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CLINICAL PROFILE OF ACUTE CHILDHOOD ATAXIA IN A COHORT OF IRAQI CHILDREN

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ABSTRACT

Background: Acute Childhood ataxia is a common indication for pediatric emergency room admission. Ataxia is the primary clinical sign of cerebellar illness, is the inability to coordinate movement that is not brought on by weakness, involuntary movements, or abnormal muscle tone. There is anatomical localizing value for each of the three forms of ataxia caused by cerebellar disease: limb, truncal, and gait. Lesions of the vermis or brainstem cerebellar connections cause gait and truncal ataxia, while lesions of the ipsilateral cerebellar hemisphere cause limb ataxia. **Objectives:** Is to find out the clinical features, causes, investigation, and management of acute childhood ataxia under the age of 15 years. **Methods:** A prospective study was performed at Ibn Sena Teaching Hospital from the 1st of January to the 31st of December 2024. Acute ataxia in children under the age of 15 years is found by the full neurological history and examination. The questionnaire was consisted from three parts. Part one for demographic information of the study patients. Part two for clinical manifestation of the study participants. Part three for the investigation done for each patient. **Results:** The study included 128 patients, preschool age was the predominant age groups, male/ female ratio was 1.13:1. The study found that fever was present in 54 (42.1 %) patients, previous viral infection within 2-3 weeks in 97 (61.7%) patinets, and recent varicella infection 20 (15.6%) patients. Moreover; Ataxia was present in 128 (100%) patients, followed by dysdiadochinesia in 62 (48.4%) patients, intention tremor in (43.7%) patients, scanned speech in 48 (37.5%) patients, nystagmus in 46 (35.9%) patients. Investigation were done as lumber puncture in 83 (64.8%) patients with abnormal result in 49 (59.1%) patients. MRI done in 103 (80.4%) patients with abnormal result in 46 (44.6%) patients, EEG done for 25 (19.5%) patients with abnormal finding in 16 (64%) patinets, EMG/NCS were requested in 13 (10.1%) patients which are all positive. Additionally; toxicology screen, drug level requested accordingly if available. Acute cerebellar ataxia was found the most common cause among 51 (39.8%) patients, central nervous system infections (meningitis/encephalitis) among 22 (17.1%) patients, drugs and toxins among 14 (10.9%) patients, Gullian Barre syndrome (GBS) among 13 (10.1%) patients, acute disseminated encephalomyelitis (ADEM) among 7 (5.4%) patients, epilepsy among 7 (5.4%) patients, brain tumors among 4 (3.1%) patients, trauma among 4 (3.1%) patients, posterior fossa abscess, psychological and Migraine each of them 2 (1.5%) patients. **Conclusion:** There are variety of causes of acute ataxia in children, putting in mind that there is many lives threatening condition can cause acute ataxia. The highest incidence of acute ataxia was observed in preschool age children. Males were found to be affected more than females. Every case with acute ataxia should be evaluated thoroughly starting with history, physical and neurological examination with appropriate laboratory and radiological investigations. Additionally; every case of acute ataxia should be admitted to hospital to exclude life threatening conditions for early detections of this conditions and starting appropriate treatment accordingly.

KEYWORDS: Acute, Cerebellar ataxia, Iraq, Mosul, Pediatric.

1. INTRODUCTION

Acute Childhood ataxia is a common indication for pediatric emergency room admission.[1] Ataxia refers to unsteadiness of walking or slight incoordination that is generally noticeable while moving or maintaining a sitting position in a previously healthy child with a symptom evolution time of less than 72 hours.[2] Drug intake and acute post-infectious cerebellar ataxia are the two most prevalent causes of acute ataxia in previously healthy children who develop an ataxic gait

unexpectedly.[3] To diagnose acute ataxia in children, the first step is to rule out central nervous system infections and mass lesions (e.g. tumors, abscesses).[4] Diagnosing childhood ataxia involves assessing the temporal course and neurological abnormalities.[5] The etiology has a significant role in the outcome of all types of childhood ataxia.[6] Management of pediatric patients with acquired non-progressive ataxia who presented with acute onset ataxia is include whole history, clinical neurological examination, investigation, treatment, and follow-up.[7]

Ataxia is the primary clinical sign of cerebellar illness, is the inability to coordinate movement that is not brought on by weakness, involuntary movements, or abnormal muscle tone.[8] When afferent input into the cerebellum through the spinocerebellar pathways is compromised, sensory ataxia occurs.[9] There is anatomical localizing value for each of the three forms of ataxia caused by cerebellar disease: limb, truncal, and gait. Lesions of the vermis or brainstem cerebellar connections cause gait and truncal ataxia, while lesions of the ipsilateral cerebellar hemisphere cause limb ataxia.[10] Motor coordination problems can include clumsiness, poor balance, uneven movement, and difficulty in performing fine or smooth movements.[11] Rapid onset within 24 hours might cause inability to walk in younger children, speech abnormalities, and loss of fine motor skills in older children.[12] Typically progress to marked gait abnormality with no motor modality spared.[13]

The aim of this study was to find out the clinical features, causes, investigation, and management of acute childhood ataxia under the age of 15 years.

