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Can Adhd Present In Dyke-Davidoff-Masson Syndrome? A Case Report

Dr. Gaurav Uppal[*1], Dr. Sonali Malik[2], Dr. Rohan Chauhan[3], Dr. Maryum Maryum[4], Dr. Betsy Babu[5], Dr. Sneh Bhabulkar[6], Dr. Manpreet Kaur[7], Dr. Vijayalakshmi V, Dr. Sathyan S, Dr. Sathyan S, Dr. Asha Dhandapani

[*1]Consultant Psychiatrist, Satyam Hospital, Ludhiana, India
[2]Consultant Radiologist, Supreme Scan, Ludhiana, India
[3]Junior Resident, Fortis Hospital, Mall Road Ludhiana, India
[4]Psychiatrist, Ireland
[5]Psychiatrist, United Kingdom
[6]Psychiatrist, United Kingdom
[7]Junior Resident Psychiatry , Government Medical College , Amritsar , India
[8]Consultant Neurologist , Usa
[9]Consultant Psychiatrist , Wales
[10]Psychiatrist , United Kingdom
[11]Consultant Psychiatrist, Wales

***Corresponding Author:**
Dr. Gaurav Uppal,
Consultant Psychiatrist, Satyam Hospital, Ludhiana
Email ID: drgauravuppal@gmail.com

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ABSTRACT

Background: Dyke-Davidoff-Masson Syndrome (DDMS) is an uncommon neurological disorder primarily characterized by unilateral cerebral hemiatrophy with compensatory cranial changes. Clinical manifestations typically include seizures, hemiparesis, and developmental delays. While various neuropsychiatric presentations have been sporadically reported, the occurrence of Attention-Deficit/Hyperactivity Disorder (ADHD) as a comorbidity remains undocumented.

Case Presentation: We describe a 4-year-old boy with a known diagnosis of DDMS since birth who subsequently developed generalized tonic-clonic seizures and, independently, began exhibiting cardinal features of ADHD—namely inattention, hyperactivity, and impulsivity. These symptoms persisted even after seizure control was achieved. Neuroimaging revealed the classic structural hallmarks of DDMS, including left-sided cerebral atrophy, ipsilateral ventricular dilatation, and a notable midline shift. Following multidisciplinary evaluation, the child was started on methylphenidate for ADHD, despite concerns regarding a lowered seizure threshold. This therapeutic approach led to significant behavioral improvement with no recurrence of seizures.

Conclusion: This case highlights a previously unreported association between DDMS and ADHD, underscoring the importance of broad, individualized management strategies in children with structural brain anomalies. Further research is warranted to clarify the shared neurobiological pathways and refine treatment options for this rare co-occurrence.

1. INTRODUCTION

Dyke-Davidoff-Masson Syndrome (DDMS), first described in 1933 by Dyke, Davidoff, and Masson, is characterized by unilateral cerebral hemiatrophy and compensatory changes such as thickening of the skull and enlargement of the paranasal sinuses on the affected side [1]. DDMS can be congenital, often attributed to intrauterine vascular insults, or acquired postnatally due to infections, trauma, or vascular compromise during critical periods of neurodevelopment [2,3].

Clinically, patients typically present with hemiparesis, seizures (often generalized tonic-clonic), and developmental delay [4–6]. Neuroimaging reveals atrophy of one cerebral hemisphere with ipsilateral ventricular dilation, sulcal prominence, and compensatory calvarial thickening [7,8]. Despite a robust literature detailing the structural and neurological manifestations of DDMS, the neuropsychiatric outcomes have not been systematically explored. Attention-Deficit/Hyperactivity Disorder (ADHD), a frequent neurodevelopmental disorder with an estimated prevalence of 5–10%, is characterized by inattention, hyperactivity, and impulsivity, potentially persisting into adulthood [9–11]. Dysregulation of cortico-striato-thalamo-cortical (CSTC) loops and dopaminergic pathways, particularly in prefrontal regions, basal ganglia, and cerebellum, is a key mechanism implicated in ADHD [12,13].

Interestingly, regions typically implicated in ADHD overlap with the structural abnormalities seen in DDMS, suggesting a possible mechanistic link [14,15]. Focal neuronal loss, Wallerian degeneration, and altered neuronal migration in DDMS may compromise CSTC circuits, potentially leading to ADHD-like symptoms [16,17]. Emerging data suggest that early brain insults—such as perinatal hypoxic-ischemic events—elevate the risk of neurodevelopmental disorders, including ADHD [18,19]. However, ADHD remains under-recognized in DDMS, often confounded by seizure activity and cognitive deficits that can obscure the primary features of ADHD [20,21].

Therapeutically, stimulant medications are first-line treatments for ADHD but can potentially lower the seizure threshold [22,23]. Non-stimulant alternatives (atomoxetine, guanfacine) are sometimes preferred in epileptic populations with poorly controlled seizures [24]. Nonetheless, consensus guidelines specific to DDMS are lacking, and individualized, multidisciplinary management remains the optimal approach [25–27]. This case report aims to highlight a rare presentation of ADHD in a child with DDMS and to discuss diagnostic and treatment considerations in this unique population.

