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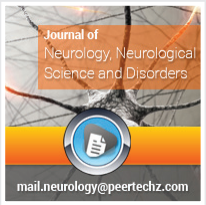

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Research Article

The Clinico-Radiological Spectrum of Dyke-Davidoff-Masson Syndrome in adults

Abstract

Background: Dyke-Davidoff-Masson syndrome (DDMS) is characterized by cerebral hemiatrophy, epileptic seizures, contralateral hemiplegia/hemiparesis, and mental retardation.

Aims: In this study, clinical and radiological investigations of seven patients who were diagnosed with DDMS as adults age were evaluated and discussed with the literature.

Study Design: The medical records of 7 patients diagnosed with DDMS at hospital were retrospectively and cross-sectionally examined.

Methods: Seven patients with DDMS diagnosed at adulthood between 2010 and 2016 were studied in our study. Patients' age, gender, etiological risk factors, presenting symptoms, lateralization, neurological examinations, computed tomography (CT) and / or magnetic resonance imaging (MRI) results were evaluated.

Results: Seven patients (4 male, 3 female) were included in our study. The mean age ± SD of the patients was 46±21 years. Clinical presentation of 6 patients was epileptic seizure. One patient was presented with head trauma due to a fall. Three of the patients were experiencing seizures for the first time, and 3 had a known history of epilepsy. Severe mental retardation was detected in 2 of the patients presenting with seizures. Two patients had complex partial seizures, 3 patients had generalized tonic clonic seizures (GTC), and 1 had GTC and myoclonic seizure. Mental retardation was mild in 3 patients and severe in 2 patients. A congenital cause was detected in 1 patient in the etiologic investigation. In 2 patients, a childhood of menengitis history was determined to be the acquired cause. In four patients, the etiology was not identified. We observed left hemisphere involvement in 4 patients and right hemisphere involvement in 3 patients. Brain imaging was performed by CT only in 4 patients and by MRI only in 3 patients. All patients were diagnosed with DDMS at adulthood. All patients had hemiatrophy in unilateral hemisphere. Atrophy in basal ganglia was detected in 5 patients, and atrophy in brain stem in 4 patients. Calvarial thickening was observed in 4 patients. Hyperpneumonitization was seen in mastoid cells in 3 patients. Sinus hyperpneamonitization, including in the paranasal and frontal sinuses, was seen in 6 patients.

Conclusion: DDMS is a rare syndrome diagnosed by clinical and radiological findings. Even it is diagnosed with epileptic seizure, hemiparesis, or mental retardation in childhood, it can also be diagnosed in adulthood symptomatically (mild-severe) or asymptomaticlly in adulthood with radiological findings, as it were in our study. As a result, DDMS is a syndrome with wide clinical and radiological spectra that can be variably symptomatic at different stages of life.

Introduction

Dyke-Davidoff-Masson syndrome (DDMS) is characterized by cerebral hemiatrophy, convulsions, facial asymmetry, controlateral hemiparesis/hemiplegia, and mental retardation. Radiological characteristics include the loss of unilateral cerebral parenchyma, compensatory changes in the calvarial bones on the same side (such as thickening), hyperpneumatization of the paranasal sinuses, elevation of the temporal bone, brainstem atrophy, and thalamic atrophy [1,2].

Diagnosis, which is important in terms of the approach to the disease and the treatment, is based on clinical findings, computerized tomography (CT), and magnetic resonance imaging (MRI). In this study, clinical and radiological

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investigations of seven patients who were diagnosed with DDMS as adults are evaluated and discussed.

Material and Methods

Seven patients diagnosed with DDMS were studied. Patients' age, gender, etiological risk factors, birth, childhood motor-mental developmental history, presenting symptoms, lateralization of hemiatrophy, neurological examinations, and CT and/or MRI results were evaluated. Descriptive statistics are used to describe the features of the data in this study.

Results

Seven patients (4 male, 3 female) were included in our study. The mean age ± SD of the patients was 46±21 years. (Table 1) shows the main characteristics of the patients.

Six of the patients presented with seizure. While one patient had epileptic seizures from birth, the starting ages of the other patients were 7, 8, 10, 12, and 13 years. The mean age of seizure onset was 7.1 years (range 0–13 years). Three of the patients were experiencing seizures for the first time, and 3 had a known history of epilepsy. Two patients had complex partial seizures, 3 patients had generalized tonic-clonic seizures (GTC), and 1 had GTC and myoclonic seizures. In 2 cases, the patients had refractory seizures. Severe mental retardation was detected in 2 of the patients presenting with seizures, while mild mental retardation was detected in 3 patients.

The seventh patient was an 83-year-old male with right-hemisphere involvement and no history of epileptic seizures who presented to the hospital with head trauma due to a fall. The asymptomatic patient was coincidentally diagnosed with DDMS.

