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CASE REPORT **OPEN ACCESS**

A WOMAN WITH DYKE-DAVIDOFF-MASSON SYNDROME: A CASE REPORT

Fitriani Tri Rahayu*, Hexanto Muhartomo, Aditya Kurnianto, Arinta Puspita Wati

***Correspondence**: drfitrianitrirahayu@gmail.com
Department of Neurology, Faculty of Medicine, Universitas Diponegoro, Semarang, Indonesia

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ABSTRACT

Introduction: Dyke-Davidoff-Masson Syndrome (DDMS) is a rare neurologic condition associated with drug-resistant epilepsy (DRE). Almost 100 DDMS cases were reported, and only 21 were reported among adults. This case report presented Dyke-Davidoff-Masson Syndrome in a 30-year-old woman.
Case: A 30-year-old woman presented with generalized tonic-clonic seizures the day before admission. Each seizure lasted 5 minutes and reoccurred up to three times daily. She communicated but was slow in answering questions post-seizure. Neurological examination revealed left hemiparesis and contracture. Electroencephalography showed diffuse abnormalities. Brain magnetic resonance imaging (MRI) revealed DDMS with crossed cerebellar atrophy. Although anticonvulsant treatment controlled seizures, motor impairments like hemiparesis and joint contractures persisted since childhood.
Discussion: DDMS is a cerebral palsy-related condition resulting from an injury associated with calvarium immaturity or childhood brain damage. Prenatal and post-natal injury may be the etiologic factors of DDMS. The clinical features of DDMS are cerebral hemiatrophy or hypoplasia, facial asymmetry, seizure, and contralateral hemiplegia with diverse combinations and severity. Specifically, in this case, the MRI showed right-sided brain atrophy, enlarged right lateral ventricle, and crossed cerebellar atrophy, all consistent with DDMS. Compensatory changes in the right skull, like thickened bones and enlarged frontal sinus, were also observed. Symptomatic management is the mainstay of DDMS treatment, one of which is antiepileptic medication. Surgery is indicated as the last therapeutic option in some instances.
Conclusion: DDMS cases in adults are rare. At present, there is no management algorithm for DDMS. Thorough history taking and physical and radiology examinations are required for DDMS diagnosis.

Keywords: adult, cerebral hemiatrophy, Dyke-Davidoff-Masson Syndrome, seizure

Introduction

Dyke-Davidoff-Masson Syndrome (DDMS), or cerebral hemiatrophy, is a rare neurologic condition. It was first described by Dyke, Davidoff, and Masson when they observed nine patients with hemiplegia and plain skull x-ray changes. It was associated with drug-resistant epilepsy.[1] The classical findings are hemiparesis, seizure, facial asymmetry, and intellectual disability. Pharmacological therapy is not sufficient for seizures in most cases. There are differences between adult and pediatric DDMS management.[2] The clinical and radiology characteristics are based on the intensity of cerebral injury.[3]

The frequency of DDMS is still unknown, but most DDMS cases occur in children, and most literature is based on case reports or series.[4,5] The diagnosis of DDMS was established according to the correlation between radiologic and clinical characteristics. Many clinicians misdiagnose and mistreat DDMS because of its rarity.[6] Almost 100 DDMS cases were reported, and only 21 were reported among adults.[7] This case report presented DDMS in a 30-year-old woman.

Case Report

A 30-year-old woman presented with a seizure episode that occurred one day before admission. The seizure began while she was engaged in physical play, with initial stiffness in her body, followed by her eyes rolling upwards and a generalized tonic-clonic episode. The seizure lasted approximately 5 minutes, during which she lost consciousness. Following the episode, she exhibited typical postictal drowsiness, sleeping for 15 minutes. Upon waking, she could resume normal activities and communicate, though with a slight delay in response. The patient reports experiencing up to three seizures daily with similar characteristics, each followed by postictal fatigue and weakness in her left extremities. While hospitalized, she experienced another seizure, which mirrored her previous episodes in presentation. She denies fever, vomiting, headache, blurred vision, or tingling symptoms.

