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CASE REPORT **OPEN ACCESS**

NON-TRAUMATIC SUBARACHNOID HEMORRHAGIC WITH BRAIN ABSCESS DUE TO EISENMENGER SYNDROME: A RARE CASE REPORT

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ABSTRACT

Background: Non-traumatic subarachnoid hemorrhage in patients with congenital heart disease, such as Eisenmenger syndrome, may involve multiple organs and carries a risk of cerebral abscess due to multiorgan mechanisms.
Case: A 20-year-old male with untreated congenital heart disease since childhood was admitted with severe headache, neck stiffness, projectile vomiting, fatigue, lip curling, slurred speech, and stroke-like symptoms. Neurological deficits included right-dominant tetraparesis, accompanied by cyanosis and clubbing of fingers. CT scan revealed a subarachnoid hemorrhage extending into the perimesencephalic cistern with a thick-walled hypodense lesion, suspected to be a brain abscess. Laboratory findings revealed secondary erythrocytosis, characterized by elevated hematocrit and erythrocyte levels.
Discussion: In this case, the subarachnoid hemorrhage was non-traumatic and perimesencephalic, likely caused by a mycotic aneurysm associated with Eisenmenger syndrome. The brain is a frequent target of septic emboli linked to mycotic aneurysms that trigger hemorrhage. Hemodynamic instability contributes to arterial endothelial degeneration, while hyperviscosity from congenital heart disease and septic embolism facilitates aneurysm formation. Rupture of such aneurysms leads to a sudden rise in intracranial pressure, worsening neurological outcomes.
Conclusion: Subarachnoid hemorrhage in Eisenmenger syndrome is strongly associated with mycotic aneurysms from septic emboli, which may progress to cerebral abscess due to diffuse hypoxia from impaired cardiac output. Early diagnostic evaluation is crucial to establish the etiology, and heightened vigilance is needed in Eisenmenger cases, given the elevated risk of subarachnoid hemorrhage through septic emboli and aneurysm rupture.

Keywords: aneurysma mycotic, brain abscess, Eisenmenger syndrome, septic embolism, subarachnoid hemorrhage

Introduction

Subarachnoid hemorrhage (SAH) is a rare but high-risk condition associated with high morbidity and mortality. The prevalence of SAH reaches 5% of all strokes, with an incidence rate of 9 cases per 100,000 people.[2,9,10]

According to Kok Wai Giang et al., the incidence of subarachnoid hemorrhage in patients with congenital heart disease (CHD) is approximately 2-3% per 10,000 cases per year. This risk is nearly eight times higher in children and young adults than in the general adult population. A recent meta-analysis by Ramez et al. indicated a significantly increased risk of hemorrhagic stroke, specifically SAH, in patients with congenital heart disease, when analyzed across both children and young adults.[20,23]

Eisenmenger syndrome (ES) is a rare congenital heart disease with a prevalence of only 5% of all CHD cases. 11 This disease is characterized by chronic hypoxemia and multiorgan involvement, including secondary erythrocytosis, hyperviscosity, risk of arrhythmias, risk of infection, and progressive heart failure (HF) associated with the congenital heart disease. [11,12,26]

According to Ruiz et al., Eisenmenger syndrome is associated with brain abscesses, occurring in 0.3% to 1.3% of cases per year, caused by Staphylococcus spp. and Streptococcus spp. in patients who have not undergone cervico-cervical surgery. [13]

This case report is significant because there remains a lack of literature discussing ES in conjunction with SAH and brain abscess. It also supports the advancement of knowledge and raises awareness in cases of congenital heart disease with a long-term risk of subarachnoid hemorrhage and cerebral abscess complications.

