CASE REPORT

Clinical spectrum of Dyke-Davidoff-Masson syndrome in the adult: an atypical presentation and review of literature

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SUMMARY

Dyke-Davidoff-Masson Syndrome (DDMS) is a rare condition usually diagnosed in paediatric patients with clinical features of hemiparesis, seizures, mental retardation and contralateral cerebral hemiatrophy on neuroimaging. This report follows the case of a 22-year-old man presenting with seizures and hemiatrophy and hemiparesis. On review of cases the most common neuroimaging findings were cerebral hemiatrophy (100%) followed by hemicalvarial thickening (71.4%) and hyperpneumatisation of sinuses (71.4%). Apart from our patient, all nine cases with data on epilepsy control had drug-resistant epilepsy. The onset of seizures in adulthood, block vertebra, short stature, absence of mental retardation and well-controlled epilepsy on monotherapy makes our case exceptional—even bringing to mind the possibility of a DDMS variant. This report exhaustively reviews the wide range of clinical and radiological manifestations of DDMS in the adult, thereby adding to the literature on an unusual syndrome that causes significant neurological morbidity.

BACKGROUND

Initially reported in 1933 by Dyke *et al*,¹ the Dyke-Davidoff-Mason Syndrome (DDMS) is a rare neurological disorder usually diagnosed in the paediatric age group characterised by hemiparesis, facial asymmetry, seizures, mental retardation (MR) and associated cerebral hemiatrophy on neuroimaging.² Only 20 cases of the syndrome have been reported in the adult population. Of these, four are from one local case series.³ It is not surprising therefore that

the diagnosis for DDMS can be delayed to more than 50 years after the initial manifestations.⁴

This report follows the case of a 22-year-old Filipino male presenting with seizures, left-sided hemiatrophy and weakness.

CASE PRESENTATION

A 22-year-old man came to our clinic complaining of recurrent fits. His first episode occurred 3 months earlier when he had sudden involuntary extension and stiffening of his left upper extremity lasting 2 min. The following month, he had stiffening of the left upper extremity now followed by deviation of gaze to the left side and generalised tonic clonic movements of all extremities lasting for 3 min. Two more similar episodes occurred in the next 2 months with the latest one occurring a day before his consult.

Birth history showed a non-consanguineous union resulting in a full-term normal birth at home. Prior to giving birth, however, his mother had an unrecalled infection necessitating the use of unrecalled antibiotics. No perinatal complications were noted. He developed at par with age. Growing up, his parents noticed that his left side was smaller and weaker than his right. At the time of the initial consult, he had already finished a vocational course in computer programming and pursued further collegiate studies to earn a full degree in education.

On examination he was alert, oriented, following commands, with a perfect score of 30 in the minimal state examination. He had hemiatrophy, grade 4/5 weakness and hyper-reflexia all on the left side (see [figure 1](#)). Cranial nerve and sensory examinations were unremarkable. He had a height of 139.7 cm, falling below his expected height of 166.4 cm calculated from his midparental height.

He was prescribed carbamazepine 600 mg per day. Neuroimaging revealed hemiatrophy on the right cerebrum and brainstem with hemicalvarial thickening and associated polymicrogyria. His electroencephalogram was within normal limits. Spinal MRI revealed a block T7–T8 vertebra deemed to be congenital in aetiology (see [figure 2](#)). His clinical and neuroimaging features led to a diagnosis of DDMS. Bone ageing revealed mature bone structure. Endocrine hormones (thyroid stimulating hormone, free thyroxine, follicle stimulating hormone, leutinizing hormone, cortisol,

Figure 1 (A–C) Different views showing hemiatrophy of the patient's left side.

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Figure 2 (A–B) Axial T1-weighted MRI showing hemicalvarial thickening (white arrow) and hemiatrophy of right cerebral hemisphere. (C–E) Axial T2-weighted MRI showing volume loss on the right side of the brainstem. (F) Block vertebra (white arrowhead), T7–T8 vertebrae, with residual intervertebral disc material and exuberant spur formation abutting the thecal sac.

adrenocorticotrophic hormone) were all normal. Psychological evaluation that included the Wechsler Adult Intelligence scale, revised Slosson intelligence test and test of non-verbal intelligence revealed the absence of MR.

