Indonesian Journal for Health Sciences
Vol. 10, No. 2, September 2026, pp. 148-155
ISSN 2549-2721 (Print), ISSN 2549-2748 (Online)

DYKE DAVIDOFF MASSON SYNDROME IN A 34-YEAR-OLD FEMALE: A RARE CASE

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Article History:
Submitted: 27/01/2026
Accepted: 12/08/2026
Published: 11/09/2026

Keywords:
Dyke-Davidoff-Masson Syndrome,
Visuospatial Impairment
Seizures

ABSTRAK

Abstract:
Dyke–Davidoff–Masson Syndrome (DDMS) is a rare neurological disorder with fewer than 100 cases reported worldwide. It is characterized by cerebral hemiatrophy, calvarial thickening, and hyperpneumatization of paranasal sinuses, often associated with hemiparesis, seizures, and developmental delay. Adult cases with symptoms persisting for more than three decades are exceptionally uncommon and offer valuable insight into long-term disease progression. Case Presentation: A 34-year-old woman was reported with a history of epileptic seizures since the age of 9 months, occurring 3–4 times per month and persisting into adulthood. The seizures were characterized by impaired consciousness accompanied by right-sided tonic–clonic motor manifestations and were preceded by a visual aura. The patient also exhibited right hemiparesis and delayed motor development. Neurological examination revealed right-sided central facial (cranial nerve VII) and hypoglossal (cranial nerve XII) paresis, right-sided limb spasticity, and visuospatial dysfunction. Non-contrast head computed tomography (CT) demonstrated left cerebral hemiatrophy, left lateral ventricular dilatation, left calvarial thickening, and hyperpneumatization of the left frontal sinus. This case was most appropriately classified as focal epilepsy of structural etiology secondary to DDMS, with a seizure type consistent with focal impaired consciousness seizure with observable manifestations.

***Abstrak*:**
Dyke-Davidoff-Masson Syndrome (DDMS) adalah kelainan neurologis yang sangat jarang, dengan jumlah laporan kasus di dunia kurang dari 100. Penyakit ini ditandai oleh hemiatrofi serebral, penebalan kalvaria, dan hiperpneumatisasi sinus paranasal, disertai hemiparesis, kejang, dan gangguan perkembangan. Laporan kasus pada pasien dewasa dengan riwayat gejala lebih dari tiga dekade sangat jarang ditemukan, sehingga dapat memberikan gambaran mengenai perjalanan penyakit tersebut. Kasus: Dilaporkan seorang perempuan berusia 34 tahun dengan riwayat bangkitan epilepsi sejak usia 9 bulan yang terjadi 3–4 kali per bulan dan menetap hingga dewasa. Bangkitan ditandai dengan gangguan kesadaran disertai manifestasi motorik tonik–klonik pada ekstremitas kanan yang didahului oleh aura visual. Pasien juga mengalami hemiparesis kanan dan keterlambatan perkembangan motorik. Pemeriksaan neurologis menunjukkan parese sentral nervus VII dan XII kanan, spastisitas ekstremitas kanan, serta gangguan fungsi visuospasial. CT-scan kepala tanpa kontras menunjukkan hemiatrofi serebri kiri, dilatasi ventrikel lateralis kiri, penebalan kalvaria kiri, dan pembesaran sinus frontalis kiri. Kasus ini dikategorikan sebagai epilepsi fokal dengan etiologi struktural akibat DDMS, dengan tipe bangkitan focal impaired consciousness seizure with observable manifestations.

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How to Cite:
R. Defryantho, F. Langkole, N.F. Ahmadi, "Dyke Davidoff Masson Syndrome in a 34 Year Old Female: a Rare Case", Indonesia. J. Heal. Sci., vol. 10, no. 2, pp. 148-155, 2026.