Case Report

Anamnesis

A 20-year-old young man was admitted to the emergency department with a severe headache with a severe scale (VAS 9) for 1 week. His headache worsened severely one week before admission, and he was not responsive to any analgesics. The patient felt nauseated and vomited with yellow vomiting while her headache worsened. A history of unconsciousness, dizziness, and vertigo was denied. Other complaints include being confused and having difficulty communicating for five days before admission, with fatigue that always felt tired quickly, especially when walking a distance of about 100 m (exercise intolerance), and experiencing cyanosis of the lips and clubbing of the fingers for a few years. Also, he complained of right-sided lip weakness, slurred speech (dysarthria), swallowing problems (dysphagia), attention deficits, and multiple spastic tetraparesis. History of trauma or impact to the head is denied. The patient has a history of congenital heart disease, but has never required treatment. The patient was hospitalized, and further diagnostic imaging workup was not conducted because the MRI was unavailable. Moreover, after three days, the patient was hospitalized, and he passed away.

Physical Examination

The physical examination revealed a general condition that appeared moderately ill, with a level of consciousness (GCS 13; E4 V4 M5). His vital signs were unstable, with blood pressure 110/70 mmHg, heart rate 68 bpm (Figure 2), respiratory rate 26 breaths per minute, peripheral oxygen saturation 59%, and body temperature 37.5 °C. A right-sided lip weakness was noted on his face, as were cyanotic lips in the physical examination. There is a pansystolic murmur on the 5th midaxillary anterior ICS while auscultation, superior and inferior extremities were found clubbing fingers in the physical examination (Figure 1). The neurological status examination revealed a stiff neck, and the Kernig sign was positive. Attention deficits, dysarthria, multiple spastic tetraparesis with muscle strength of superior extremities 3333/4444 and inferior 3333/4444. Cranial nerve examination revealed paresis of the facial nerves, palsy of 7 and 12 of the central type, and pathological reflexes, including a Babinski reflex.

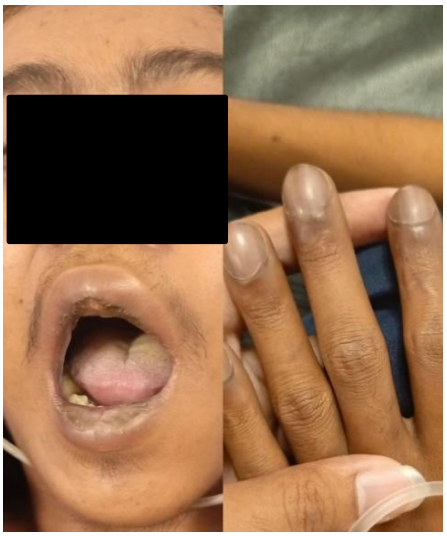

Figure 1. Clinical cyanotic and clubbing finger with facial palsy (paralysis of N. VII and XII central type, UMN)

ECG Finding

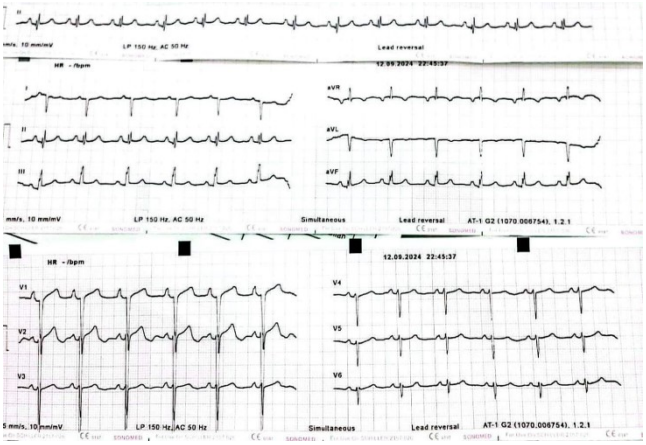

Figure 2. ECG finding appeared sinus rhythm with old anterolateral infarction, right axis deviation, and Incomplete RBBB lead inferior

Laboratory Examination

Complete Blood Count laboratory examination indicated polycythemia/secondary erythrocytosis with hemoglobin 24.9 gr/dL (high), erythrocytes 8.87 million/ul (high), hematocrit 79.9% (high), leukocytes 10,270/uL (normal), platelets 168,000/dl (normal).

