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Original Article

Etiological Factors of Opsoclonus Myoclonus Ataxia Syndrome: A Single Center Experience with Eight Children

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ABSTRACT

Objective: Opsoclonus myoclonus ataxia syndrome (OMAS) is a rare neurological disorder characterized by acute/subacute onset multi-directional chaotic eye movements, accompanied by myoclonus and cerebellar ataxia; as well as sleep disturbance, cognitive dysfunction, and behavioral disturbance can be observed.

Methods: We examined the information of eight patients (four females, four males) who applied to the hospital with OMAS between 2013 and 2020 from the medical records of the patients.

Results: The median age of onset of the initial symptoms was 17.5 months (8-30 months). The most common initial complaints were abnormal eye movement and gait unsteadiness, respectively. Paraneoplastic OMAS was observed in three patients (37.5%), whereas idiopathic and infection-related OMAS was detected in three, and two patients, respectively.

Conclusion: We emphasize that all symptoms of OMAS may not occur simultaneously, therefore comprehensive systemic investigations, and close observation should be made in patients with suspected OMAS.

Keywords: OMAS, opsoclonus, myoclonus, ataxia, neuroblastoma

INTRODUCTION

Opsoclonus myoclonus ataxia syndrome (OMAS), also known as dancing eye syndrome, is a rare neurological disorder, characterized by rapid, chaotic, and synchronous eye movements (opsoclonus), spontaneous muscle jerking (myoclonus), ataxia, and irritability.[1-3]

Ganglioneuroma/blastoma or neuroblastoma is detected in almost half of the pediatric OMAS patients; on the other side, the other half develops due to infections and idiopathic causes.[4,5] There is an autoinflammatory process in the background of this disease. Although no single pathogenic autoantibody has been identified in individuals with OMAS, increased B cell function and the presence of oligoclonal bands in cerebrospinal fluid (CSF)

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support the underlying autoimmunity and the importance of B cells in the pathophysiology of OMAS.[6,7]

Therefore, the recovery of neurological symptoms respond to immunotherapy. There are many treatment options as immunotherapy, including corticosteroids, adrenocorticotrophic hormone (ACTH), intravenous immunoglobulin (IVIG), cyclophosphamide, plasmapheresis, rituximab, and mycophenolate mofetil.[8-10]

In this report, we describe eight children with OMAS with different etiological factors and clinical presentations.

MATERIALS AND METHODS

Erciyes University Children's Hospital is a tertiary hospital in the city of Kayseri, in Central Anatolia, Turkey. This hospital is the sole pediatric referral center, serving a population of approximately 10 million, including the surrounding cities in the Cappadocia region. We reviewed the information of eight patients who applied to the hospital with the OMAS symptoms between 2013 and 2020 from the medical records retrospectively. Written informed consent was obtained from the parents of the included children. This study was approved by the Ethics Committee of Erciyes University, Faculty of Medicine (approval date: 06/01/2021, number: 2021/18).

RESULTS

We examined eight patients (4 females, 4 males) who presented with at least a symptom of OMAS (Table 1). The median age of onset of the initial symptoms was 17.5 months (8-30 months). The initial complaints of the patients were tremor, gait unsteadiness, myoclonic jerking, involuntary eye movements. The most common initial complaints were opsoclonus and gait unsteadiness (ataxia), respectively. Paraneoplastic OMAS was observed in three patients (37.5%), as idiopathic and infection-related OMAS was detected in the others. The median time from treatment to OMAS symptom remission was ten days (7-25 days) for seven patients. Opsoclonus symptoms continue in the high-risk neuroblastoma patient.

The first patient was admitted to the hospital at the age of 23 months with abnormal eye movements and gait unsteadiness. Although the neural structures were normal in the brain and spinal magnetic resonance images (MRI), a mass of 45x30x57 mm was detected in the posterior mediastinum (Figure 1). The tumor was totally resected, and he was diagnosed with ganglioneuroma. His complaints improved 14 days after the surgery and no additional treatment was required.