2. PATIENTS AND METHODS

A prospective study was performed at Ibn Sena Teaching Hospital from the 1st of January to the 31st of December 2024. The studied patients were under 15 years of age, and were collected from hospitalized children in pediatric ward, emergency room and out patient's clinic. All previously healthy children who developed acute unsteadiness over 72 hours or less were recruited. Data was collected through a specially designed questioners including history information which were collected directly from parents, followed by full clinical and neurological examination. The data collected were include demographic information. Moreover; patients' duration of symptoms, preceding history of upper respiratory tract infection or gastroenteritis, or history of similar episodes. Furthermore; family history of same condition, history of fever previously or during episodes, recent vaccination, drug history, history of epilepsy, varicella infection (Chicken pox), trauma, vomiting and any associated conditions. Clinical examination was done searching for; alertness and disturbance level of consciousness, speech volume, scanned, dysarthria, cranial nerve involvement, meningeal signs, motor system examinations, gait abnormality, cerebellar sign (Nystagmus, Dysdiadochinesia, Intention Tremor, Finger nose test, Heel shin test (older patients), truncal ataxia, Titubation, Dysmetria).

128 cases were enrolled in this study, with different age and gender. According to each case scenario investigation were done: LP aspirations especially those who had fever, meningeal signs, disturb level of consciousness or any suspicion of CNS infection, aspiration of 2 ml CSF fluid for (1- protein: which is abnormal if its more than 40 mg/dl, 2- WBC cells: abnormal if its more than 5 / mm^3 lymphocyte, PMN is abnormal, 3- Gram stain, 4- culture and sensitivity). Magnetic resonance imaging MRI of brain and spine with or without contrast were requested for most of the cases and the report of the radiologist will confirm the diagnosis, CT scan of the head in some cases with history of trauma, EMG/NCS are requested to those cases with acute unsteadiness who progress to ascending paralysis and suspicion of GBS which is done by neurophysiologist, EEG were requested to those cases with epilepsy, Toxicology screening for blood and urine were requested for those cases with suspicion of drug poisoning , Drug level , for those on AED , if available. All cases had basic investigation as CBC, ESR, GUE, LFT, RFT, RBS, CXR if there is suspicion of chest infection.

The study inclusion criteria are: 1- Acute ataxia of less than 72 hours told by parents or found by examinations. 2- Absence of known genetic predisposing factors such as familial neuro degenerative disorders. 3- No family history of metabolic disease. The collected data was analyzed and adjusted using the SPSS version 30 statistical program. P-value were used to show if there is any significant difference between results.

3. RESULTS

The number of children with acute ataxia recruited during the study period was 128 cases. The most common age is preschooler (4-6 years) which founded to be 73 cases (57%), followed by school age child (7-12 years) founded in 29 cases (22.6%), then adolescent age (13-15 years) founded in 15 cases (11.7%), and lastly toddlers age (1-3 years) founded in 11 cases (8.5%). With a median age (5.1 years), minimum age (2.5 years) and maximum age was (13.6 years). As showed in table 3.1:

Table 3.1: Distribution of the study patients according to their age.

Age groups	Number of cases	Percentage %
1 - 3 year	11	8.5 %
4 - 6 year	73	57 %
7 - 12 year	29	22.6 %
13 - 15 year	15	11.7 %
Total	128	100 %

This study included 67 (52.3%) boys, and 61 (47.6%) girls. With a male to female ratio (1.1:1) as shown in figure 3.1 below:

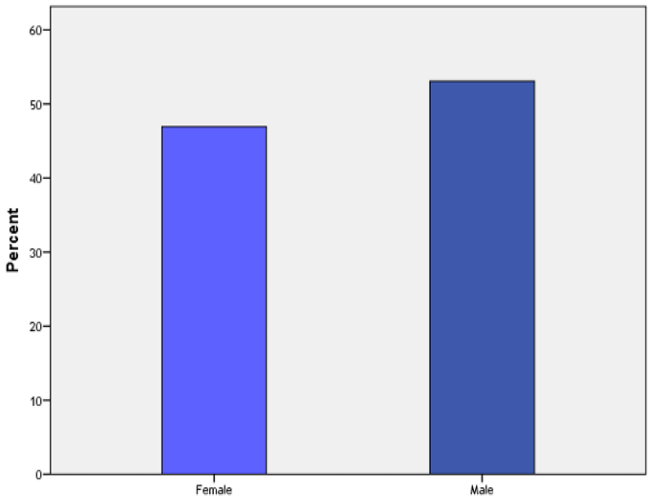

Figure 3.1: Gender distribution of the study participants.