Radiology Imaging

Early neuroimaging modality using non-enhanced brain CT on the first day in the emergency department revealed the findings as shown below (Figures 3 and 4).

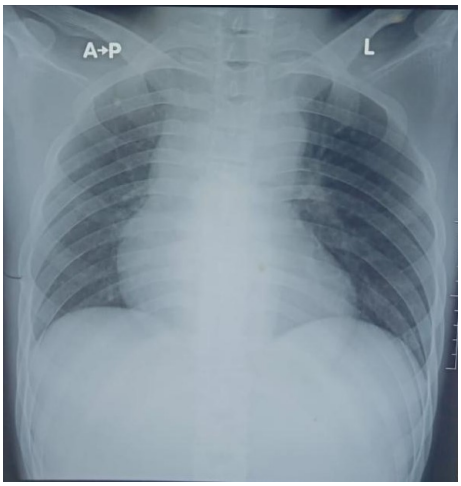

Figure 3. Thorax X-Ray appeared cor boot shaped appearance with pulmo no abnormalities appearance with pulmo no abnormalities

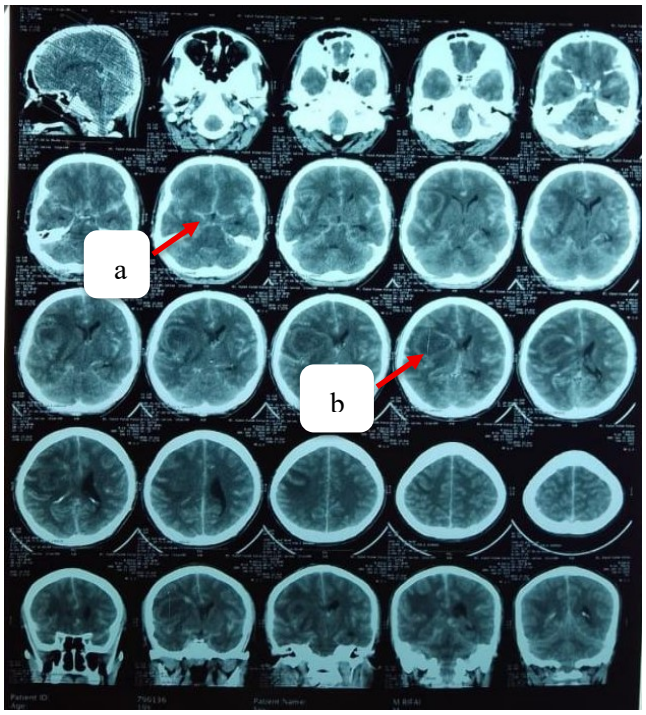

Figure 4. CT Scan appeared a). Massive Subarachnoid Hemorrhage with filling to the perimecencephalic cistern of the cortical sulcus, to the bilateral fissure sylvii and the bilateral cerebral periphalk b). Identified a single mass with a hypodense, slightly enhanced lesion in the brain parenchyma of the right temporoparietal lobe, measuring 4 x 4.2 x 3.1 cm, with suspected brain abscess/tuberculoma. Increased intracranial pressure

Diagnosis Finding

The clinical diagnosis in this case is Tetraparesis with Decrease of Consciousness due to Non-Traumatic Subarachnoid Hemorrhage with Intracranial Mass (mass/brain abscess, tuberculoma) due to Congenital Heart Disease, Eisenmenger Syndrome. The topical diagnosis in this case includes the internal common carotid artery, anterior cerebral artery, anterior communicating artery, and aqueductus of the Sylvian ventricular system. The etiological diagnosis was perimeencephalic subarachnoid hemorrhage, with suspected brain abscess/tuberculoma. The Siriraj Stroke Score was 2.5, which is the interpretation of a hemorrhagic stroke. The Hunt and Hess Scale score was grade 3, indicating drowsiness and confusion, as well as mild motor deficit. The World Federation of Neurosurgeons Scale score was grade III, with a GCS of 13-14, and motoric deficits were present.

