Acute hyperkinetic movement disorders in Italian paediatric emergency departments

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ABSTRACT

Introduction Limited data exist on epidemiology, clinical presentation and management of acute hyperkinetic movement disorders (AHMD) in paediatric emergency departments (pED).

Methods We retrospectively analysed a case series of 256 children (aged 2 months to 17 years) presenting with AHMD to the pEDs of six Italian tertiary care hospitals over a 2-year period (January 2012 to December 2013).

Results The most common type of AHMD was tics (44.5%), followed by tremors (21.1%), chorea (13.7%), dystonia (10.2%), myoclonus (6.3%) and stereotypies (4.3%). Neuropsychiatric disorders (including tic disorders, psychogenic movement disorders and idiopathic stereotypies) were the most represented cause (51.2%). Inflammatory conditions (infectious and immune-mediated neurological disorders) accounted for 17.6% of the cases whereas non-inflammatory disorders (including drug-induced AHMDs, genetic/metabolic diseases, paroxysmal non-epileptic movements and idiopathic AHMDs) accounted for 31.2%. Neuropsychiatric disorders prevailed among preschoolers and schoolers (51.9% and 25.2%, respectively), non-inflammatory disorders were more frequent in infants and toddlers (63.8%), whereas inflammatory conditions were more often encountered among schoolers (73.3%). In 5 out of 36 Sydenham's chorea (SC) cases, tics were the presentation symptom on admission to emergency department (ED), highlighting the difficulties in early diagnosis of SC. Inflammatory disorders were associated with a longer hospital stay and a greater need of neuroimaging test compared with other disorders.

Conclusions This study provides the first large sample of paediatric patients presenting to the ED for AHMDs, helping to elucidate the epidemiology, aetiology and clinical presentation of these disorders.

What is already known on this topic?

- Differential diagnosis of acute-onset hyperkinetic movement disorders is broad, with both inflammatory and non-inflammatory conditions reported as the most common aetiology.
- Psychogenic movement disorders account for a considerable proportion of acute movement disorders in childhood.
- Children are more prone than adults to extrapyramidal effects of antidopaminergic drugs.

What this study adds?

- Tics are the most frequently encountered movement disorder in the paediatric emergency department (44.5% in our cohort).
- Neuropsychiatric disorders are the leading cause of admission to emergency department for acute-onset hyperkinetic movement disorders.
- Autoimmune and inflammatory disorders are the most demanding forms regarding neuroimaging need and days of hospitalisation.

INTRODUCTION

Movement disorders (MD) are defined as either an excess (hyperkinesias) or a paucity (hypokinesias) of voluntary and automatic movements.[1 2] Hyperkinetic MDs can be further classified into tics, chorea, dystonia, tremor, myoclonus and stereotypies.[3 4] While most MDs are chronic neurological disturbances, some can develop acutely.[5] Since several MDs may be treatable, timely recognition and diagnosis is crucial.[6]

MDs are an uncommon cause of admission in paediatric emergency department (pED), almost exclusively presenting as acute hyperkinetic movement disorders (AHMD). The literature on AHMDs is very limited, with most of the studies being based on outpatient clinic and specialist neurology services data[7 8] or focusing on only one type of MD, such as dystonia.[9]

Paediatricians must differentiate benign forms from conditions potentially resulting in significant morbidity.

The aim of this study was to improve knowledge about epidemiology, clinical presentation and aetiology of AHMDs as a chief complaint in children presenting to pEDs, in order to provide support

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for the clinical management to emergency and primary care physicians.

MATERIALS AND METHODS

Study setting and participants

This retrospective, multicentre case series was collected in the pED of six Italian hospitals (Turin, Padua, Genoa, Florence, Rome and Catania).

Patients aged 2 months to 17 years presenting from January 2012 to December 2013 with a primary complaint of AHMD were systematically included. Gait disorders related to unilateral weakness, vestibular dysfunction, ataxia, pain and epilepsy were excluded. Moreover, we excluded patients already diagnosed with conditions causing AHMDs.

Data collection and definitions

The clinical records were extracted from emergency department (ED) databases and analysed. The following data were collected: age, gender, triage code, any prior disease, main symptoms, specialist consultations, neuroimaging studies (CT and MRI), other diagnostic tests, final diagnosis, hospital admission and duration of hospitalisation, where applicable.

AHMDs were classified basing on the phenomenology dominating the clinical presentation on pED admission, according to standard clinical classification criteria in childhood.[3 4]

Patients were subdivided into four age classes: (1) <3 years (infants/toddlers); (2) 3–6 years (preschoolers); (3) 6–12 years (schoolers); (4) >12 years (teenagers).

The following triage codes were used: red (highly critical conditions), yellow (very urgent), green (urgent), white (non-urgent).

Based on discharge diagnosis, patients were classified into three diagnostic categories previously established according to similar studies[7 8]: (1) neuropsychiatric disorders (NPD) (tics, stereotypies and psychogenic movement disorders (PMD)); (2) inflammatory disorders (ID) (Sydenham's chorea (SC), paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS), autoimmune encephalitis and opsoclonus-myoclonus syndrome (OMS)); (3) non-inflammatory disorders (NID) (drug-induced AHMDs, metabolic/genetic disorders, tumours, paroxysmal non-epileptic movements, physiological or essential tremors).

Statistical analysis

We described the clinical and demographic features both of the overall sample and of the three diagnostic subgroups. The three groups were compared by means of the X^2 test for categorical variables and Student's t-test for continuous variables after reviewing for appropriateness. The statistical significance was set at $p<0.05$. SPSS software (V.24.0) was used to perform all statistical analyses.