OUTCOME AND FOLLOW-UP

Three years after his initial consult, there is no progression of his initial deficits. His epilepsy is well controlled only having annual breakthrough seizures when he lacks sleep. He already managed to obtain his Bachelor's degree in education.

DISCUSSION

An early study on paediatric hemiplegia showed that imaging findings consistent with DDMS were only apparent in children with weakness manifesting prior to 3 years of age.⁵ Thus, pathologies such as cerebrovascular insults occurring during the gestational period up to 3 years of age would most likely have the highest propensity to result in DDMS. Prolonged ischaemia during this period can decrease the production of brain-derived neurotrophic factors, resulting in brain atrophy.⁶ Subsequently, structures such as the calvarium and the sinuses tend to grow inward to compensate.⁷

The 21 cases on DDMS in the adult are summarised in [tables 1 and 2](#). In contrast to a study done on a predominantly paediatric

population² that showed male sex dominance, 57.7% of cases were female. Only four cases (1, 6, 19 and 20) identified possible aetiologies for the syndrome—of these acquired cases, three are attributed to encephalitis. It is important to note that all four patients had associated epilepsy that began before 3 years of age. In contrast, both patients (18 and 21) with adult-onset seizures had no identified specific possible aetiology for DDMS. Perhaps, this is because the possible aetiologies of DDMS in the four patients, meningitis and premature birth, can cause cortical damage severe enough to create seizurogenic foci that in turn leads to an earlier onset of seizures compared with the 'idiopathic' group of DDMS.

Of those with seizures (76.1%), the onset of fits was during childhood except for our patient and 17. Peculiar presentations included hemianopia (Cases 1 and 2) and hemidystonia for case 5. It is also worthwhile to note that the series of Sia featured two brothers, (Cases 17 and 18) bringing to mind a possible genetic cause of the syndrome.³

MR is only present in 61.1% of the reviewed cases. Among patients with data on seizures and MR, it is worthwhile to note that all patients with seizures before 3 years of age (Cases 1, 2, 4, 6, 9 and 18) had MR while those with seizure onset after 3 years of age (Cases 5, 8, 11, 12, 13, 14, 15 and 21) only 2 (Cases 11 and 15) had MR. This suggests that patients with both epilepsy and MR have earlier onset of seizures

Table 1 Dyke-Davidoff-Masson syndrome: reported cases in adults

Author, patient age and sex	Seizure type and age of onset	Laterality	Mental retardation	Focal deficits	Neuroimaging modality and features	Additional information
1. Roy, <i>et al</i> ⁷ 42/F	Left partial motor with secondary generalisation 2.5 years old	Right	Present	(+) Left homonymous hemianopsia (+) Left spastic hemiparesis (+) Left extensor plantar response	MRI (+) Calvarial thickening (+) Hyperpneumatisation of sinuses and mastoid cells (+) Crossed cerebellar hemiatrophy	(+) Meningoencephalitis at 28 months. (+) Depression. (+) Several combinations of AEDs initially, improved later in life. <i>Management:</i> AED, muscle relaxant, physiotherapy.
2. Adebayo, <i>et al</i> ⁶ 27/F	Generalised tonic-clonic seizures 2 years old	Right	Present	(+) Left homonymous hemianopsia (+) Left spastic hemiparesis (+) Left extensor plantar response	MRI (+) Calvarial thickening (+) Hyperpneumatization of sinuses and mastoid cells	(+) Febrile seizure at 2 years of age. (+) Drug-resistant seizures. <i>EEG:</i> low amplitude background over the right hemisphere and bursts of generalised sharp-and-slow wave complexes. <i>Management:</i> AED (valproic acid), physiotherapy, cognitive therapy.
3. Sarikaya, <i>et al</i> ¹⁷ 36/F	None	Left	Present, mild	(+) Right hemiparesis	CT (+) Basal ganglia atrophy (+) Crossed cerebellar hemiatrophy (+) Calcifications in the lentiform nucleus	(+) Left frontal alopecia. (+) Associated left Internal carotid artery (ICA) occlusion on Computed tomography angiography (CTA) and Magnetic resonance angiography (MRA). (+) Poor control on phenytoin, phenobarbital, carbamazepine. <i>Management:</i> AED (valproic acid and topiramate).
4. Malik, <i>et al</i> ¹⁸ 18/F	Generalised tonic-clonic seizures 9 months	Left	Present, mild	(+) Right spastic hemiparesis (+) Right hyper-reflexia (+) Right extensor plantar response	CT (+) Calvarial thickening	(+) Drug-resistant seizure. <i>Management:</i> AED (valproic acid, topiramate), physiotherapy.
5. Arora, <i>et al</i> ¹⁹ 18/F	Seizures, unspecified 4 years old	Right	None	(+) Facial asymmetry (+) Left hyper-reflexia (+) Left hemidystonia	MRI (+) Calvarial thickening (+) Hyperpneumatisation of sinuses and mastoid cells (+) Elevated sphenoid and petrous ridge (+) Encephalomalacia	<i>Management:</i> multiple AEDs, physiotherapy.
6. Tasdemir, <i>et al</i> ²⁰ 28/F	Seizures, unspecified 1.5 years old	Right	Present	(+) Left hemiparesis	MRI (+) Crossed cerebellar hemiatrophy (+) Encephalomalacia	(+) Encephalitis at 1.5 years of age.