Discussion

Subarachnoid Hemorrhage related to this case is a non-traumatic perimesencephalic Subarachnoid Hemorrhage due to a mycotic aneurysm associated with long-term infection and a history of heart disease, Eisenmenger Syndrome, and congenital heart disease.[4,26] According to HoH et al, subarachnoid hemorrhage (SAH) has a low frequency of occurrence but high morbidity and mortality rates. According to Lozada et al, the prevalence of SAH reaches 5% of all types of stroke, with an incidence rate of 9 cases per 100,000 population. [9,10] Signs and symptoms obtained based on the patient's medical history in this case are presenting with sudden, acute, severe headache accompanied by stiffness, projectile nausea and vomiting, decreased consciousness, cranial nerve injury, hemiparesis, and cardiac symptoms with cyanosis and clubbing fingers. [3,5,6,13]

Eisenmenger syndrome is a multisystem disorder characterized by hepatorenal, immunological, neurological, and orthopedic complications. Eisenmenger syndrome (ES) is characterized by chronic hypoxemia and multiorgan involvement, including secondary erythrocytosis and hyperviscosity/microthrombosis.[11,12,26] Hyperviscosity syndrome and secondary erythrosis can turn up with manifestations through the underlying process in the form of a ventricular septal defect with Eisenmenger syndrome.[25] This event will cause coagulation disorders so that the process of hypoxemia occurs, inducing vascular endothelial disorders that reflect symptoms of hyperviscosity and disorders in the form of systemic microvasculopathy.[11,18,20] With inclusion, the mechanism due to the occurrence of congenital heart defects, especially in this case, namely Eisenmenger Syndrome with dominant ventricular septal defect (VSD), results in a process, namely septic embolism that induces mycotic aneurysms.[4,13,16,26]

Septic embolism due to the right-to-left shunt mechanism in Eisenmenger cases with dominant

ventricular septal defect (VSD), which induces coagulopathy factors, septic-induced coagulopathy, and results in systemic inflammation and vascular injury to the endothelium, and has implications for septic thrombus/embolism and endothelial dysfunction that leads to the formation of mycotic aneurysms. [3,13]

This mechanism leads to unstable hemodynamic pressure on the vascular endothelium, resulting in degeneration of the basal lamina interna and loss of the tunica media in the cerebral arteries of the circle of Willis, which triggers the formation of aneurysms.[11,12,23]

In conclusion, venous blood flow in patients with a history of congenital heart disease with low oxygen concentration circulating in arterial blood results in chronic hypoxia as a result of hyperviscosity syndrome and secondary erythrosis, which induces chronic hypoxemia. Hypoxemia disrupts mitochondrial function, which is typically associated with increased hemoglobin concentrations, erythrocytes, and hematocrit (secondary erythrocytosis). This compensatory mechanism increases the blood capacity for supply and demand to peripheral tissues, thus creating a fertile area for microorganisms to grow in the brain parenchymal tissue, especially in the gray/white matter area, which has implications for the formation of brain abscesses because the capsule of brain abscesses is formed in a vulnerable area of cerebral artery damage.[2,13,14,17,23,26]

Brain abscess is characterized by local cerebritis and central necrosis within the brain parenchymal tissue that experiences diffuse hypoxia due to the complexity of microthrombosis and cerebral endothelial mitochondrial dysfunction. The mechanisms of vascular damage (ischemic/hemorrhagic) induce local disruption of the blood-brain barrier. The brain becomes vulnerable to bacterial infection when the blood-brain barrier is damaged (trauma/infection). In this case, signs and symptoms of abscess in patients are increasing headache, focal neurological disorders, nausea and vomiting, and meningeal signs.[13,14,19,24] According to Ruiz et al, explained the association of Eisenmenger Syndrome with the incidence of brain abscess with a percentage of 0.3% - 1.3% of cases per year with a prevalence of about 8% in some cases with a history of infection in brain tissue with staphylococcus spp agents as the most common organisms in patients who have lead into neurosurgery, and streptococcus spp agents as the most common organisms in patients who have not undergone neurosurgery. Signs and symptoms depend largely on the specific location of the abscess, with the most common location being the frontal and parietal regions. The abscess capsule tends to be thinner and more fragile around the lateral region, which is related to reduced blood flow in this area compared to the cerebral cortex. Hematogenous spread is the most common route in patients with congenital cyanotic heart disease, such as Eisenmenger syndrome. [2,13,14,22]