The patient's epilepsy history began in early childhood, initially manifesting as febrile seizures at the age of 2. Her first seizure was generalized, triggered by a high fever, and lasted approximately 3 minutes without any focal signs. These febrile seizures continued to occur with a frequency of 2 to 3 episodes per year until the age of 5, after which they gradually decreased in frequency and eventually ceased altogether. At the age of 10, she experienced her first unprovoked seizure, which was not associated with fever and marked the onset of her epilepsy. These seizures were generalized tonic-clonic in type, characterized by full-body stiffening, complete loss of consciousness, and followed by a postictal phase that included pronounced drowsiness and transient confusion. Over the years, the frequency of these unprovoked seizures increased progressively, particularly during her late teenage years and early twenties, eventually becoming more regular, with episodes occurring almost monthly by the time she reached 25 years of age.

The patient did not receive regular anticonvulsant therapy until she reached 25, by which time her seizure frequency had escalated to multiple episodes per month, significantly impacting her daily life. At this point, she was prescribed antiepileptic medication; however, adherence to the medication regimen was inconsistent. Over the following years, the frequency and severity of her seizures showed considerable fluctuation, with specific periods characterized by temporary remission where she experienced no seizures. It is worth noting that her most recent documented seizure took place approximately one year before this admission, during a time when she was not undergoing any consistent treatment or taking any routine antiepileptic medication as part of her management plan.

The physical examination revealed that she was moderately ill, compos mentis, and her vital signs were typical. Her neurology examination revealed left hemiparesis and contracture. The patient demonstrates left-sided hemiplegia, particularly in the upper and lower limbs on the left side of the body. Upon closer observation, the affected limbs show a significant discrepancy compared to their counterparts on the right side, with the left limbs appearing notably smaller. This size difference strongly indicates muscle atrophy, likely resulting from prolonged disuse over an extended period. Additionally, a subtle facial asymmetry is observed, with the left side appearing slightly flattened compared to the right, consistent with the diagnosis of Dyke-Davidoff-Masson Syndrome. These features suggest that the patient's hemiplegia and associated motor deficits likely began in early childhood and progressed over time (Figure 1).

The laboratory examinations and chest X-ray results were within normal limits and showed no significant abnormalities. On the other hand, the electroencephalography findings predominantly demonstrated low voltage patterns, which indicate diffuse abnormalities in the brain's electrical activity. Brain MRI revealed cerebral hemiatrophy with chronic infarct in the right frontoparietotemporooccipital lobe, diploic thickening of right frontoparietoocipital bone, small left cerebellum, and right frontal sinus hyper pneumatization with Dyke-Davidoff-Masson syndrome with crossed cerebellar atrophy impression (Figure 2). Based on the clinical history, physical examination, and radiology examination, her diagnosis was generalized tonic-clonic seizure due to Dyke Davidoff Masson Syndrome (Figure 3). The anticonvulsant treatment successfully controlled her seizures, but her motor impairments have likely persisted since childhood.