The second case was of a 16-month-old girl. She was admitted to the hospital with a complaint of involuntary eye movement. Neuron specific enolase (NSE) level, CSF examination, electroencephalography (EEG), brain and whole vertebral MRI, and abdominal ultrasonography (USG) were normal. She was followed up without any treatment because she had no additional complaints. When gait unsteadiness was noticed approximately eight months later, a solid tumor of 9.5x4.5x4 cm in size was detected in the left adrenal gland in the abdomen MRI (Figure 2). The tumor was completely resected by surgery, and its pathology was reported as a ganglioneuroblastoma. Since there were no tumor cells throughout the surgical margin and NSE was within normal limits, no needed additional chemotherapy. A single dose of IVIG was given only. Her complaints were improved ten days after the surgery.

At the age of 8 months of the patient three, a palpable mass was noticed in the abdomen. A paravertebral tumor and cranial metastases were detected on his MRI (Figure 3). The tumor was resected, and the pathology resulted in neuroblastoma. Metaiodobenzylguanidine scintigraphy revealed a left residual paravertebral mass, diffuse lymph node involvement in the abdomen, and metastasis in the brain. Therefore, she was considered a high-risk neuroblastoma according to the TPOG-NBL-2009 protocol. She was referred to our hospital for autologous harvest after receiving four courses of induction chemotherapy. The abnormal eye movement of the patient was noticed. Brain MRI was normal, no residual tumor was observed in abdominal MRI after four courses of chemotherapy. The opsoclonus was interpreted as a paraneoplastic. Autologous stem cell transplantation (ASCT) was performed, and the remaining chemotherapy was given. After the completion of planned chemotherapy, no residual tumors or metastasis were observed in the radiological imaging studies. The level of NSE level was within normal limits and the disease was considered complete remission. The complaint of opsoclonus did not ease. Therefore, IVIG was administered monthly for seven total doses for about a year, no change was observed. Currently, she is being followed up without any treatment for about two years.

Patients 4 and 5, one of them with gastroenteritis and the second one with upper respiratory tract infection were presented with abnormal eye movements. Any etiological factor could not be identified, but the viral infection was considered, symptoms resolved in about one week with the symptomatic treatments such as an antipyretic and empiric antibiotic. The etiology of OMAS could not be found in three patients all, only one patient received two doses of ACTH (75 U/kg/dose), and all patients' complaints improved in a short time.

NSE level, CSF examination, EEG, brain and whole vertebral MRI, abdominal USG, blood metabolic tests were performed for screening in all patients diagnosed with viral infection-associated or idiopathic OMAS. No etiological cause could be found. As the complaints of these patients regressed in a short time, no further evaluation was needed.

DISCUSSION

OMAS is a rare autoimmune neurological disorder with a median age of 18 months.[11] It is a clinical neurological syndrome characterized by opsoclonus, myoclonus, ataxia with or without behavioral abnormalities. This disease consists of involuntary eye movements, multifocal muscle jerks, and severe ataxia.[12] Opsoclonus is observed as involuntary eye movements in all directions, which could continue during asleep at 6-15 Hz. While

Table 1. The information of patients

Patients	Age at diagnosis (months), Gender	Initial symptoms	OMAS etiology	Abnormalities of EEG or MRI	First-line treatment	Time from treatment to OMAS symptom remission	Age at last follow-up, and neurologic status
Patient 1	23 mo, Male	Involuntary eye movements, Gait unsteadiness	Ganglioneuroma	EEG: Irregularity in ground rhythm, MRI: posterior mediastinal mass (45x30x57 mm)	Surgery	14 days	9 yo, Normal
Patient 2	24 mo, Female	Tremor, Gait unsteadiness, nystagmus	Ganglioneuroblastoma	MRI: Left surrenal mass (9.5x4.5x4 cm)	Surgery, IVIG (one dose)	10 days	8 yo, Normal
Patient 3	8 mo, Female	Involuntary eye movements	Neuroblastoma	MRI: paravertebral tumor and metastatic nasal cavity tumor	Surgery, ASCT, IVIG (7 doses)	Not resolved	3 yo, Opsoclonus continues
Patient 4	20 mo, Male	Involuntary eye movements	Viral infection	Normal	Observation, symptomatic, antibiotic treatment	9 days	6 yo, Normal
Patient 5	15 mo, Female	Involuntary eye movements	Viral infection	Normal	Observation, symptomatic treatment	7 days	5 yo, Normal
Patient 6	8 mo, Female	Abnormal eye movements	Idiopathic	Normal	Observation	10 days	4 yo, Normal
Patient 7	30 mo, Male	Gait unsteadiness, Myoclonic jerking	Idiopathic	Normal	ACTH (2 doses)	20 days	14 yo, Normal
Patient 8	13 mo, Male	Gait unsteadiness, Involuntary eye movements	Idiopathic	MRI: Myelination in bilateral periventricular white matter	Observation	25 days	5 yo, Normal