Intracerebral hemorrhage, in this case, a non-traumatic subarachnoid hemorrhage, induces coagulopathy due to neuroinflammation that disrupts the vascular system, bringing out the establishment of septic emboli as a manifestation of congenital heart disease, especially in this case, Eisenmenger Syndrome. Coagulopathy due to septic emboli can cause hemostasis disorders.[16,19,21,22]

Intracerebral hemorrhage induce a mechanism of increased intracranial pressure with Transient Global Cerebral Ischemia (TGCI) which has an impact on the mechanism of Early Brain Injury (EBI) such as diffuse hypoxia due to microthrombosis, where cerebral endothelial dysfunction occurs, disruption of the Blood-Brain Barrier (BBB) so that an excitotoxicity mechanism occurs which has an impact on the occurrence of neuroinflammatory processes, neurovascular damage and neurodegeneration of brain parenchymal tissue due to diffuse infection in the brain, namely cerebritis and in this case with a brain abscess that is directly affected induce mycotic aneurysms.[1,3,5,8,10,13]

This mechanisme induce massive mitochondrial dysfunction which causes abnormalities in cerebral energy metabolism and neuronal cell death occurs when the neuronal inflammatory process triggers astrocytes and microglia cells to get over brain metabolic disorders which are aggravated by activation of the sympathetic nervous system, failure of cerebral autoregulation, systemic inflammation, and platelet activation which causes microthrombosis which has an impact on the occurrence of increased intracranial pressure and intracranial vasospasm.[8,9,10,21]

The correlation between intracranial vasospasm in intracerebral hemorrhage, especially subarachnoid hemorrhage, and oxyhemoglobin concentration in the blood is significant. This correlation is also observed in congenital heart disease and ruptured endothelial walls of blood vessels, causing constriction of arterial smooth muscle after bleeding due to rupture of a mycotic aneurysm.[5,6,8] Cerebral Vasospasme is a complication of the occurrence of a massive decrease in blood vessel diameter due to constriction of arterial smooth muscle after and degeneration of vascular endothelium and neuroinflammatory responses that can result in cerebral ischemia due to the mechanism of subarachnoid hemorrhage which has an impact on the occurrence of Delay Cerebral Ischemia (DCI) due to the impact of the prolonged cerebral vasospasm process and causes cytotoxic edema (intraneuronal)

due to cell membrane dysfunction and osmotic shifts. This develops into vasogenic edema (perivascular and extracellular edema) due to the apoptosis and death of neuronal cells (astrocytes and glia), related to the mechanism of disruption of the blood-brain barrier (BBB). Cerebral edema is associated with massive inflammation of the nervous tissue, excitotoxicity, impaired cerebral autoregulation, microthrombosis, and oxidative stress (Figure 5).[1,6,7,8,10,11,21]

