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CASE REPORT **OPEN ACCESS**

FAHR DISEASE IN ELDERLY PATIENTS WITH BRONCHOPNEUMONIA AND HISTORY OF ISCHEMIC STROKE: A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Background: Fahr's Disease (Fahr's Syndrome) is a rare condition characterized by idiopathic bilateral basal ganglia calcifications, typically occurring in the lateral parts of the globus pallidus, dentate nuclei, and caudate nuclei. Diagnosis is based on clinical features of neuropsychiatric and somatic symptoms in conjunction with radiological findings. We report an unusual case of Fahr's disease in a 70-year-old woman with bronchopneumonia and a history of chronic ischemic stroke identified through a CT-SCAN.
Case: The patient was a 70-year-old female, admitted to our emergency department with bronchopneumonia, who presented with declining consciousness and focal seizures for 3 hours before entering the hospital. Her brain's CT scan revealed calcification in the bilateral lentiform nuclei, bilateral cerebellar dentate nuclei, and left corona radiata, consistent with Fahr's disease. The patient was also diagnosed with bronchopneumonia and chronic ischemic stroke.
Discussion: Fahr's Disease is linked to abnormal calcium metabolism, leading to calcifications in the basal ganglia. While chronic ischemic stroke may not directly cause Fahr's Disease, it increases the risk of cerebrovascular events in affected individuals. The correlation between infection and Fahr's Disease is poorly understood, although it may exacerbate neurological symptoms.
Conclusion: Basal ganglia calcification in Fahr's disease may be associated with an increased risk of stroke and bronchopneumonia, which can worsen the patient's condition. Further research is needed to understand the relationship between Fahr's disease, ischemic stroke, and infections.

Keywords: basal ganglia calcification, bronchopneumonia, Fahr's disease, ischemic stroke

Introduction

Basal ganglia calcification is also known as Fahr's disease or Fahr's syndrome. Fahr's disease is a sporadic neurological disorder caused by a rare inherited condition with a prevalence of <1/1,000,000.1-2. In diagnosing Fahr's Disease, it is essential to differentiate between Fahr's Disease and calcification of Fahr's Syndrome, such as hypoparathyroidism or metabolic disorders.[3] Calcification usually occurs in the lateral part of the globus pallidus, dentate nucleus, and caudate nucleus.[4-5]

In most cases, Fahr's disease is inherited in an autosomal dominant pattern and shows genetic variability.[6] Fahr's disease can be caused by brain infection and metabolic disorders, including hypoparathyroidism, pseudohypoparathyroidism, hypothyroidism, toxins such as lead and hypervitaminosis D. Brain infections can be caused by toxoplasmosis, rubella, cytomegalovirus, neurocysticercosis, HIV, and tuberculosis. Other rare genetic disorders, such as neurofibromatosis, tuberous sclerosis, Cockayne syndrome, and Wilson's disease, can also precipitate Fahr's disease.[1]

Fahr's disease has complex clinical symptoms, which can include cognitive impairment, movement disorders, and psychiatric disorders.[5-7] Symptoms can manifest as cognitive decline, dysarthria, dysphagia, and extrapyramidal signs such as rigidity and tremors.[8] Neuropsychiatric symptoms accompanied by movement disorders occur in 55% of all manifestations of this disease.[6] The variability of these symptoms is caused by the anatomical division of the basal ganglia into dorsal and ventral systems. The dorsal striatum plays a crucial role in motor and cognitive functions, whereas the ventral striatum is involved in motivational functions.[9]

Diagnosing Fahr's disease requires an imaging device such as a CT-SCAN, MRI, or plain cranial X-ray. Other tests that can be used are blood and urine tests for hematology and biochemistry indices.[10-11] This disease still has no cure, but management and treatment strategies mainly focus on reducing symptoms, such as prophylaxis for seizures and treatment of causative factors. However, there is some evidence to suggest that early diagnosis and treatment can reduce the calcification process for full recovery of mental function.[12]