ACTH: Adrenocorticotrophic hormone, ASCT: Autologous stem cell transplantation, EEG: Electroencephalography, IVIG: Intravenous immunoglobulin, mo: Month-old, MRI: Magnetic resonance imaging, OMAS: Opsoclonus myoclonus ataxia syndrome, yo: Year-old

myoclonus is observed mainly in the form of irregular jerks in the body muscles, it makes children extremely difficult to walk with ataxic movements in the body and limbs. Diagnosis can be delayed since all classical features may not be presented initially. The median age of our patients' diagnosis was 17.5 months, and the most common complaints were opsoclonus and gait unsteadiness. Recently, neuropsychological disorders such as learning disability, language, and mental retardation in children with OMAS have been reported.[4] OMAS symptoms typically resolve with immunosuppressive therapy, although the recurrent course is common and long-term neuropsychological disorders persist in 80% of patients.[5,13]

Fortunately, only one patient still had neurological symptoms, probably because of the advanced stage neuroblastoma and late diagnosis. The rapid improvement of the neurological symptoms of the other two patients who have neuroblastic tumors can be attributed to the low-grade and the ability to cure with surgery alone. A series of six cases revealed that all neurological symptoms of patients diagnosed with OMAS due to neuroblastic tumor improved after treatment.[5] Another series of seven adult cases stated that the neurological symptoms of OMAS associated with coronavirus disease-2019 infection persisted despite the treatment in two patients.[14] Ben Achour et al.[12] reported that only three patients in a series of 15 children with OMAS still had neurological symptoms in about three-year follow-up. We think that our patients with non-paraneoplastic OMAS do not have permanent neurological symptoms due to their young age, early detection of symptoms and rapid response to treatment.

OMAS in children can be seen as a paraneoplastic syndrome and non-paraneoplastic syndrome (idiopathic, para-infectious). These

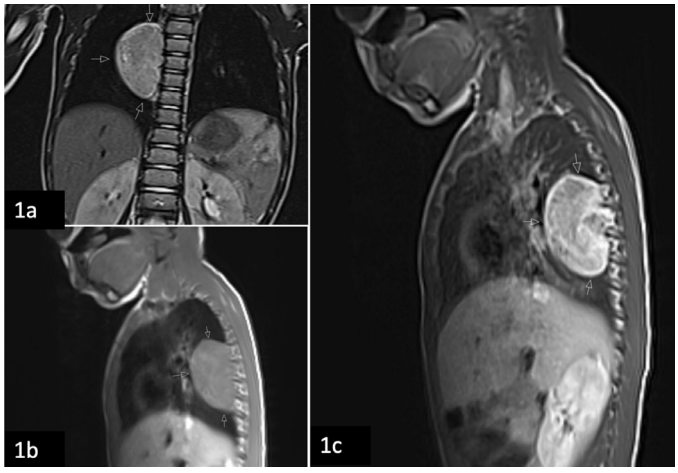

Figure 1. Posterior mediastinal right paravertebral ganglioglioma. (A) Coronal fat suppressed T2 weighted image and (B) sagittal T1 weighted image the tumor shows well defined margin and homogeny signal intensity, (C) post gadolinium T1W, sagittal plane: the tumor reveals relatively homogenous moderate enhancement pattern