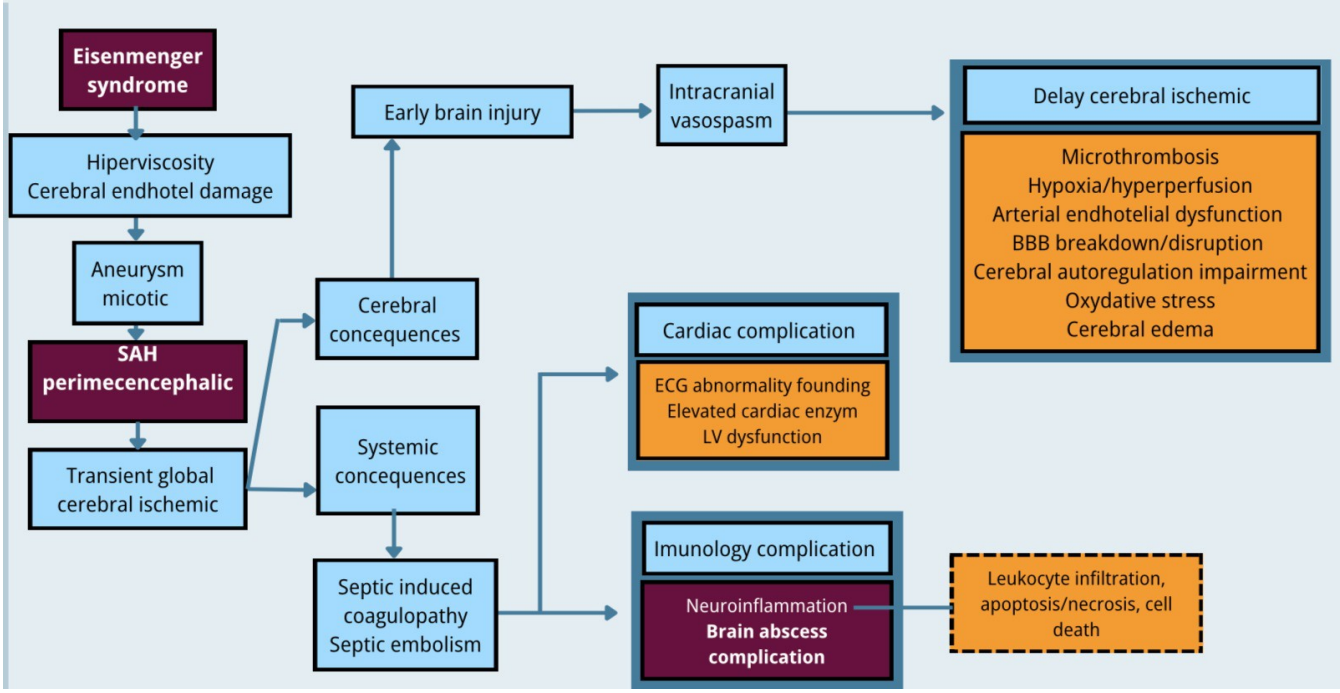

Figure 5. Clinical pathophysiology of medical history with congenital heart disease, especially Eisenmenger syndrome, due to non traumatic perimeencephalic subarachnoid hemorrhage with cerebral infection (brain abscess) induced with mycotic aneurysm and septic embolism (Illustration by the authors)