Fahr's disease in elderly patients has several differences and is quite challenging because of its similarity with other neurodegenerative conditions, such as Alzheimer's disease or Parkinson's disease.[13] Notably, reports of Fahr's disease complicated by pneumonia are rare. One documented case involves a patient with Fahr's disease presenting with aspiration pneumonia and sepsis, exacerbated by COVID-19 infection.[14] To our knowledge, no published case has reported the co-occurrence of Fahr's disease, bronchopneumonia, and ischemic stroke. In this manuscript, we report on a 70-year-old woman with Fahr's disease, bronchopneumonia, and a history of ischemic stroke identified by CT scan.[8,14] This case report aims to explore the relationship between Fahr's disease, ischemic stroke, and bronchopneumonia, and to highlight the importance of early detection and appropriate management in preventing further complications in patients with this disease.

Case Report

A written informed consent for publication was obtained from Mrs. T, a 70-year-old woman, brought by her family to the Emergency Room of Wiradadi Husada General Hospital on August 25, 2024, with decreased consciousness and seizures for 3 hours before admission to the hospital. The patient was found falling from her bed with focal seizures. The patient was still in a state of seizures when they arrived at the emergency room and did not respond when given sound or touch stimulation. The patient had clonic seizures in the right hand, like a tremor, and the right side of the face was twitching. The patient was given an anti-convulsant injection of diazepam 10 mg. The patient was then infused with a 0.9% Sodium Chloride solution, followed by injections of citicoline 500 mg, methylcobalamin 500 mcg, and tranexamic acid 500 mg. Because the seizures continued, the patient was injected with a second diazepam injection, with a dose of 5 mg, and then the seizures stopped. Post-seizure, the patient was still unconscious and had weakness in the limbs.

The patient never had a history of seizures before, but he has a history of stroke. During the stroke, the patient felt heavy in the right part of his hand and foot when he visited the neurology clinic, but he was not hospitalized. The patient has no history of diabetes mellitus or hypertension. The patient's family said that the patient had a history of coughing up yellow-green sputum since 7 days ago, and often had shortness of breath and fever. There was no family history of Fahr's disease, and no similar complaints were reported among family members. On physical examination, the patient appeared to have focal seizures in his right hand and twitching on the right side of his face. His blood pressure was 120/82 mmHg, pulse 102x/minute, respiratory rate 22x x/minute, temperature 36 °C, and oxygen saturation 92% room air. On thorax examination, coarse wet bronchi were found in both lungs. Neurological status exams revealed a GCS of E1V1M1. Cranial nerve examination for Nervus VII and XII was difficult to assess. We found bilateral motor weakness in the upper and lower extremities (2/2//2/2).

Laboratory findings revealed hemoglobin 9.30 g/dL (N: 12.00–16.00 g/dL), hematocrit 28.20% (N: 37.00–48.00%), erythrocyte count 3.38×10⁶/µL (N: 4.66–5.80×10⁶/µL), leukocytes 26,870/µL (N: 3,800–11,000/µL) with neutrophil segment 90% (N: 60–70%), sodium 139 mmol/L (N: 136-145 mmol/L), potassium 4.60 mmol/L (N: 3.40-5.10 mmol/L), chloride 100 mmol/L (95-108 mmol/L), random blood glucose 174 mg/dL (N: 70-180 mg/dL), urea 40 mg/dL (10-50 mg/dL), and creatinine 0.70 mg/dL (N: 0.50-1.20 mg/dL). Serum calcium level was not performed, so metabolic causes of intracranial calcification, such as hypoparathyroidism, could not be ruled out.