2. CASE PRESENTATION

Perinatal and Neonatal History

A male infant was born at term via normal vaginal delivery to non-consanguineous parents. The prenatal course was unremarkable, with no documented intrauterine infections, maternal substance use, or traumatic injury. Birth weight was within the normal range, and Apgar scores were 8 and 9 at one and five minutes, respectively. However, within the first month of life, subtle neurological signs emerged: difficulty with feeding, increased irritability, and asymmetric limb usage. A cranial ultrasound at 4 weeks of life revealed significant left-sided cerebral atrophy, raising suspicion for DDMS.

Neuroimaging

Magnetic Resonance Imaging (MRI) provided a more detailed view, confirming left hemispheric atrophy with ipsilateral ventricular dilatation, sulcal prominence, and a 7-mm midline shift to the affected side. These classical radiological findings corroborated the diagnosis of DDMS.

Developmental History

Global developmental delays were noted across multiple domains. Neck control was achieved at around 6 months (expected at 3 months), and independent sitting was attained by 10 months (expected at 6 months). Expressive language was severely delayed; the child produced first intelligible words only by 2 years. Fine motor and social milestones were also lagging behind age norms, albeit slightly less profoundly than gross motor and language domains. A preference for right-hand usage was evident by 8 months, presumably compensating for weakness in the left upper limb. By 18 months, a mild gait asymmetry became apparent, though it did not severely impede ambulation.

Onset of Seizures

At 3 months of age, the child experienced the first generalized tonic-clonic seizure, lasting approximately 15 minutes and resolving spontaneously. Seizures recurred irregularly, escalating in frequency by 9 months to around two to three episodes per month. Initial treatment with phenobarbitone (2.5 mg/kg/day, later escalated to 5 mg/kg/day) offered only partial seizure control, with breakthrough episodes occurring every 6 weeks. By 18 months, after multidisciplinary consultation, phenobarbitone was discontinued, and oxcarbazepine was initiated. This medication achieved better control, reducing seizure frequency to approximately one episode every two months.

Emergence of Behavioral Changes

By 2.5 years of age, the child displayed escalating hyperactivity, restlessness, and a marked inability to focus on tasks consistent with his developmental level. Preschool teachers reported significant behavioral management challenges, describing frequent fidgeting, difficulty remaining seated, and suboptimal peer interactions. These behavioral symptoms intensified, prompting referral to child psychiatry at 4 years of age.

Psychiatric Evaluation

Comprehensive assessment included parental interviews, direct observation, and standardized rating scales. On the Conners' Parent Rating Scale, the child scored 97, indicative of moderate to severe ADHD symptomatology. Observations confirmed

inattention, high activity levels, and impulsivity across structured tasks. There was no evidence suggesting other contributory factors such as medication side effects, sleep disorders, or significant emotional distress from seizures. A diagnosis of ADHD, combined type, was established based on DSM-5-TR and ICD-10 criteria.

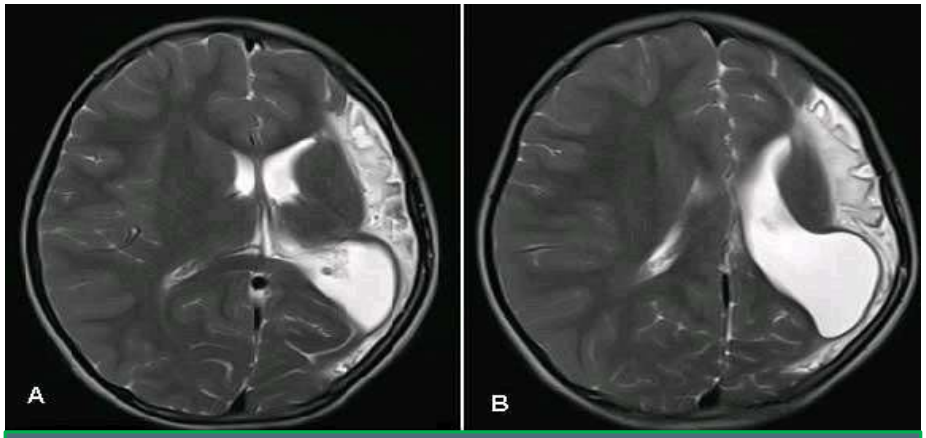

Figure 1 MRI T2W axial images show left cerebral hemiatrophy, dilatation of ipsilateral lateral ventricle and ipsilateral mid line shift of 7 mm

Cognitive and Psychological Assessment

Using the Wechsler Preschool and Primary Scale of Intelligence (WPPSI), the child's overall IQ was 68 (borderline intellectual functioning). Relative strengths were noted in non-verbal tasks, whereas verbal comprehension and working memory were significantly deficient. Screening for anxiety and depressive symptoms was unremarkable, although mild frustration-related tantrums were reported.

