vivid, formed visual hallucinations that can have a dream-like quality, with preserved insight and a high percentage of hypnagogic hallucinations.- The case presented in the current report displayed hallucinations typical of mid-brain and thalamic injuries, with formed hallucinations and a hypnagogic component, but without the bizarre or Lilliputian qualities sometimes seen in peduncular hallucinosis.

References:

1. Busner J, Targum SD. The clinical global impression scale. applying a research tool in clinical practice. the clinical global impression scale. Psychiatry (Edgmont) 2007;4(7):28–37. [Europe PMC free article] [Abstract] [Google Scholar]
2. WHO MONICA Project, Principal Investigators. The World Health Organization MONICA Project (Monitoring trends and determinants in cardiovascular disease): a major international collaboration. J Clin Epidemiol. 1988; 41:104–14. [Abstract] [Google Scholar]
3. Strong K, Mathers C, Bonita R. Preventing stroke: saving lives around the world. Lancet Neurol. 2007; 6:182–87. [Abstract] [Google Scholar]
4. Dalal P, Bhattacharjee M, Vairale J, Bhat P. UN millennium development goals: can we halt the stroke epidemic in India? Ann Indian Acad Neurol. 2007; 10:130–36. [Google Scholar]

5. Fisher M, Norrving B. The International Agenda for Stroke. 1st Global Conference on healthy lifestyles and non-communicable diseases control. Moscow, Apr 28-29, 2011.
6. Steinwachs DM, Collins-Nakai RL, Cohn LH, Garson A Jr, Wolk MJ. The future of cardiology: utilization and costs of care. J Am Coll Cardiel. 2000;35(5Suppl B):91B–98B. [Abstract] [Google Scholar]
7. World Health Organization. The global burden of disease: 2004 update. Available at: http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html. Accessed January 15, 2015.
8. Radensky PW, Archer JW, Dournaux SF, O'Brien CF. The estimated cost of managing focal spasticity: a physician practice patterns survey. Neuro Rehabil Neural Repair. 2001; 15:57–68. [Abstract] [Google Scholar]
9. Feigin VL, Lawes CM, Bennett DA, Barker-Collo SL, Parag V. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. Lancet Neurol. 2009;8(4):355–69. [Abstract] [Google Scholar]
10. de Hann RJ, Limburg M, van der Meulen JHP, Jacobs HM, Aaronson NK. Quality of life after stroke: impact of stroke type and lesion location. Stroke. 1995; 26:402–08. [Abstract] [Google Scholar]
11. King RB. Quality of life after stroke. Stroke. 1996; 27:1467–72. [Abstract] [Google Scholar]
12. Hop JW, Rinkel JE, Algra A, van Gijn J. Quality of life in patients and partners after aneurysmal subarachnoid hemorrhage. Stroke. 1998; 29:798–804. [Abstract] [Google Scholar]
13. Robinson RG. Neuropsychiatric consequences of stroke. Annu Rev Med. 1997; 48:217–29. [Abstract] [Google Scholar]
14. Robinson RG, Jorge R. Kaplan & Sadock's Comprehensive textbook Of Psychiatry. 9th ed. Philadelphia: Wolters Kluwer; 2009. Neuropsychiatric aspects of cerebrovascular disease. In: Sadock BJ, Sadock VA, Ruiz P editors; p. 426. [Google Scholar]
15. Schubert DS, Taylor C, Lee S. Physical consequences of depression in the stroke patient. Gen Hosp Psychiatry. 1992; 14:69–76. [Abstract] [Google Scholar]
16. Parikh RM, Robinson RG, Lipsey JR. The impact of post stroke depression on recovery in activities of daily living over a 2-year follow-up. Arch Neurol. 1990; 47:785–89. [Abstract] [Google Scholar]
17. Bielaski M, Bondurant H. Psychosis following a stroke to the cerebellum and midbrain: a case report. Cerebellum Ataxias. 2015; 2:17. [Europe PMC free article] [Abstract] [Google Scholar]
18. Abdulla KH, Saini SM, Sharip S, Rahman AH. Psychosis post corona radiates and lentiform nucleus infartion. BMJ Case Rep. 2015;2015: pii. bcr2014208954. [Europe PMC free article] [Abstract] [Google Scholar]
19. Wong A, Lau AY, Yang J, Wang Z, Liu W, Lam BY, et al. Neuropsychiatric symptom clusters in stroke and transient ischemic attack by cognitive status and stroke subtype: frequency and relationships with vascular lesions, brain atrophy and amyloid. PLoS One. 2016;11(9): e0162846. [Europe PMC free article] [Abstract] [Google Scholar]