Continued

Table 1 Continued

Author, patient age and sex	Seizure type and age of onset	Laterality	Mental retardation	Focal deficits	Neuroimaging modality and features	Additional information
7. Tasdemir, <i>et al</i> ²⁰ 38/F	None	Left	Present	(+) Right hemiatrophy	MRI (+) Encephalomalacia	
8. Ayas, <i>et al</i> ²¹ 26/M	Complex partial seizures <i>10 years old</i>	Left	None	(+) Right hemiparesis (+) Right hemiatrophy (+) Facial asymmetry	MRI (+) Hyperpneumatisation of sinuses and mastoid cells (+) Brainstem atrophy	
9. Ayas, <i>et al</i> ²¹ 28/F	Generalised tonic-clonic seizures <i><1 year old</i>	Left	Present	(+) Right hemiparesis (+) Facial asymmetry	CT (+) Basal ganglia atrophy (+) Hyperpneumatisation of sinuses and mastoid cells	(+) Drug-resistant seizures.
10. Ayas, <i>et al</i> ²¹ 83/M	None	Right	None	(+) Left hemiparesis (+) Facial asymmetry	CT (+) Basal ganglia atrophy (+) Calvarial thickening (+) Hyperpneumatisation of sinuses and mastoid cells (+) Brainstem atrophy	(+) Presented with history of fall.
11. Ayas, <i>et al</i> ²¹ 44/M	Generalised tonic-clonic Seizures <i>7 years old</i>	Right	Present	(+) Left hemiparesis	CT (+) Basal ganglia atrophy (+) Hyperpneumatisation of sinuses and mastoid cells (+) Brainstem atrophy	
12. Ayas, <i>et al</i> ²¹ 31/M	Complex partial seizure <i>8 years</i>	Left	None	(+) Right hemiparesis (+) Facial asymmetry	MRI (+) Basal ganglia atrophy (+) Calvarial thickening (+) Hyperpneumatisation of sinuses and mastoid cells	(+) Drug-resistant seizures.
13. Ayas, <i>et al</i> ²¹ 46/F	Generalised tonic-clonic seizures <i>12 years old</i>	Left	None	(+) Right hemiparesis (+) Facial asymmetry	MRI (+) Basal ganglia atrophy (+) Calvarial thickening (+) Hyperpneumatisation of sinuses and mastoid cells	
14. Ayas, <i>et al</i> ²¹ 64/F	Generalised tonic-clonic Seizures <i>13 years old</i>	Right	None	(+) Left hemiparesis	CT (+) Calvarial thickening (+) Hyperpneumatisation of sinuses and mastoid cells	