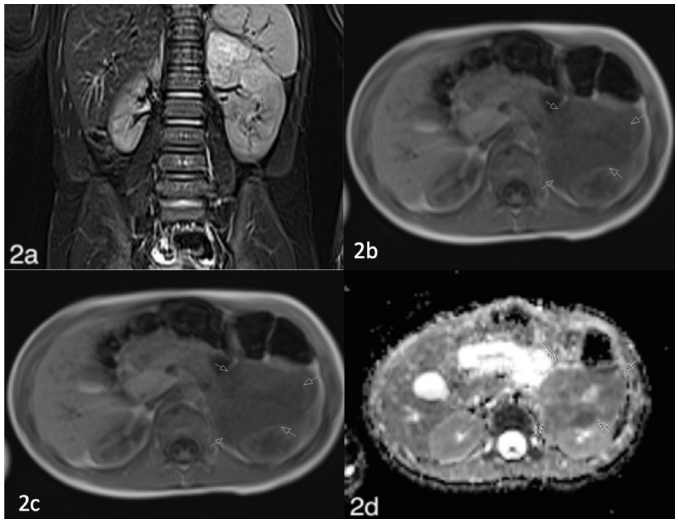

Figure 2. Patient 2, a 2-years-old female with left adrenal gland ganglioneuroblastoma. (A) coronal fat suppressed T2W hyperintense, (B) T1W hypointense homogenous signal intensity mass. The tumor infiltrating the left kidney, (C) diffusion-weighted image and (D) ADC map demonstrates restrictive diffusion (White arrows)

paraneoplastic and infectious associated opsoclonus/OMAS (IAO) are the most common causes in children; however, IAO is more dominant in adults. Neuroblastoma is seen as paraneoplastic in 50% of children, whereas small cell lung cancer and breast cancer are most common in adults. OMAS is accompanied by 1-2% of children with neuroblastoma. It is most seen in children under three years of age, the median age is 18 months.[13] The neuroblastoma that occurs with OMAS has an excellent oncologic outcome, is usually localized, insignificant, and differentiated. Complete

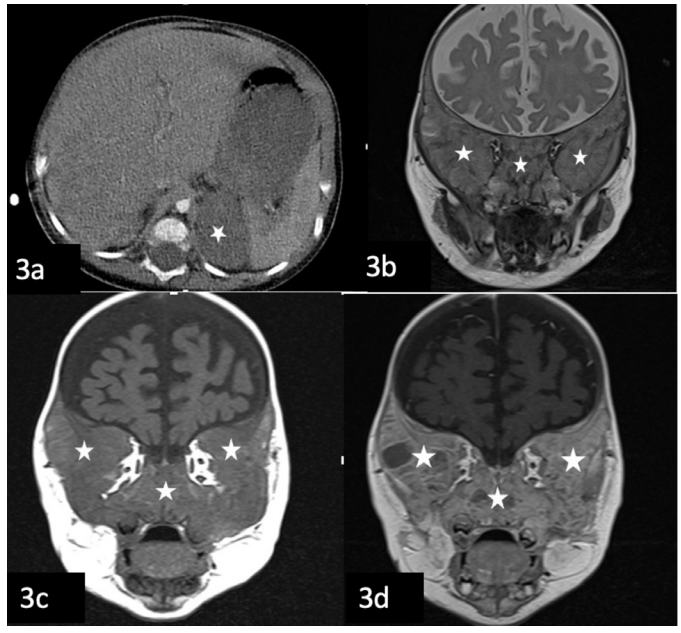

Figure 3. (A) Axial CT imaging shows smoothly contoured hypodense mass lesion in left surrenal gland localization. Coronal T2W (B) hyperintense, coronal T1W (C) hypointense and postcontrast images (D) show extensive metastatic bone lesions in the frontal bone, sphenoid bone, filling the nasal cavity, showing heterogeneous contrast enhancement

remission can be achieved only by surgery or sometimes with surgery plus standard chemotherapy. The association of high-risk neuroblastoma and OMAS is very rare.[15,16] Paraneoplastic OMAS developed in our three cases, the median age at diagnosis was 23 months. Although two of them had a tumor in the abdomen, the posterior mediastinal tumor was detected in the other. The two patients' diagnosis were ganglioneuroblastoma and ganglioneuroma, and complete remission could be achieved with only surgery. In our high-risk neuroblastoma patient, remission could be achieved with chemotherapy and ASCT in addition to surgery. But the neurological symptoms (opsoclonus) continues.

The second most common etiology of OMAS in children is infection. Viral infection is most common in IAO. Bacterial infections such as rickettsia, mycoplasma, salmonella, streptococcus, borreliosis, tuberculosis; fungal infections such as cryptococcosis and protozoal infections such as malaria were also reported to be the causes.[17,18] Among viruses, human immunodeficiency virus, influenza, mumps, herpes viruses, hepatitis C virus, arboviruses, and enteroviruses were identified.[19-21] In IAO patients, the agent could not be identified, but the symptoms of infection and OMAS improved with a symptomatic approach considering the viral infection.