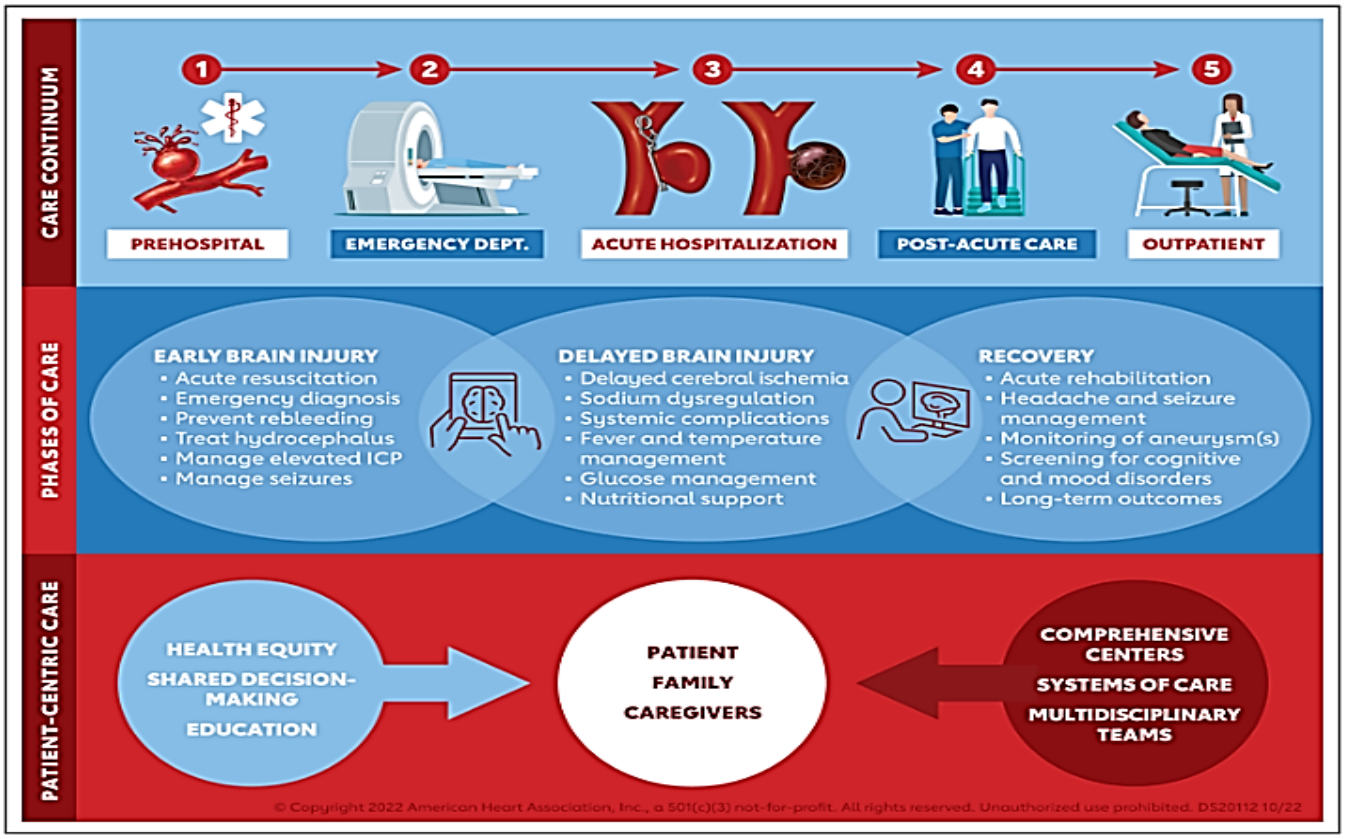

Figure 6. Guideline aims to cover the whole course and care continuum of SAH from the 2023 AHA/ASA Guide to SAH[9] (Illustration by the authors)

The patient's medical history was reviewed to establish a diagnosis, which showed that his headache worsened severely one week before admission, and he was not treated with any analgesics. The patient felt nauseated and vomited with yellow vomiting while her headache worsened. Other complaints include being confused and having difficulty communicating for five days before admission, with fatigue that always felt tired quickly, especially when walking a distance of about 100 m (exercise intolerance), and experiencing cyanosis of the lips and clubbing of the fingers for a few years. Also, he complained of right-sided lip weakness, slurred speech (dysarthria), swallowing problems (dysphagia), attention deficits, and multiple spastic tetraparesis.

A physical examination revealed weakness in the right-sided lip, accompanied by cyanotic discoloration. There is a pansystolic murmur on the 5th midaxillary anterior ICS while auscultation, superior and inferior extremities were found clubbing fingers in the physical examination. The neurological status examination revealed a stiff neck, and the Kernig sign was positive. Attention deficits, dysarthria, multiple spastic tetraparesis with muscle strength of superior extremities 3333/4444 and inferior 3333/4444. Cranial nerve examination revealed paresis of facial nerves, palsy of 7 and 12 of the central type, and pathological reflexes, including a Babinski reflex. A non-contrast CT scan result in this patient, Subarachnoid Hemorrhage was found in the perimecencephalic cistern that filled into cortical sulcus to the bilateral sylvian fissure to bilateral cerebral periphalk, and there was identified single mass with hypodense slight enhanced lesion in the brain parenchyma of the right temporoparietal lobe, involving intracranial enhancement, so the patient was proposed for MRI examination with contrast for further details.