Facial asymmetry was detected in 6 of the cases in accordance with cerebral hemiatrophy. Contralateral hemiparesis consistent with cerebral hemiatrophy was detected in all patients. Atrophy in the extremities was also detected in addition to hemiparesis in a 26-year-old patient. In 2 cases, we found that hemiparesis had started at under 2 years of age. In 3 patients, it had started after 2 years of age. Despite detailed anamnesis, clear information regarding the onset of hemiparesis in 2 patients could not be obtained. In one patient with severe mental retardation, a psychiatric disorder involving agitation and aggressive behavior was observed.

A congenital cause was detected in 1 patient in the etiologic investigation. In 2 patients, a childhood history of meningitis was determined to be the acquired cause, while in 4 patients, the etiology was not identified. We observed left-hemisphere involvement in 4 patients and right-hemisphere involvement in 3 patients.

Brain imaging was performed by CT only in 4 patients and by MRI only in 3 patients. All patients had hemiatrophy in unilateral hemisphere (Figure 1). Atrophy of the basal ganglia was detected in 5 patients and atrophy of the brain stem in 4 patients (Figure 2). Calvarial thickening was observed in 4 patients. Hyperpneumatization was seen in mastoid cells in 3 patients (Figure 3). Sinus hyperpneumatization, including in the paranasal and frontal sinuses, was seen in 6 patients (Table 2).

Discussion

DDMS was first reported by Dyke, Davidoff, and Masson in 1933 [3]. The major findings are unilateral cerebral atrophy, contralateral hemiparesis, and epilepsy. Because the symptoms are commonly seen in the early stages of life, DDMS is usually diagnosed in childhood.

In a study, 19 patients had a mean age of 6.8 years, and another study with 26 patients reported an average age of 11 years [2,4]. However, unlike those studies, which had large numbers of patients, in our study, all of the DDMS diagnoses were made at adult age (mean age 46±21). Generally the disease is diagnosed in childhood. There are single case reports in the literature for diagnosis in adulthood [5,6].

Functionally, males have more hemispheric asymmetry than females, and this may cause differences in neuronal connectivity [7, 8]. According to a hypothesis the presence of circulating androgens can create a hyperplastic state in a developing male's brain, which leads to wider neuronal remodeling after injury than in a female brain [9]. In our study, 4 of the 7 patients (57.1%) were male. Most DDMS reports in the literature are single case reports. However, in Unal et al.'s study [4], males were 73.5% of the DDMS patients. Similarly, in a study of 19 patients, 57.8% were male [2]; a study by Yorulmaz et al. [10] had 85% (9 of 13) male patients, and a study by Dix and Cail [11] had 52.2% (12 of 23) male patients.

In our study, 6 patients presented with seizure. Three of the patients were having seizures for the first time, and 3

Table 1: Main characteristic of patients.

Patient	Age	Gender	Laterality	Etiology	Clinical Findings	HP onset age	Seizure onset age	Type of epilepsy
1	26	M	L	Acquired	Seizure,HP	>2	10	CPS
2	28	F	L	Congenital	MR,epilepsy,HP	<2	0	GTC-myoclonic
3	83	M	R	Unidentified	HP	Unidentified	-	-
4	44	M	R	Unidentified	MR,epilepsy,HP	Unidentified	7	GTC
5	31	M	L	Unidentified	Seizure,HP	<2	8	CPS
6	46	F	L	Acquired	Seizure,HP	>2	12	GTC
7	64	F	R	Unidentified	Epilepsy,HP	>2	13	GTC

MR: Mental retardation HP: Hemiparesia GTC:Generalized tonic clonic seizure CPS: complex parsiel seizure

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patients had a known history of epilepsy. There were similar seizure symptoms in all 19 patients of another study [2], and a different study reported seizures in 17 of 26 patients [4]. Epileptic seizures with DDMS are more common in adolescence and are often resistant to treatment [12–14]. In our study, while one patient had epileptic seizures from birth, the starting ages for the other patients were 7, 8, 10, 12, and 13 years. The mean age of seizure onset was 7.1 years (range 0–13) years. In the literature, late-onset epilepsy cases have been described at 18 and 19 years of age [15, 16]. In a DDMS clinic, epileptic seizures similar to absence seizures were seen most frequently during adolescence, whereas there were no patients with absence seizures in our cases [13,14,17]. Two of our patients had complex partial seizures, 3 patients had GTC, and 1 had GTC and myoclonic seizures. Two patients had refractory seizures. A 31-year-old man and a 28-year-old woman with left-hemisphere involvement both had a history of refractory seizures. In the literature, DDMS patients with refractory seizures have been reported [18–20]. There was no epileptic seizure history in the 83-year-old patient with right-hemisphere involvement, similar to a patient not in an epilepsy clinic reported in the literature [21]. The 83-year-old patient who applied to the hospital with head trauma is thought to have been diagnosed with DDMS at an advanced age because of his lack of history of epilepsy and his minimal hemiparesis. Two of our patients had severe mental retardation, 3 patients had mild retardation, and 2 patients were mentally normal. In some cases, mental retardation and epileptic seizures may not occur even after many years [16]. Mental retardation was detected in 4 patients in one study and in 10 patients in another study [2, 4]. However, in these studies, because some of the patients were aged 1 year, no rate of mental retardation could be given.