Although the etiology of OMAS is still unknown; however, it is claimed as an underlying autoimmune mechanism. Considering the other family members of children who developed OMAS, it was observed that they are prone to autoimmunity compared to the control group.[22] Evidence shows that the abnormal immune-

mediated response targeting the central nervous system plays a vital role in the pathophysiology of OMAS, the cause of which is likely paraneoplastic or para-infectious.[6] Neuronal autoantibodies (anti-Hu, antineuronal, anti-neurofilament) were detected in the adults' serum and rarely in pediatric patients. Common surface-binding autoantibodies against the cerebellar structure and neuroblastoma cells have been identified.[23] One of them is Glutamate receptor δ2 autoantibody in the cerebellum.[24] Several inflammatory changes, such as increased IgG, IgM, and/or oligoclonal bands, may occur in the CSF of a patient with OMAS.

Furthermore, the demonstration of B cell activation in the CSF supports the autoimmune mechanism. In childhood OMAS, especially B-cell activating factor (BAFF) increase is observed. These levels decrease with immunotherapeutic treatment, such as steroids and ACTH.[6,7,16]

Because of the immune mechanism, immunomodulatory treatment, especially corticosteroid, has been the cornerstone of OMAS therapy. In the last 30 years, treatment regimens consisted of corticosteroids and ACTH. Many symptoms improved with this treatment regimen, but OMAS in children tend to continue for a long time with recurrence and behavioral disorders. Therefore, steroid therapy should be continued for a long time and may have serious side effects. Different treatment protocols have been used to prevent long-term use of steroids and to avoid side effects. In addition to this classical treatment, IVIG, rituximab, therapeutic plasma exchange, chemotherapeutic drugs such as methotrexate and cyclophosphamide have used.[2,25,26] Furthermore, the purpose of OMAS treatment is not only to reduce neurological symptoms but also to improve learning and behavioral skills. ACTH and steroids effectively reduce neurological symptoms in OMAS, whereas they are not sufficient in neuropsychological recovery and prevent relapse. IVIG is good at reducing neurological symptoms with a different mechanism of action, but it is insufficient to improve neuropsychological symptoms. Therefore, recent studies suggest that aggressive multiple aggressive therapies can improve outcomes in neuropsychological symptoms of OMAS.[27] In a study, the combination of pulse steroid, s IVIG, rituximab, and/or therapeutic plasma exchange was effective in both neurological and neuropsychological healing.[24,28,29] In other studies, immunotherapy with ACTH instead of steroids, combined with IVIG and rituximab has been found effective in terms of benefits and side effects.[30,31] Furthermore, cyclophosphamide added treatments reduced B-cell activity in CSF.[32] ASCT is increasingly used to treat autoimmune diseases. Transplantation has been reported in autoimmune diseases with neuroinflammatory factors such as chronic demyelinating polyneuropathy, multiple sclerosis, and myasthenia gravis. ASCT in autoimmune diseases removes pathogenic autoreactive cells and replace them with de novo repertoire immune cells.[33] We used two doses of ACTH in a patient with idiopathic OMAS, and the symptoms improved without the need for long-term use. Although the disease is in remission with surgery and ASCT in our patient diagnosed with high-risk neuroblastoma, neurological symptoms continue.

Study Limitations

Our study also has limitations. Firstly, no functional studies were performed in the included patients. Secondly, our patient cohort size was small. Larger cohorts will be needed to reveal the clinical spectrum of OMAS.

CONCLUSION

Neuroblastic tumors should be considered in all cases of OMAS in children. Early diagnosis and treatment of underlying diseases are essential to reduce complications and increase the chance of neurological recovery. We would also like to emphasize that all OMAS symptoms with different etiological factors may not occur at the same time; therefore, detailed evaluation and close observation should be made in patients with suspected OMAS.

Ethics

Ethics Committee Approval: This study was approved by the Ethics Committee of Erciyes University Faculty of Medicine (approval date 06/01/2021, number 2021/18).

Informed Consent: Written informed consent was obtained from the parents of the included children.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: V.G., H.S.D., H.G., M.K., A.K.B., H.P., Concept: V.G., E.Ü., H.P., Design: V.G., G.G., H.P., Data Collection or Processing: V.G., H.S.D., A.K.B., Analysis or Interpretation: V.G., E.Ü., H.G., H.P., Literature Search: V.G., M.K., Writing: V.G., A.K.B., H.P.

Conflict of Interest: The authors declare that they have no conflict of interest

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