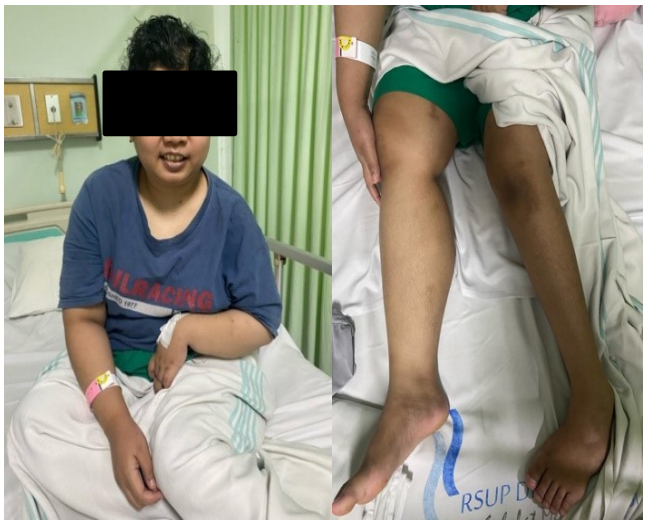

Figure 1. Patient's Clinical Condition

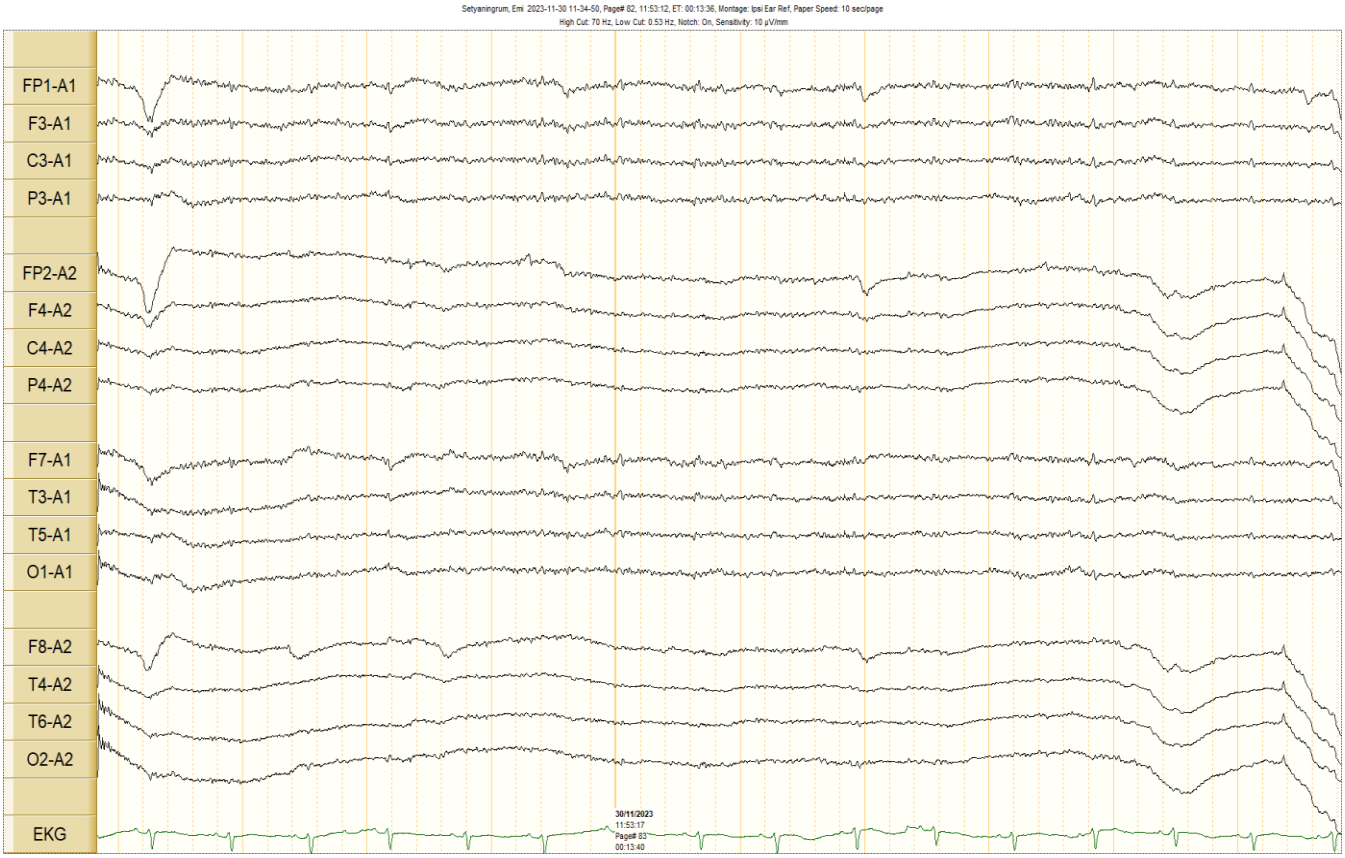

Figure 2. Electroencephalography (Generalized Low Voltage indicated Diffuse Electroencephalographic Abnormalities)