Treatment Initiation

Given the concurrent diagnoses of DDMS, seizure disorder, and ADHD, the treatment plan was carefully developed by a multidisciplinary team of pediatric neurologists, psychiatrists, and pediatricians. Methylphenidate (10 mg/day) was started at a low dose and gradually titrated, keeping the child on a stable dose of oxcarbazepine. Parents were counseled extensively regarding the risks and benefits of stimulant therapy in the context of an active seizure disorder.

Response to Treatment

Over the next two months, marked improvements were observed in hyperactivity, attention span, and overall behavioral regulation. By the fourth month, the Conners' score decreased to 57, suggesting moderate symptom severity, and parents estimated a ~30% improvement in day-to-day functioning. Crucially, no seizure exacerbations were documented during this period, implying that methylphenidate did not adversely influence seizure threshold in this case.

Long-Term Follow-Up

Continued follow-up revealed incremental gains with consistent medication use and supportive behavioral interventions. Parental management training and regular sessions with behavioral therapists contributed to sustained improvements. The child's multidisciplinary team continues to monitor seizure control, developmental progress, and emotional well-being, with periodic reassessment of the ADHD treatment regimen.

3. DISCUSSION

DDMS is notably rare, often presenting with unilateral cerebral hemiatrophy, hemiparesis, seizures, and developmental delay [28,29]. Although psychiatric manifestations—such as learning difficulties and behavioral disturbances—are documented, ADHD is exceedingly rare in this population. Our case demonstrates how DDMS-related structural and functional disruptions could intersect with neurobiological circuits implicated in ADHD.

Intersection of DDMS and ADHD

The characteristic atrophic changes and neuronal loss in DDMS can disrupt CSTC pathways vital for attention and executive control [7,30]. The child's left hemisphere involvement is particularly relevant, as left-sided lesions can impair language and executive functions, possibly potentiating ADHD-like symptoms [16,31,32]. Moreover, recurrent seizures might further

compound cognitive and behavioral deficits, emphasizing the need for detailed neuropsychological and psychiatric evaluations in DDMS.

Therapeutic Challenges

Managing ADHD in a child with an underlying seizure disorder is complex, given concerns about stimulants lowering the seizure threshold [22,23,33]. The success of methylphenidate in this case, without exacerbating seizures, highlights the importance of careful dosing and close monitoring. Oxcarbazepine's efficacy in controlling seizures may have provided an additional protective buffer. Nevertheless, treatment approaches must be individualized, considering alternatives like atomoxetine or alpha-2 agonists (guanfacine) if seizures are poorly controlled.

Neuroimaging Correlates

Advanced neuroimaging in DDMS reveals marked hemispheric atrophy, ventricular enlargement, and compensatory bone thickening. These structural changes offer insights into functional deficits and prognostication [7,8,34]. In this case, the significant left hemispheric atrophy is consistent with deficits in domains governed by left hemispheric function—language and certain aspects of executive control—that overlap substantially with ADHD symptomatology.

Multidisciplinary Management

Optimal care demands collaborative input from neurology, psychiatry, radiology, pediatrics, and developmental specialists [25,26,35]. This integrated approach ensures robust seizure control, accurate diagnosis of ADHD, and ongoing monitoring for medication side effects or breakthrough seizures. Parent education and psychosocial support are equally pivotal, fostering adherence and enhancing the child's developmental trajectory.

Implications and Recommendations

This unique presentation broadens the clinical spectrum of DDMS to include ADHD. It underscores the need for a high index of suspicion and systematic screening for neuropsychiatric comorbidities in children with structural brain abnormalities. Future prospective studies are warranted to elucidate prevalence rates, underlying pathophysiological mechanisms, and optimal treatment algorithms for ADHD in DDMS.

4. CONCLUSION

In summary, this report of a 4-year-old with DDMS, coexisting seizure disorder, and ADHD illustrates the intricate interplay between structural brain anomalies and neurodevelopmental disorders. The successful use of methylphenidate without seizure exacerbations emphasizes that stimulant therapy can be considered in specific clinical contexts, provided seizures are well-controlled and patients are carefully monitored. This finding paves the way for more detailed investigations into the neurobiological underpinnings of DDMS and the potential vulnerabilities that predispose affected children to psychiatric comorbidities like ADHD. Comprehensive, interdisciplinary management remains paramount to optimizing long-term outcomes.

5. SCOPE FOR THE FUTURE

Further research should focus on larger cohorts of DDMS patients to quantify the prevalence and spectrum of psychiatric comorbidities, especially ADHD. Advanced imaging methods such as diffusion tensor imaging (DTI) and functional connectivity analyses could shed light on how focal structural deficits disrupt widespread neural networks involved in attention and executive functioning. Additionally, well-designed clinical trials assessing the efficacy and safety of various ADHD pharmacotherapies—including newer stimulants and non-stimulants—in the context of seizure risk are needed to establish evidence-based guidelines.

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