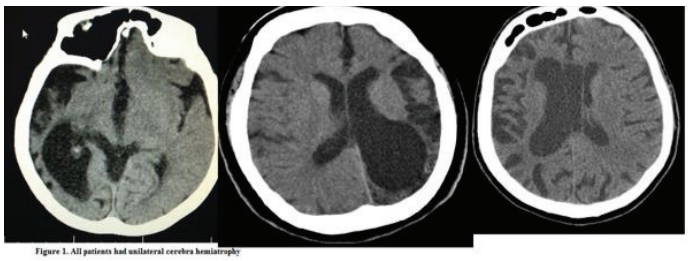

Figure 1: All patients had unilateral cerebra hemiatrophy.

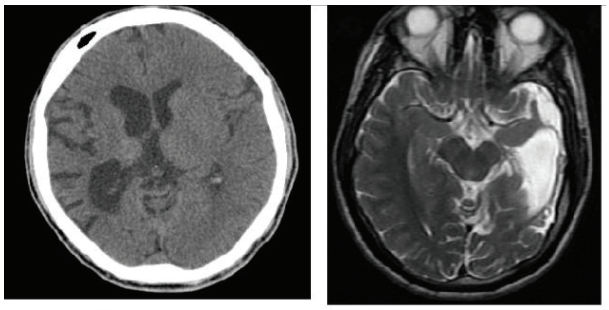

Figure 2: Brain images showed that atrophy in basal ganglia and brain stem.

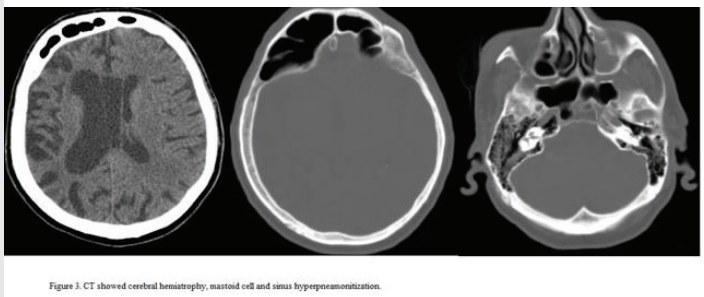

Figure 3: CT showed cerebral hemiatrophy, mastoid cell and sinus hyperpneamonitization.

In our study, facial asymmetry consistent with cerebral hemiatrophy was detected in 6 patients. In examinations of 26 patients in a different study, facial asymmetry was detected in only 8 patients [4]. In our study 44-year-old patient had no facial asymmetry consistent with cerebral hemiatrophy. Contralateral hemiparesis consistent with cerebral hemiatrophy was detected in all patients. In a study, hemiparesis/hemiplegia was reported in 11 of 19 patients [2], and another showed hemiparesis in 16 of 26 patients [4]. Atrophy was also detected in the extremities, in addition to hemiparesis, in a 26-year-old patient in our study. We learned that in 2 of our cases, hemiparesis started at under 2 years of age, and in 3 patients, it started after 2 years of age. Certain information regarding the onset of hemiparesis was not available in the detailed history of 2 patients aged 44 and 83 years.

DDMS has been reported to result from intrauterine conditions in which calvarium maturation has not yet been completed or if brain damage occurred in the first 3 years of life [22]. There are congenital and acquired etiologies. Congenital causes include congenital malformation, infection and vascular injury (occlusion in the middle cerebral artery),

Table 2: Radiological features of patients.