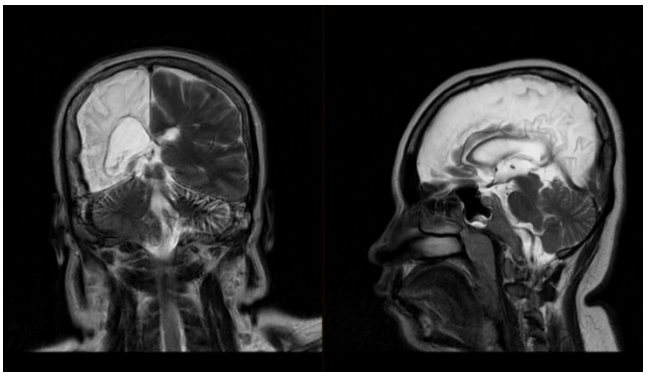

Figure 3. Coronal and Sagittal Brain Epilepsy MRI (Dyke-Davidoff-Masson syndrome with crossed cerebellar atrophy)

Discussion

Dyke-Davidoff-Masson Syndrome is a cerebral palsy-associated condition due to an injury associated with calvarium maturation or brain injury during childhood. Prenatal and postnatal injuries are etiologic factors of DDMS. There are no specific gender or hemisphere preferences, but most cases occur in children and involve the left hemisphere.[1,2,6] A case series stated that DDMS had an age range of 5 – 62 years with a mean of 34.1 ± 21.7 years.[3] It is different from our patient, who was female and had a right hemisphere abnormality, but her age was within the age range stated in the literature.

The etiology of DDMS is cerebral injury and probably occurs intrauterine, in which the maturation of the calvarium has not been completed yet, or brain injury in the first three years of life. DDMS is subdivided into two types based on its etiologies, including congenital DDMS, which appears in childhood, and its pathophysiology, including fetal vascularization obstruction, infection, and vascular injuries; and acquired DDMS, which also appears in childhood, and its etiology mechanisms including perinatal hypoxia, infection, cerebrovascular abnormalities, anoxia, hypoxia, tumor, prolonged febrile seizure, birth trauma, and cranial trauma. The mechanism of progressive cerebral hypoplasia and neurological disorders is hypothesized due to sequential ischemic events, which are caused by various reasons that limit the brain's neurotrophic factor synthesis.[6,8–10] Brain injury can also affect brain development and result in the loss of neurons that comprise the growth of the developing brain, leading to intellectual disability, seizures, and learning disability.[11] As stated in the literature, our patient had acquired DDMS. The form of cerebral hemiatrophy is generally associated with prenatal or early postnatal brain injury, such as hypoxia, vascular events, or trauma. Although the patient experienced recurrent febrile seizures starting at age 2, this is more likely a consequence of the underlying brain abnormality rather

than its cause. Additionally, although her growth milestones were reportedly typical, a detailed cognitive assessment, such as an IQ test, would be necessary to quantify any cognitive impairment, a known feature in patients with DDMS.