New sections in this 2023 SAH guideline (Figure 6) include nursing care. The risk factors for aneurysm development and rupture, as well as the management of unruptured aneurysms, are not included in this guideline because these topics are addressed in a separate guideline for managing unruptured intracranial aneurysms. The new, important emphases in this guideline are shared decision-making, health equity, and systems of care.[5,8,9]

Treatment given to this patient includes respiratory support (O2 3 L/min), hypervolemic prevention by administering crystalloid infusion, intracranial pressure control in the form of a 30° head tilt, mannitol for intracranial pressure prevention, seizure prophylaxis control with levetiracetam (15-20 mg/kg for 30 minutes), and, in psychiatric conditions such as anxiety, with haloperidol. Analgesics and antipyretics are paracetamol 1 g every 8 hours, gastroprotectors, and antiemetics for symptomatic complaints. Due to a diffuse infection process, a combination of a 3rd-generation cephalosporin and broad-spectrum antibiotics is administered, specifically ceftriaxone every 12 hours and metronidazole every 8 hours. Specific management includes hypertension management and the prevention of intracranial vasospasm with a dose of oral nimodipine 60 mg every 4 hours. Nimodipine is a type of calcium channel blocker that plays a role in reducing the risk of ischemic cerebral damage and improving neurological conditions, as well as serving as a neuroprotectant. Citicoline was administered every 12 hours as a neuroprotectant to prevent apoptosis and further neuronal cell death. The patient was given sildenafil prophylaxis for congenital heart disease, and phlebotomy was performed to prevent hemodilution due to hyperviscosity and secondary erythrosis due to Eisenmenger syndrome.

Patients with subarachnoid hemorrhage, particularly those with signs of cerebral vasospasm, such as neurological deficits, should be considered for triple H hyperdynamic therapy (hypervolemia, hypertension, and hemodilution) to maintain cerebral perfusion pressure. Invasive therapies such as neurosurgical surgery may be considered if possible.

The limitations in this case report is limitation of diagnostic modalities due to further enforcement cannot be carried out and the limitations of research that discuss to the presence of congenital heart disease that has an impact on the occurrence of non-traumatic subarachnoid hemorrhage through the mechanism of septic embolism and mycotic aneurysm with complications in the form of cerebral abscess, so it is interesting to raise this case as a learning material and further scientific progress in the next year after.

Conclusion

Several meta-analyses have shown a significantly increased risk of stroke in patients with congenital heart disease.[20,23] The mechanism of the relationship between SAH and several heart diseases is related to mycotic aneurysms in patients with a history of congenital heart disease, as in this case, specifically Eisenmenger Syndrome (ES). Neurological complications are common in patients with SAH with a history of congenital heart disease, especially in this case of Eisenmenger syndrome, requiring early and aggressive attention, particularly systemic involvement and mechanisms of infection (cerebral abscess).[11,12] It is important to consider the limitations of diagnostic modalities and the application of early assessment/grading systems. Given case reports such as this, consideration of factual and rapid diagnosis, starting with care in the Emergency Department, and early intervention are crucial for conditions such as this.[1,3,6,9]

This case report is expected to provide helpful information in managing complex cases of SAH with systemic comorbidities. It is even hoped to increase awareness of patients with a history of congenital heart disease with complications of SAH and cerebral abscess against the occurrence of mycotic aneurysm and septic embolism in cases of Eisenmenger Syndrome (ES).

Acknowledgment

We thank all contributors who participated in our survey for creating this case report. Hopely, this will help optimize for scientific progress development about rare cases of Congenital Heart Disease, especially Eisenmenger Syndrome with Stroke Hemorrhagic, especially non-traumatic and Cerebral Abscess complications.

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