Table 3.2 shows comparison between males and females regarding their ages. It's evident that there is a statistically significant differenc between the two groups (P value = 0.049) regarding this issue.

Table 3.2: Comparison between the males and females regarding their ages.

Age groups	Total number	Total Percentage %	Gender	
			Male No= 69	Female No = 61
1- 3 year	11	8.5 %	7	4
			63.6%	36.4%
4 - 6 year	73	57 %	36	37
			49.3%	50.6%
7 - 12 year	29	22.6%	13	16
			44.8%	55.2%
13 - 15 year	15	11.7%	11	4
			73.3%	26.7%

Table 3.3 shows that vomiting founded in 51 cases (39.8%), Fever during illness was 54 cases (42.1%), history of recent vaccination 4 cases (3.1%) before the illness within 2-3 weeks, history of trauma founded 4 cases (3.1%) which founded as the cause of acute ataxia and transferring to neurosurgical department, history of previous viral infection within illness or previous 2 weeks are 97 cases (75.7%), Recent varicella infection as chicken pox exanthema was reported in 20 cases (15.6%) during the illness or before 2 weeks.

Table 3.3: Historical features in children with acute ataxia.

History finding	Number of cases	Percentage %
Vomiting	51	39.8 %
Fever	54	42.1 %
History of recent vaccination	4	3.1 %
History of recent Trauma	4	3.1 %
Previous viral infection	97	75.7 %
Recent varicella infection	20	15.6 %

The drugs and toxins which were found 7 cases with Carbamazepine (3 cases founded with high blood level), two cases had with phenobarbital (with urine and serum level were high), one case with Diazepam (serum and urine level was high), one case with Clonazepam. One case founded with high urine level of Diazepam and Phenobarbital toxicology screen. 2 cases developed symptoms after administration of antihistamine drugs for upper respiratory infection and the symptom disappear after discontinue the medication. One case who was addicted to alcohol and Diazepam tablets develops acute Ataxia after stressful conditions happen to him in the street.

About the signs which was founded by clinical neurological examination all of 128 cases had acute ataxic gait (100%), followed by dysdiadochinesia founded in 62 cases (48.4%), intention tremor founded in 56 cases (43.7%), hypotonic 50 cases (39%), scanned speech 48 cases (37.5%), nystagmus 46 cases (36.7%), reflexes affected in 45 cases (35.1%), dysmetria 41 cases (32%), meningeal signs founded in 38 cases (29.6%), Romberg sign 32 cases (25%), Heel shin test 28 cases (21.8%), disturbed consciousness 26 cases (20.3%), truncal ataxia 17 cases (13.2%), titubation 8 cases (6.2%). hospital admission occurred to 113 cases (88.2%) most of the cases was admitted to hospital for 1-2 weeks, with a medium range 12.6 days, follow up was done within 2 weeks after discharge for 52 cases (40.6%). As shown in table 3.4:

Table 3.4: Frequency of clinical signs in a child with acute ataxia.

Findings	Number of cases	Percentage %
Ataxic gait	130	100
Dysdiadochinesia	62	48.4
Intention tremor	56	43.7
Hypotonia	50	39
Scanned speech	48	37.5
Nystagmus	46	36.7
Reflexes	45	35.1
Dysmetria	41	32
Meningeal signs	38	29.6
Romberg sign	32	25
Heel shin test	28	21.8
Disturbed consciousness	26	20.3
Trunckal ataxia	17	13.2
Titubation	8	6.2

About investigation which was requested to the cases by physicians: Lumber puncture (CSF aspiration) done to 83 cases (64.8%), founding that (49 / 83 cases with abnormal results (59.1%). Magnetic resonance imaging (MRI) was done to 103 cases (80.4%), which is seen and interpreted by radiologist. Abnormal results and finding in 46 cases (44.6%) as following: Acute disseminated encephalomyelitis ADEM 7 cases (6.7%), brain abscess (posterior fossa) 2 cases (1.9%), brain tumor (posterior fossa) 4 cases (3.8%), focal lesions (sclerosis) 5 cases (4.8%), brain atrophy 13 cases (12.6%), tubercular meningitis 3 cases (3.8%), meningitis/encephalitis 9 cases (8.7%), bilateral thalamic lesions 3 cases (3.8%). Electromyogram / nerve conduction study (EMG / NCS) was requested to the child who presents with acute ascending flaccid paralysis especially lower limb with areflexia, 13 cases (10.1%) which revealed a diagnosis of GBS (Gullain Barre syndrome), this test were done by an expert neurophysiologist. Electroencephalography (EEG) done for 25 cases (19.5 % of total cases) which is interpreted by an expert neurophysiologist, revealed normal results in 9 cases (36 % of 25 cases who had EEG), generalized epilepsy in 8 cases (32 %), focal epilepsy in 8 cases (32 %). As shown in table 3.5.