The clinical features of DDMS are cerebral hemiatrophy or hypoplasia, facial asymmetry, drug-resistant seizure, intellectual disability, and contralateral hemiplegia with various combinations and severity. Several studies showed that DDMS patients can also present with oral manifestations like delayed unilateral teeth eruption, hypoplasia, and taurodontism. Limb asymmetry may not be found in DDMS patients. Contralateral cerebellum atrophy is a rare finding in DDMS. [12–14] Cerebellar hemisphere atrophy occurs with damage to supratentorial brain tissues and decreased blood flow and metabolism in the acute phase, resulting in atrophy in the chronic phase. Its pathophysiology is not clearly understood, but is most likely associated with the involvement of corticopontocerebellar tracts connecting the cerebellum and the contralateral cerebral hemisphere and the severing of neuronal functional ligaments.[15]

Seizure is the most common symptom, and most seizures in pediatric DDMS are refractory seizures. The classic triad of DDMS is epilepsy, intellectual disability, and hemiplegia or hemiparesis, but this triad is only found in 16.6% of patients. Facial asymmetry is found in 33.7% of patients.[13,16] A literature review showed that patients often had seizures at night, particularly right after falling asleep. At the same time, there are instances where seizure activity can be influenced by physiological changes during sleep; nocturnal seizures in DDMS are likely related to the brain's structural abnormalities, including cerebral atrophy and impaired connectivity between hemispheres. These abnormalities can disrupt regular electrical activity, leading to seizures, regardless of the time of day. In addition, vagal nerve excitation and the "wearing-off" phenomenon of antiepileptic drugs occur at night, both of which contribute to nocturnal epilepsy. Hemiparesis may not be found in several DDMS cases.[10,17] Our patients had hemiparesis and seizure, which did not fulfill the classic triad of DDMS. The interesting finding in our case is contralateral cerebellum atrophy, a rare finding in DDMS cases.

Hemiatrophy and "shifted falx" signs may be revealed in ultrasonography at 29 weeks of gestation. A skull plain radiograph may reveal unilateral calvarium thickening with paranasal sinuses and mastoid air cell expansion.[18] Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are gold-standard DDS diagnostic modalities. Total and subtotal cortical hemiatrophy are the pathognomonic radiologic findings in DDMS. Unilateral cerebral atrophy may be found in the cerebral peduncle, thalamus, pons, contralateral cerebellum, and surrounding areas. The other radiologic findings in DDMS are prominent cortical sulcus, lateral ventricle dilatation, frontal sinus and mastoid cells hyperpneumatization, calvarium bone thickening, elevated temporal bone, elevated ipsilateral greater wing of sphenoid and petrous ridge, and skull compensatory hypertrophy.[1,8,19]

Hyperpneumatization is common in the frontal sinuses because the frontal sinuses last and expand up to adolescence. The imaging features are more prominent as the patients become older.[18] Hyperintensity areas may be found in T2-MRI, and fluid-attenuated inversion recovery (FLAIR) may reveal encephalomalacia and gliosis due to vascular or infectious injuries. There are three patterns of cerebral hemiatrophy in MRI: pattern I is diffuse subcortical atrophy, pattern II is diffuse cortical atrophy with porencephalic cysts, and pattern III is infarct history with gliosis in the medial cerebral artery area. [8,20]

The differential diagnoses of DDMS are Sturge-Weber syndrome, Rasmussen encephalitis, basal ganglia germinoma, linear nevus syndrome, Fishman syndrome, Silver-Russell syndrome, Parry-Romberg syndrome, and hemimegaloencephaly. Thorough history taking, clinical examination, and neuroimaging are necessary to differentiate and exclude them.[3,16,18,21,22] Sturge-Weber syndrome, Rasmussen encephalitis, and Silver-Russell syndrome can be differentiated through clinical examination and cross-sectional neuroimaging. Rasmussen encephalitis does not show calvarial changes with similar clinical history; Sturge-Weber syndrome commonly shows enhancing pial angiomas, cortical calcification, and facial port-wine nevus; and Silver-Russell syndrome has classic phenotypes including clinodactyly, delayed bone age with normal intellectual and head circumference.[23]