INTRODUCTION

Dyke–Davidoff–Masson syndrome (DDMS) was first recognized and reported by Dyke, Davidoff, and Masson in 1933 [1]. It is also known as cerebral hemiatrophy and remains a rarely encountered neurological condition, with precise global prevalence and incidence unknown; fewer than 100 cases have been reported worldwide [1] [2]. The classical phenotype comprises hemiparesis, facial asymmetry, recurrent seizures, learning difficulties, and developmental delay [3]. Not all patients develop intellectual disability, and seizures may arise months to years after hemiparesis becomes apparent. Speech and language difficulties may also occur [4]. DDMS may be congenital or acquired. The condition most commonly becomes evident in childhood as a consequence of an in utero, perinatal, or early-life brain insult [3].

The congenital or primary form results from intrauterine or neonatal brain injury, typically of a vascular origin, with symptoms apparent at birth or shortly thereafter. The acquired form can develop in the neonatal period or later in life and may be triggered by trauma, infection, cerebrovascular abnormalities, hemorrhage, ischemia, immune-mediated disorders, tumor, or idiopathic causes [5] [6]. Neuroimaging features include unilateral cerebral volume loss (hemiatrophy), ventriculomegaly, compensatory calvarial thickening, and hyperpneumatization of the paranasal and frontal sinuses [2].

Here we present a 34-year-old woman with clinical and radiological findings characteristic of DDMS. This report underscores the importance of thorough history-taking, physical examination, and neuroimaging in establishing the diagnosis of this uncommon entity.

CASE PRESENTATION

A 34-year-old woman presented to Mega Buana Palopo Hospital with a seizure characterized by tonic–clonic movements of the right extremities lasting less than 5 minutes, accompanied by frothing at the mouth and upward deviation of the eyes. She was conscious before the seizure, developed impaired consciousness during the event, and regained consciousness after its cessation. Prior to the seizure, she experienced a visual aura described as seeing bright lights. According to the 2025 ILAE classification, these episodes were focal impaired consciousness seizures with observable manifestations, characterized by tonic–clonic motor activity involving the right extremities and preceded by a visual aura [7]. There was no documented bilateral tonic–clonic propagation; therefore, the seizures could not be definitively classified as focal-to-bilateral tonic–clonic seizures. The patient had experienced seizures 3–4 times per month for more than 30 years, with a consistent pattern. She was born at term by cesarean section without antenatal, perinatal, or postnatal complications. According to her mother, the first seizure occurred at 9 months of age without signs of infection. Motor development was delayed; at one year old she was unable to walk and dragged her leg. Her formal education ended at junior high school. There was no history of head trauma and she had not been receiving regular neurological follow-up. She is the second of three children; her siblings have normal development. The patient had a history of antiseizure medication use, specifically carbamazepine 200 mg administered twice daily. However, the medication was not taken regularly, indicating poor treatment adherence. According to the medical records, there was no history of status epilepticus. The patient had also never undergone medical rehabilitation or physiotherapy. Following the most recent hospitalization, the patient did not return for follow-up visits, precluding assessment of long-term clinical outcomes.

On examination she was alert and hemodynamically stable. There were no signs of meningeal irritation (negative