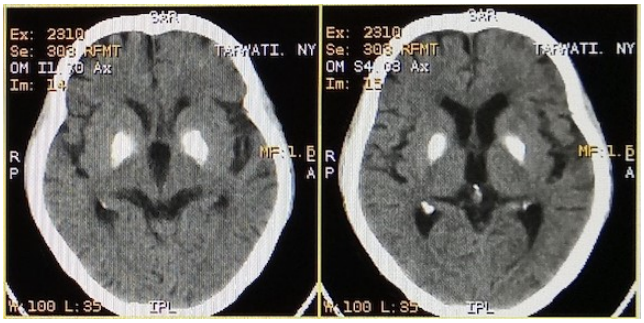

Figure 1. CT-SCAN non-contrast of Head (Source: Radiology Unit of Wiradadi Husada Hospital, Purwokerto)

A non-contrast head CT scan (Figure 1) demonstrated calcification in the bilateral lentiform nucleus, bilateral cerebellum dentate nucleus, and left corona radiata, which is suspected of Fahr's Disease. Thin hypodensity in the left frontal lobe white matter is consistent with the history of chronic ischemia.

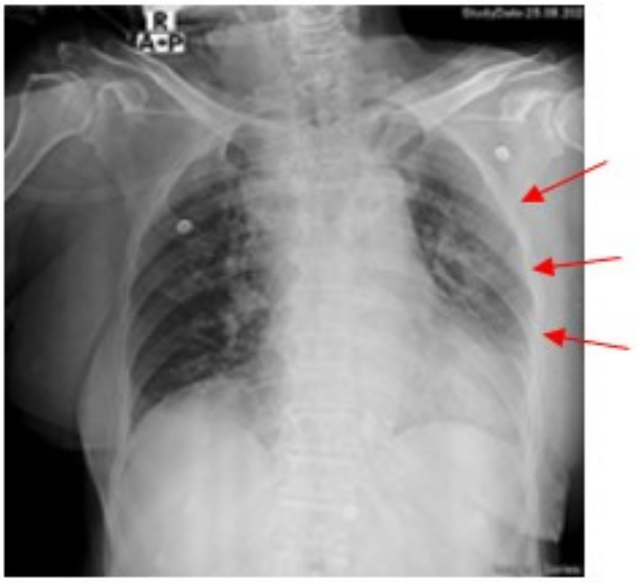

Figure 2. Thorax X-Ray AP (Source: Radiology Unit of Wiradadi Husada Hospital, Purwokerto)

The patient's chest X-ray (Figure 2) revealed increased bronchovascular markings, suggesting bronchopneumonia. Based on the image, laboratory analysis, and physical examinations, the diagnosis of Fahr's Disease, chronic stroke infarct, and bronchopneumonia was supported. The patient was treated with phenytoin therapy as an anticonvulsant, ceftriaxone, and azithromycin for the management of bronchopneumonia. The patient also received other supportive therapy. Following the treatment, the patient showed an improved clinical condition, including increased consciousness, enhanced motor strength, and relief from shortness of breath.

Discussion

Fahr's Disease is characterized by an abnormal calcification of the basal ganglia consisting of calcium carbonate and phosphate.[15-16] This disease is more common in men than in women and usually begins to appear after the age of 30.[17] Although it has been reported to be more common in men, our case highlights the variability and uncertainty in the manifestation of this disease, with the case presented occurring in an older woman.

The pathophysiological mechanisms of calcium and other mineral deposits in the extracellular space and around blood vessels remain incompletely understood. The calcification process usually begins in the vessel wall and the perivascular space and may extend to neurons. Tissue damage occurs due to impaired iron transport and the production of free radicals, which ultimately trigger calcification. This calcification occurs around an area called the Nidus, which is composed of mucopolysaccharides and related materials. The ongoing mineralization process in the basal ganglia tends to compress the blood vessels, causing impaired blood flow, nerve tissue damage, and the formation of mineral deposits.[18]

In general, the distribution of intracranial calcification tends to be extensive and symmetrical if it is based on endocrine, toxic, metabolic, or degenerative etiology. Pathological calcification lesions due to infectious diseases, vascular disorders, or neoplasms are usually scattered and asymmetrical [19]

Diagnosing Fahr's disease requires a detailed anamnesis and physical examination, including neurological examination. Supporting imaging examinations, such as CT-SCAN, are very helpful in determining the etiology of pathological intracranial calcification.[20]

In this case, the patient came with focal seizures for 3 hours. Previously, the patient was in good health but had a history of bronchopneumonia for the past seven days, along with frequent shortness of breath and fluctuating fever. The patient had a history of stroke, but the only complaint at that time was a sensation of heaviness in the right arm and leg. A visit was made to the neurology clinic without requiring hospitalization. There was no history of seizures, toxin exposure, metabolic disorders, or hypoxia. No family history of Fahr's disease was reported. Therefore, this case can be classified as a sporadic form of Fahr's disease.