In hemimegaloencephaly, there is a hamartomatous overgrowth of the cerebral hemisphere with the ipsilateral enlarged lateral ventricle, which causes apparent atrophy of the contralateral hemisphere. In Parry-Romberg syndrome, there are microhemorrhages, calcification, white matter abnormalities, cortical dysgenesis, meningeal thickening, and enhancement.[18] Basal ganglia germinoma is a rare tumor. It commonly presents with progressive hemiparesis and cerebral hemiatrophy, with radiology examination, which shows cystic areas, focal hemorrhage, and minor surrounding edema with calvarial changes. Fishman syndrome or encephalocraniocutaneous lipomatosis is a rare neurocutaneous syndrome, including unilateral ocular lipodermoid, which frequently presents with seizures, neuroimaging reveals cortical calcification and hemiatrophy. Linear nevus syndrome frequently

presents with facial nevus, refractory seizure, intellectual disability, and unilateral ventricle dilatation, which resembles cerebral hemiatrophy.[2]

In our case, the physical examination revealed left hemiparesis and contracture. The radiologic findings are in line with radiologic features of DDMS, including hyperpneumatized frontal sinus, contralateral cerebellar and cerebral hemispheres atrophy, and calvarial thickening (diploic thickening of right frontoparietoocipital bone), and the differential diagnoses of DDMS can be excluded by clinical and radiologic findings.

There is no established management protocol for DDMS at present.[6] The primary treatment of DDMS is symptomatic, one of which is antiepileptic medication. Surgery is indicated as a last-choice therapy in some instances.[7,24] The anti-convulsant may be given as monotherapy or combination therapy.[19,25] If DDMS occurs in adulthood, anti-epileptic medication is preferred over surgical management if the seizures are under control. In addition, the patients should have supportive physiotherapy, speech therapy, and occupational therapy to improve their quality of life.[16,26] The physiotherapy includes transcutaneous electrical stimulation, conventional physiotherapy, and task-specific exercise. The goal of physiotherapy is functional recovery and increasing brain plasticity.[27] Our patient had a combination of antiepileptic medication, including 10 mg diazepam IV, 200 mg phenytoin IV q24h, and 500 mg valproic acid per oral q12h. The anticonvulsant treatment successfully controls her seizures, and no surgery is indicated for her. Furthermore, she was advised to undergo physiotherapy to improve her motor function.

Surgical management with cerebral hemispherectomy is the therapeutic choice for DDMS with refractory seizures, with an 85% success rate.[8,16,19,25] The prognosis is better if hemiparesis occurs after 2 years and there is no refractory or prolonged seizure.[6,8,21] The diagnosis is often delayed and may be missed because many patients do not present with a triad of DDMS, the symptoms appear at different times, or due to its rarity. Early diagnosis can increase the success rate of supportive therapy.[9,28] Our patient had a good prognosis because symptoms appeared after the age of 2 years, and she had no refractory seizures after having antiepileptic medication.

Conclusion

Dyke-Davidoff-Masson Syndrome is a rare neurological disorder characterized by cerebral hemiatrophy, seizures, and hemiparesis. In this case, the patient exhibited long-standing hemiparesis and recurrent seizures, which were effectively managed with antiepileptic medication. However, the motor deficits, including hemiparesis, remained unchanged, reflecting the irreversible nature of the condition.

The diagnosis of DDMS was confirmed through brain MRI, the gold standard examination for this syndrome. MRI revealed characteristic findings, including right cerebral hemispheric atrophy, compensatory hypertrophy of the skull, and crossed cerebellar atrophy. These imaging results and the clinical presentation were essential for an accurate diagnosis.

In conclusion, early recognition of DDMS and using MRI as a diagnostic tool is crucial for patient management. While symptomatic treatment, particularly seizure control, can improve the patient's quality of life, motor deficits such as hemiparesis are typically irreversible. A multidisciplinary approach, including regular neurological evaluations and supportive therapies, is recommended for long-term management.

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