nuchal rigidity, Lasègue, Kernig, and Brudzinski signs). Cranial nerve testing showed normal function of cranial nerves I, II, III, IV, VI, VIII, and IX–XI. There was right-sided central paresis of cranial nerves VII and XII (figure 1). Motor testing revealed full strength (5/5) in the left limbs; on the right, strength was Medical Research Council grade 3 in the upper extremity and 4 in the lower extremity, with spasticity of the right upper limb (figure 2). Physiological reflexes were brisk on the right (biceps, triceps, patellar, and Achilles). Pathological reflexes (Babinski, Chaddock, Oppenheim, Hoffmann–Tromner) were absent. On the Mini-Mental State Examination (MMSE), the patient achieved a score of 24/30. Cognitive screening using the Montreal Cognitive Assessment–Indonesian Version (MoCA-INA) yielded a score of 22/30. The patient demonstrated particular difficulty with tasks involving visuospatial function, especially figure-copying tasks. Interpretation of the cognitive assessment results was undertaken with consideration of the patient's educational background, which was limited to junior high school level. Furthermore, the patient had been left-hand dominant since childhood; therefore, the potential impact of right-sided hemiparesis and spasticity on test performance was considered minimal. Non- contrast head CT (Figure 3) demonstrated left cerebral hemispheric atrophy with hyperpneumatization of the left frontal sinus and left-sided calvarial thickening, as well as dilatation of the left lateral ventricle. There was no midline shift. Electroencephalography (EEG) and magnetic resonance imaging (MRI) were not performed because these diagnostic modalities were unavailable at the hospital where the patient received treatment. Consequently, the evaluation of this case was based on the combination of clinical manifestations and findings from a non-contrast head computed tomography (CT) scan. Written informed consent for publication was obtained from the patient. Ethical approval was granted under No. 016/UMBP/KEPK/V/2025.

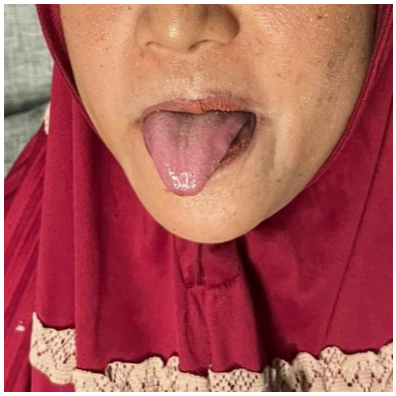

Figure 1. Right central paresis of cranial nerve XII.

Figure 2. Spasticity of the right upper extremity.

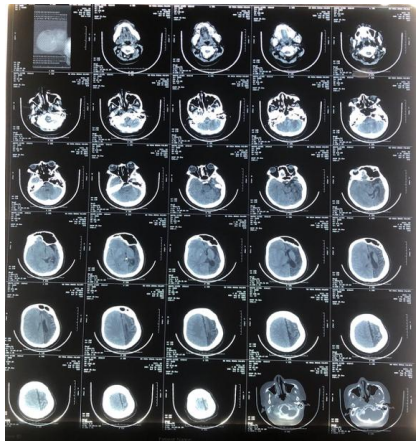

Figure 3. Non-contrast head CT showing left cerebral hemispheric atrophy with enlargement of the left frontal sinus and left-sided calvarial thickening, accompanied by dilatation od the left lateral ventricle. No midline shift.

DISCUSSION

The diagnosis of *Dyke-Davidoff-Masson Syndrome* (DDMS) was established based on the presence of

characteristic clinical features, including early-onset seizures, delayed motor developmental, right hemiparesis, and mild cognitive impairment, as well as radiological evidence of left cerebral hemiatrophy with associated compensatory calvarial changes. The original description by Dyke, Davidoff, and Masson in 1933 detailed radiographic changes on skull radiographs and pneumoencephalography in nine patients who exhibited hemiparesis, seizures, facial asymmetry, and intellectual disability [8] [9] [10]. Early brain injury may lead to compensatory cranial changes such as calvarial thickening and sinus enlargement ipsilateral to the affected hemisphere. Intellectual disability is not universal, and seizures can occur long after the onset of hemiparesis [9].