Table 3.5: MRI finding which found in children with acute ataxia.

Investigation	NUMBER	%
MRI	103	80.4
Normal	57	55.4
Brain atrophy	13	12.6
Meningitis / encephalitis	9	8.7
ADEM	7	6.7
Focal cerebellar lesion	5	4.8
Brain tumor Posterior fossa	4	3.8
Bilateral thalamic lesions	3	2.9
Tubercular meningitis	3	2.9
Abscess (posterior fossa)	2	1.9

After complete neurological history and full examination of all cases, founded that the most common cause of acute ataxia in children is: acute cerebellar ataxia 51 cases (39.8%), central nervous system infections (meningitis/encephalitis) 22 cases (17.1 %), toxins and drugs 14 cases (10.9 %), Gullain Barre syndrom (GBS) 13 cases (10.1%), epilepsy 7 cases (5.4%), acute disseminated encephalomyelitis (ADEM) 7 cases (5.4%), brain tumor (posterior fossa) 4 cases (3.1 %), trauma 4 cases (3.1%). Moreover; psychological, migraine, posterior fossa abscess founded among 2 cases (1.5 %) for each of them respectively. These finding above are shown in table 3.6 below:

Table 3.6: Causes of ataxia among the study participants.

Causes	Number	%
Acute cerebellar ataxia ACA	51	39.8
Meningitis / Encephalitis	22	17.1
Toxins and drugs	14	10.9
Gullain Barre syndrome GBS	13	10.1
Acute disseminated encephalomyelitis ADEM	7	5.4
Epilepsy	7	5.4
Trauma	4	3.1
Brain tumor	4	3.1
Abscess	2	1.5
Migraine	2	1.5
Psychological	2	1.5
Total	128	100

4. DISCUSSION

Regarding the age of the acute ataxia cases, it was founded that the mean age for all cases was (5.1 years), this is going with Mahboube Zarei et al.[14] and Pavone et al.[15] Moreover; the study founded that ataxic males were more than ataxic female (1.1 /1), this is in agreement with Min Zhang et al[16] and Lancella et al et al.[17]

The study showed that all of the patients with ataxia were presented ataxic gait. Which is similar to Amanda Miranda Brito Araújo et al study findings[7] and Jeesuk et al[18] Additionally; the study showed that antecedent infection was positive in 97 cases (75.7 %), which is in agreement with Müge Baykan et al.[19] Other clinical features which were found in this study as; dysdiadochinesia, intention tremor, hypotonic, scanned speech, nystagmus, reflexes affected, dysmetria, meningeal signs, Romberg sign, Heel shin test, disturbed consciousness, truncal ataxia, and titubation, were present in variable percentages, which closed to what was found by Giacomo Garone et al[20] and Joanna C. Jen et al.[21] studies' findings. All these signs and symptoms depend on the age of the child, duration of symptoms and examination, cooperation of the child, experience of the examiner, and the cause of ataxia.

On the other hand; cerebrospinal fluid examination found abnormal in (59.1%) of children with ataxia, which higher than what is found by Harry T. Whelan et al (43%)[22], Anyhow; different study setting and sample size can lead to this difference. While MRI shown in this study that abnormal result was found in (44.6%) of the

study participants and acute disseminated encephalomyelitis was the most prevalent cause for ataxia among the study participants, which is consistent with Mahboube Zarei et al study's findings (41.9%).[23] But EEG was abnormal in (64%) of the study participants who did EEG, which is close to what was found by Mohsen Javazadeh et al. (28.6%).[24]

The study found that the most common causes for ataxia in children were; acute cerebellar ataxia (39.8 %), central nervous system infections (17.1 %) and toxins and drugs (10.9 %). Comparable findings were obtained from Müge Baykan et al[19] and Elena Segal et al.[25]

5- CONCLUSION

There are variety of causes of acute ataxia in children including acute cerebellar ataxia, CNS, and toxins and drug, putting in mind that there is many lives threatening condition can cause acute ataxia (brain tumors or abscesses). The highest incidence of acute ataxia was observed in preschool age children. Males were found to be affected more than females. Every case with acute ataxia should be evaluated thoroughly starting with history, physical and neurological examination with appropriate laboratory and radiological investigations. Additionally; every case of acute ataxia should be admitted to hospital to exclude life threatening conditions for early detections of this conditions and starting appropriate treatment accordingly.

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Conflict of intertest

About this study, the authors disclose no conflicts of interest.

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