Patient	Radiologic modality		Hemiatrophy	Atrophy of basal ganglion	Atrophy of brain stem	Calvarial thickening	Hyper-pneumatization of mastoid cells	Hyper-pneumatization of sinuses
	CT	MRI						
1	-	+	+	-	+	-	+	+
2	+	-	+	+	+	-	+	-
3	+	-	+	+	+	+	+	+
4	+	-	+	+	+	-	_	+
5	-	+	+	+	-	+	-	+
6	_	+	+	+	-	+	-	+
7	+	-	+	_	-	+	-	+

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birth trauma, anoxia, hypoxia, and intracranial hemorrhage in the perinatal period. Stred et al. [23] reported a case of DDMS that developed secondary to a decrease in cerebral blood flow due to intrauterine midaortic coarctation. In congenital causes, symptoms start shortly after birth, due to damage during the intrauterine period. The fact of refractory epileptic seizure history from birth in our case with severe mental retardation, aged 28 years, suggests the presence of congenital causes in the etiology of the patient. In the literature, the patient was diagnosed with DDMS at the earliest 29 weeks of antenatal period [24]. Acquired causes are usually hemorrhagic or ischemic vascular diseases in the postnatal period. In addition, damage can be caused by trauma, tumors, and infection [25, 26]. The emergence of clinical symptoms in acquired DDMS can last into advanced childhood or adolescence, depending on the time and quality of the etiological factors [27]. In our study, a history of childhood meningitis in a 26-year-old male and a 46-year-old female suggests that the etiology was possibly acquired. In 4 patients, no cause was identified in the etiology. In a different study, the possible etiology of 12 cases was congenital in 19 patients, and there were acquired causes in 7 cases [2]. In our study, left hemisphere involvement was observed in four patients and right hemisphere was observed in three patients. Similarly in the studies, left hemisphere involvement was determined to be 69.2% [4]. In recent study showed left hemicrania in eight patients of twelve patients, and in another study, in which five patients were examined, hemiatrophy was reported in the left hemisphere in four patients and [28,29]. Some studies have suggested that the left hemisphere is more susceptible to cortical damage [30-32]. Brain blood flow in children between 1 and 3 years of age shows dominance in the right hemisphere [33]. Due to the high flow of blood in the right hemisphere, the left hemisphere is more susceptible to cerebrovascular events, especially in infants. Left hemisphere involvement in two our patients, particularly due to acquired reasons, supports this view. Brain asymmetry occurs in the prenatal period. So asymmetry in neurodegeneration implies a possible relationship between the development of cerebral lateralization and regional vulnerability in neurodegenerative disease [34].

Radiological characteristics of DDMS include unilateral cerebral parenchyma loss and compensatory changes, such as calvarial thickening, hyperpneumatization of the paranasal sinuses, and elevation of the temporal bone, and more rarely, brainstem and thalamus atrophy, in the calvarial bones on the same side [1,2,14]. Radiological findings may vary according to the duration and extent of cerebral injury [28]. In our study, CT neuroimaging only was performed on four patients. MRI only was performed on three patients. All patients exhibited hemiatrophy in the hemisphere. Calvarial thickening was observed in four patients. Compensatory skull changes reflect adaptation to atrophic brain tissue. In one study, calvarial thickening was detected in 11 of 19 patients and in 22 of 26 patients in another study [2,4]. Recent study reported that calvarial thickening and lateral ventricular enlargement were detected in all 12 patients [28]. In yet another study, it was determined that there was no relationship between parenchymal and calvarial changes and the initial findings and in the amount of morphological and pathological changes in the disease [14].

Shetty et al. [35], emphasized that compensatory changes in the homolateral head and sinus bones could develop due to the vacuum effect created by hypoplastic cerebral tissue in early life, especially in the first two years. Hyperpneumonitization was seen in mastoid cells in three patients. Sinus hyperpneamonitization, including the paranasal and frontal sinuses, was seen in six patients. In one study, enlargement of the paranasal sinuses was seen in 13 of 26 patients [4]. Atrophy in the basal ganglia was detected in five patients, and brain stem atrophy in four patients. In a series in which 28 cases were examined, 11 patients were found to have thalamic atrophy and lentiform nucleus hypoplasia [36]. Moreover, one case of hippocampal atrophy has also been reported [37]. Symmetric bilateral atrophy was reported [28]. Our patients showed no hippocampal atrophy. Congenital neurologic disease can be seen with extra neurological congenital anomalies. But our patient had no extra anomalies.

The delay in diagnosis in these patients may be due to not having all the symptoms in the patients (the classical triad of DDMS were found in two patients), the symptoms appearing at different times, or the physician does not mind the disease. Early diagnosis of DDMS provides appropriate treatment of seizures, physiotherapy for hemiparesia, and special education programs for mental retardation. If the diagnosis is delayed as in our patients, the success of the supportive treatment will be lower than early time.

DDMS is a rare syndrome diagnosed by clinical and radiological findings. Although it is diagnosed with epileptic seizure, hemiparesis, or mental retardation in childhood, it can also be diagnosed symptomatically (mild to severe) or asymptomatically in adulthood with radiological findings, as it was in our study. Therefore, DDMS is a syndrome with wide clinical and radiological spectra that can be variably symptomatic at different stages of life.

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