Dyke-Davidoff-Masson syndrome (DDMS) is a neurological disorder associated with prenatal or postnatal cerebral insult, resulting in cerebral hemiatrophy accompanied by compensatory changes of the cranial vault. Based on the timing of the cerebral injury, DDMS is classified into congenital and acquired forms. In the congenital form, the insult occurs during intrauterine life and is typically associated with a shift of midline structures toward the affected hemisphere. In contrast, the acquired form develops after birth and may result from various etiologies, including trauma, vascular abnormalities, infections, and intracranial hemorrhage [11]. In the present case, the patient experienced the first seizure at the age of 9 months, accompanied by delayed motor development and chronic hemiparesis. Computed tomography (CT) findings revealed left cerebral hemispheric hemiatrophy, calvarial thickening, frontal sinus hyperpneumatization, and ipsilateral lateral ventricular dilatation, indicating a long-standing cerebral insult that likely occurred during an early stage of brain development. Although the early-life onset of clinical manifestations and the characteristic neuroimaging features are more suggestive of the congenital form than the acquired form of DDMS, there is no direct evidence confirming that the cerebral injury occurred during the prenatal or perinatal period. Therefore, this case is more appropriately considered as probable congenital or early-life DDMS. The onset of seizures at 9 months of age with a high frequency (3–4 episodes per month for 30 years) confirms the chronic nature of the condition. According to the 2025 ILAE seizure classification, the episodes were classified as focal impaired consciousness seizures with observable manifestations, characterized by an elementary visual aura followed by right-sided tonic–clonic motor activity and impaired consciousness [7]. Motor developmental delay may suggest neurological abnormalities since the perinatal period or structural brain anomalies that developed early in life and persisted.

Dyke–Davidoff–Masson syndrome is a rare neurological syndrome characterized by a range of clinical manifestations, with epileptic seizures being one of the most commonly observed features. In the acquired form, seizures are generally focal and are associated with better seizure control and prognosis, whereas in the congenital form, most patients experience generalized tonic–clonic seizures, although absence seizures have also been reported. Although the seizure pattern in this patient does not fully correspond to the pattern most commonly reported in the congenital form, the early onset of seizures and the chronic disease course remain consistent with the clinical characteristics of DDMS. The clinical manifestations of DDMS are strongly influenced by the timing of cerebral injury; therefore, symptoms may present during infancy or remain undetected until later in life, depending on the location and extent of brain damage [5] [12].

The focal motor deficits observed in this patient, including right-sided central paresis of the cranial nerve VII) and hypoglossal (cranial nerve XII) nerves, as well as limb spasticity and hyperreflexia,

reflect involvement of the left corticospinal tract secondary to cerebral hemiatrophy, a characteristic neurological manifestation of DDMS. In this case, the patient had never undergone medical rehabilitation or physiotherapy. Nevertheless, cortical plasticity enables structural and functional reorganization of the brain following injury, a process that can be enhanced through motor training, sensory stimulation, and exposure to an enriched environment. Therefore, targeted rehabilitation interventions may facilitate functional recovery and improve clinical outcomes in patients with DDMS [13].

The patient's difficulty in reproducing drawings on the MMSE, as well as the low score in the visuospatial domain of the MoCA-INA, is consistent with the study by Zink et al. [14], which reported that visuospatial function primarily involves the right hemisphere, particularly the parietal lobe (angular gyrus and supramarginal gyrus), and is associated with the integrity of the parietal and temporal cortices [14]. In general, DDMS may present with several clinical features, particularly cognitive impairment [12] [15]. Cognitive dysfunction in epilepsy is strongly influenced by the location of seizure activity in the brain, especially when involving the hippocampus, which plays a crucial role in memory. Seizures can disrupt short-term, long-term, and spatial memory through alterations in hippocampal cell circuits. The most severe cognitive impairment often occurs in patients with epilepsy onset in early childhood, particularly in epileptic syndromes [16]. Cognitive deficits typically vary depending on the location and extent of brain damage, and visuospatial function is usually less affected compared to other domains such as attention and memory [17].

The interpretation of the MMSE and MoCA-INA results in this case should be undertaken with caution. In addition to the structural brain abnormalities associated with DDMS, cognitive test performance may also be influenced by patient-related factors, including educational attainment. The patient had completed junior high school education and had used the left hand as the dominant hand since childhood; therefore, the potential impact of right-sided hemiparesis and spasticity on test performance was likely minimal. Furthermore, information regarding the interval between the most recent seizure episode and the cognitive assessment was unavailable, precluding evaluation of the potential influence of postictal effects on the test results.