The relationship between chronic cerebral infarction (stroke) and Fahr's disease is very complex. As mentioned in the case, the patient had a history of stroke infarction confirmed by CT SCAN, which showed a thin hypodensity impression in the left frontal lobe of white matter, consistent with a history of weakness in the extremities. However, until now, there has been no study showing that chronic stroke infarction directly causes Fahr's disease.[15] On the other hand, there are documented cases where individuals with Fahr's Disease have experienced ischemic strokes. For example, a case report of a young patient with Fahr's disease who experienced an ischemic stroke.[16] Another report discusses a patient with Fahr's disease who suffered an ischemic stroke that subsequently underwent hemorrhagic transformation.[21] These cases suggest that, although patients with a history of chronic stroke infarction do not cause Fahr's Disease, individuals with Fahr's Disease may have an increased risk of cerebrovascular events, including stroke. The exact mechanism underlying this association remains unclear and requires further study.

In this case, the patient was also diagnosed with bronchopneumonia from the results of the chest X-ray. Related to the correlation of infection with the occurrence of Fahr's Disease, Snijders et al. did not find any infection that caused calcification in the cranial region, especially in the basal ganglia.[22] Meanwhile, research by Bhangal et al. revealed the possibility of adverse effects of SARS-CoV-2 infection on the central nervous system of patients with pre-existing neurological complications; however, the underlying pathophysiology has not been fully explored.[14] Then, in a study conducted by Khuram et al., two cases of patients with cerebrovascular disorders and Fahr's Disease were reported: a 69-year-old woman with bleeding in the right internal capsule and basal ganglia, and a 72-year-old woman with ischemic stroke and pericallosal artery aneurysm.[23]

In this patient, the presence of bronchopneumonia likely played a role in worsening her clinical condition. Lower respiratory tract infections can trigger systemic inflammatory responses and hypoxemia, which in individuals with neurological disorders such as Fahr's Disease, may exacerbate pre-existing brain dysfunction. Fever, hypoxia, and inflammation from bronchopneumonia can increase seizure susceptibility, worsen consciousness levels, and disturb cerebral metabolic homeostasis. While previous studies [22] have shown no direct link between respiratory infection and basal ganglia calcification, infection can precipitate neurological decompensation, especially in the respiratory tract. In this case, a combination of pre-existing neurological damage, cerebral hypoperfusion from prior stroke, and systemic stress from infection may have contributed to the deterioration observed during hospitalization. Unfortunately, to date, there is no specific treatment available to inhibit the progression of calcification in Fahr's disease.[24-25]

This case report has several limitations. First, as a single case, it cannot establish a causal relationship between Fahr's disease, ischemic stroke, and bronchopneumonia, but only suggests a possible association. Second, the absence of advanced metabolic and genetic testing limits the ability to rule out secondary causes of basal ganglia calcification or confirm an underlying genetic mutation. Third, the follow-up period was short, so the long-term progression of symptoms and the impact of treatment could not be evaluated. Finally, the findings are based on clinical presentations and available imaging from a single healthcare facility, which may limit their generalizability to other populations and settings.

Conclusion

Fahr's disease is characterized by basal ganglia calcification. Although there is no evidence that chronic ischemic stroke directly causes Fahr's disease, individuals with the disease may be at increased risk for cerebrovascular events. This case also involved bronchopneumonia, which worsened the patient's condition. Further research is needed to understand the relationship between Fahr's disease, stroke, and infection, and to explore more effective treatments to inhibit the progression of calcification.

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