Continued

Table 1 Continued

Author, patient age and sex	Seizure type and age of onset	Laterality	Mental retardation	Focal deficits	Neuroimaging modality and features	Additional information
15. Jain, <i>et al</i> ²² 30/F	Generalised tonic-clonic seizures 5 years old	Right	Present, mild	(+) Left hemiparesis (+) Left plantar extensor response	CT (+) Calvarial thickening (+) Hyperpneumatization of sinuses and mastoid cells (+) Crossed cerebellar hemiatrophy	(+) Drug-resistant seizures (phenytoin, phenobarbitone). EEG: Poorly organised background activity of bursts of generalised spike and wave discharged admixed with low and high voltage slow waveforms. Management: AED (valproic acid, levetiracetam).
16. Biçici <i>et al</i> ⁴ 54/M	Unknown	Left	Unknown	(+) Right hemiparesis (+) Right hyper-reflexia (+) Facial asymmetry	MRI (+) Calvarial thickening (+) Hyperpneumatization of sinuses and mastoid cells	
17. Sia and Astejada ³ 70/M Filipino	Generalised tonic-clonic seizures 29 years old	Left	Unknown	(+) Right hemiparesis (+) Right hemiatrophy	CT (+) Calvarial thickening (+) Hyperpneumatization of sinuses	(+) Brother with a similar condition (#18). EEG: diffuse slowing. Management: poor response to AED (phenytoin).
18. Sia and Astejada ³ 51/M	Generalised tonic-clonic seizures 5 months old	Left	Present	(+) Right spastic hemiparesis (+) Right hyper-reflexia	CT (+) Calvarial thickening (+) Hyperpneumatization of sinuses	(+) Brother with a similar condition (#17). EEG: mild diffuse left hemispheric dysfunction with multifocal and independent epileptiform discharges over the left temporal and right parietotemporal regions.
Filipino						
19. Sia and Astejada ³ 20/M	Seizures unspecified 3 years old	Right	Unknown	(+) Left hemiatrophy (+) Left spastic hemiparesis	CT (+) Calvarial thickening	(+) Premature birth at 8 months of gestation due to a motor vehicular crash. EEG: slowing of background activity more on the right with sharp wave discharges on both hemispheres.
Filipino						
20. Sia and Astejada ³ 39/F Filipina	None	Right	Present	(+) Left spastic hemiparesis	CT (+) Calvarial thickening (+) Hyperpneumatization of sinuses	(+) Meningitis at 1 year of age.
21. Our patient 25/M	Focal motor seizures evolving to generalised tonic-clonic seizures 22 years old	Right	None	(+) Left hemiatrophy (+) Left hemiparesis	MRI (+) Calvarial thickening (+) Crossed cerebellar hemiatrophy (+) Polymicrogyria, right	(+) Block vertebra. (+) Short stature. Seizures responsive to single AED. Management: AED (carbamazepine).

AED, antiepileptic drug; EEG, electroencephalogram.

Table 2 Summary characteristics of 21 cases of adult diagnosis of Dyke-Davidoff-Masson syndrome

Mean age at diagnosis (years)	39.0±18 (range: 18–83)
Sex: % Female	57.70%
Mean age of seizure onset (years)	6.7±8.2 (range: 0.52–29)
Laterality: % Right	52.40%

Unilateral weakness was a consistent finding. Imaging showed cerebral hemiatrophy in all—even involving the brainstem in five cases. (Cases 8, 9, 10, 11 and 21) Hemicalvarial thickening and hyperpneumatisation of sinuses with 15 patients each were the second most common neuroimaging findings. Polymicrogyria was only seen in our patient but a previous report of DDMS with the same feature has been reported in two cases in the paediatric population.^{8–9} In stark contrast to our patient, all other cases with data on epilepsy control showed patients on multiple antiepileptic drugs (AEDs) with difficult to control seizures. (Cases 1, 2, 3, 4, 5, 9, 12, 15, 17) All cases with data on therapy were managed with AEDs and physiotherapy only. Hemispherectomy for a paediatric DDMS patient with drug resistant epilepsy and hemiparesis resulted in seizure freedom.¹⁰ Despite the patient having adult onset seizures, it is important to emphasise that he already had features consistent with DDMS even in childhood, that of unilateral weakness and atrophy.