The differential diagnosis of DDMS includes several conditions associated with unilateral cerebral hemiatrophy, such as *Sturge–Weber syndrome*, certain brain tumors, *Silver syndrome*, and *linear nevus sebaceous syndrome*. However, accurate diagnosis requires careful correlation between the clinical history and neuroimaging findings. In the present case, the history of seizures since the age of 9 months, right-sided hemiparesis, delayed motor development, and CT findings of left cerebral hemiatrophy accompanied by calvarial thickening and frontal sinus enlargement were more consistent with a diagnosis of *Dyke-Davidoff-Masson syndrome* than with other differential diagnoses [4] [13]. Management is focused on symptoms such as seizures, hemiplegia, hemiparesis, and learning difficulties. Prognosis is more favorable when hemiparesis develops after the age of 2 years without recurrent seizures. In severe cases that are unresponsive to therapy, hemispherectomy may be an option, with a reported success rate of up to 85% in carefully selected patients [18] [19]. Physiotherapy plays a crucial role in managing hemiparesis and supporting the restoration of motor function [19].

In this case, adherence to antiseizure therapy was suboptimal, as the medication was not taken consistently. Nevertheless, the patient demonstrated a favorable clinical response to treatment. The patient had also never undergone medical rehabilitation or physiotherapy.

Furthermore, following the most recent hospitalization, the patient did not return for follow-up evaluation, precluding further assessment of long-term clinical outcomes, including the progression of neurological function and the effectiveness of seizure control.

This case provides an important clinical lesson regarding the need to consider Dyke-Davidoff-Masson syndrome (DDMS) as a differential diagnosis in patients with chronic epilepsy, hemiparesis, and a history of delayed motor developmental milestones. In this patient, seizures began at 9 months of age and persisted into adulthood; however, the diagnosis of DDMS was established only at the age of 34 years through the integration of characteristic clinical findings and neuroimaging evidence on head computed tomography (CT). The substantial diagnostic delay observed in this case underscores the possibility that DDMS may remain undiagnosed despite the presence of suggestive clinical features since early childhood. Increased awareness of the characteristic clinical and radiological manifestations of DDMS may facilitate earlier recognition, prompt diagnosis, and more appropriate multidisciplinary management.

Recent adult case reports and reviews confirm that DDMS may remain unrecognized until adolescence or adulthood and can present with heterogeneous seizure patterns, motor deficits, and cognitive manifestations [20] [21] [22] [23] [24]. Contemporary imaging reports likewise emphasize the diagnostic value of correlating clinical history with unilateral cerebral volume loss, ipsilateral ventricular dilatation, calvarial thickening, and paranasal sinus hyperpneumatization on CT or MRI [25] [26] [27] [28] [29]. Maritska et al. also highlighted the occurrence of nervous-system comorbidities in children with neuro-developmental disorders and the importance of early detection by families and health professionals [30].

CONCLUSION

This case underscores the need to consider Dyke-Davidoff-Masson syndrome (DDMS) in the differential diagnosis of focal epilepsy, particularly in patients with early-onset seizures, hemiparesis, delayed motor developmental milestones, and characteristic neuroimaging abnormalities. The prolonged diagnostic delay of more than three decades observed in this patient illustrates that DDMS may remain undiagnosed despite the presence of suggestive clinical features since early life. On the basis of the clinical presentation and neuroimaging findings, the patient's epilepsy was classified as focal epilepsy with a structural etiology secondary to DDMS, presenting as a *focal impaired consciousness seizure with observable manifestations*. Increased awareness of the clinical and radiological hallmarks of DDMS may facilitate earlier diagnosis and support more appropriate management strategies.

ACKNOWLEDGEMENTS

The authors would like to thank the patient and her family for their cooperation and willingness to participate in the completion of this case report.

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