Seizures and hemiatrophy is consistent with Rasmussen's encephalitis; however, the lack of progression in the patient's deficits make this diagnosis unlikely.¹¹ The Russel-Silver syndrome with associated short stature and hemiatrophy of limbs is a close differential but the neuroimaging findings of our patient are not consistent with this condition.¹²

A new syndrome aptly named 'unilateral cerebral polymicrogyria with hemiatrophy' (UCPH) is the closest differential for the case accounting for most of our patient's symptoms and neuroimaging findings. The syndrome first came to light in a case series of 12 children with neuroimaging features for which the syndrome was named after.¹³ After a review of the 35 reported cases, three key points make UCPH a less likely diagnosis for our patient.^{13–16}

1. None of the reported cases of UCPH have hemicalvarial thickening—a neuroimaging feature present in our patient.
2. Unlike our patient, all the patients with UCPH had onset of seizures in the paediatric age group.
3. All the patients with UCPH with data on their intelligence were found to have some degree of MR; in contrast, our patient was found to have normal intelligence after testing with a battery of psychological evaluations.

Because of these, DDMS is still the most likely diagnosis for our patient.

This review of DDMS cases showcases the gamut of clinical and radiological findings that accompany this syndrome. Absence of MR in 61.1% of adult cases suggests that MR may not be a necessary criteria for the diagnosis of DDMS and that DDMS may be a spectrum with less severe cases recognised later in life during adulthood. The absence of MR, adult onset of seizures, block vertebra, short stature and epilepsy, controlled on monotherapy makes our case exceptional—even bringing to mind a possible variant of DDMS. With majority of patients in the review having drug-resistant epilepsy, epilepsy surgery may be an underutilised treatment option. This report on DDMS in the adult, adds to the literature on a rare neurological syndrome that can significantly impact the patients' quality of life.

Patient's perspective

In the vernacular (Filipino)

Epilepsy, nung una, hindi ako maniwala na magkakaroon ako nito. Wala naman kasi sa pamilya ko ang meron nito or nagkaroon nito. Ako lang. Nung nagkaroon ako marami akong natutunan. Paano dapat alagaan ang sarili at hindi dapat istress ang sarili. Siguro ang tanging tanong ko lang ay kung gagaling ba ako? Lifetime sickness daw kasi ang epilepsy. Natuto ako maging disiplinado at huwag abusuhin ang sarili.

Nung una ayoko inumin ang gamot ko sa epilepsy, na carbamazepine dahil nagkakaroon ako ng hilo. Hindi ako makatayo or makaupo ng maayos or kahit sa paglalakad. Pero nung naglaon okay na ako.

Nasanay na at patuloy na nag-iingat na wag ako atakihin ng sakit ko.

Translated (English)

Epilepsy, at first I could not believe that I was diagnosed with the disease. No one in our family has epilepsy—just me. I learned a lot having this disease—how to take care of myself and avoid stress. I would just like to ask if I will ever be cured of the disease? I learned that epilepsy can be a lifetime sickness. I learned how to be disciplined and to not overexert myself.

At first, I did not want to drink my medicine, carbamazepine because it caused vertigo. I couldn't sit, stand or walk properly. But eventually the dizziness disappeared and I felt better. I have gotten used to my disease. I take measures to ensure that my seizures do not recur.

Learning points

- The Dyke-Davidoff-Mason Syndrome (DDMS) can also present in adulthood with findings that are variations of the classically described clinical and neuroimaging findings of DDMS.
- Absence of MR in 61.1% of adult cases suggests that MR may not be a necessary criteria for the diagnosis of DDMS.
- The absence of MR, adult onset of seizures, block vertebra, short stature and epilepsy, controlled on monotherapy makes our case exceptional.
- Epilepsy surgery may be an underutilised treatment option for DDMS.

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