RESULTS

Main clinical and demographic features

During the 2-year study period, among a total of 432.033 children admitted to the pEDs of the six hospitals involved, 256 subjects presented with AHMDs (5.9 visits per 10.000 ED attendances). Of the 256 patients, 149 were male (58.2%) and 107 were female (41.8%, M:F ratio=1.4), with a mean age of 76.6±50.7 months (range 2 months to 17 years). The age distribution was the following: infants/toddlers 27.3%; preschoolers 16.8%; schoolers 46.1%; teenagers 9.8%. On admission, 52 patients (20.3%) received a yellow code, 182 patients (71.1%) a green code and 22 patients (8.6%) a white one. Up to 204 patients (79.7%) were previously healthy, 25 children (9.8%) had a pre-existing neurological disease (migraine, epilepsy, cerebral palsy), 16 (6.3%) had a prior psychiatric disorder and 11 children (4.3%) were affected by other chronic disturbances.

With regard to pharmacological treatment, 17 patients (5.9%) were on neuroleptic or anticonvulsant drugs, 3 (1.2%) on antihistaminics and 5 (2.0%) on antiemetics.

Table 1 summarises the main demographic and clinical features of our sample.

Clinical presentation and final diagnosis

The most common AHMD was tics, seen in 114 patients (44.5%); tremors were reported in 54 patients (21.1%), chorea in 35 (13.7%), dystonia in 26 (10.2%), myoclonus in 16 (6.3%) and stereotypies in 11 (4.3%, table 1). At the time of the first clinical evaluation, 22.9% of patients had a normal neurological examination, while in 77.1% the presence of the AHMD referred at the time of triage was confirmed.

The frequency of the three AHMD subgroups significantly varied among the different age classes. Particularly, the IDs prevailed among schoolers (73.3%), the NIDs were more frequent in infants/toddlers (63.8%), while NPDs occurred more frequently both in schoolers and preschoolers (respectively 51.9% and 25.2%, table 1).

Table 2 summarises the clinical presentation and final diagnosis of our patients, in comparison with the cohorts described in the two previously published similar studies.[7 8]

Neuropsychiatric disorders

NPDs were the most frequent cause of AHMDs, identified in 131 (51.2%) patients, mostly simple or complex tic disorders (110 children), followed by PMDs (16) and stereotypies (5).

Inflammatory conditions

IDs were diagnosed in 45 patients (17.6%), Sydenham's was the only form of chorea encountered, diagnosed in 36 patients (14.2%). Remarkably, in 16 of them, a cardiac involvement was identified (44.4%). In addition, three cases of autoimmune encephalitis, two OMS (both secondary to neuroblastoma) and four cases of PANDAS were identified (table 2).

Non-inflammatory conditions

NIDs represented 31.2% (n=80) of AHMDs. Four patients had a metabolic/genetic disorder (two ceroidolipofuscinosis, one mucopolisacaridosis and one paroxysmal kinesigenic dystonia). Drug-induced AHMDs were documented in seven patients (2.7%), related to domperidone in three cases, to desloratadine in two, and to metoclopramide and haloperidol in the remaining two. In all of the children, complete remission occurred after treatment discontinuation.

In one patient, a pilocytic astrocytoma of basal ganglia was diagnosed. In 52 patients (20.3%), paroxysmal non-epileptic movements and essential or physiological tremors were diagnosed. Particularly, eight cases were classified as benign myoclonus of infancy and seven patients showed physiological tremors related to fever.

DISCUSSION

To date, few data are available about epidemiology, clinical phenotype and aetiology of children admitted to pED for AHMDs. Recently, two studies[7 8] investigated acute MDs in children; results were limited by the small cohort size and by

Table 1 Main demographic and clinical features of our sample

	Total n=256	Inflammatory conditions n=45 (17.6%)	Non-inflammatory conditions n=80 (31.2%)	Neuropsychiatric conditions n=131 (51.2%)	P value
Sex					
Female	107 (41.8%)	22 (48.9%)	46 (57.5%)	81 (61.8%)	NS (0.312)
Male	149 (58.2%)	23 (51.1%)	34 (42.5%)	50 (38.2%)	
Age (months; mean±SD (median))	76.6±50.7 (82.5)	99.9±31.8 (106)	47.8±57.6 (14.5)	86.2±43.5 (86)	0.0001
Age group					0.001
Infants/toddlers	70 (27.3%)	2 (4.4%)	51 (63.8%)	17 (13%)	
Preschoolers	43 (16.8%)	6 (13.3%)	4 (5%)	33 (25.2%)	
Schoolers	118 (46.1%)	33 (73.3%)	17 (21.3%)	68 (51.9%)	
Teenagers	25 (9.8%)	4 (8.9%)	8 (10%)	13 (9.9%)	
Triage code					0.007
White	22 (8.6%)	2 (4.4%)	3 (3.8%)	17 (13.0%)	
Green	182 (71.1%)	32 (71.1%)	53 (66.3%)	97 (74.0%)	
Yellow	52 (20.3)	11 (24.4%)	24 (30.0%)	17 (13.0%)	
Red	0	0	0	0	
MD type					0.0001
Tics	114 (44.5%)	8 (17.8%)	6 (7.5%)	100 (63%)	
Chorea	35 (13.7%)	31 (68.9%)	0	4 (3.1%)	
Tremors	54 (21.1%)	1 (2.2%)	38 (47.5%)	15 (11.5)	
Dystonia	26 (10.2%)	3 (6.7%)	20 (25%)	3 (2.3%)	
Myoclonus	16 (6.3%)	2 (4.4%)	12 (15%)	2 (4.4%)	
Stereotypies	11 (4.3%)	0	4 (5%)	7 (5.3%)	
Pre-existing disease					0.056
Not present	204 (79.7%)	39 (86.7%)	60 (75%)	105 (80.2%)	
Neurologic	25 (9.8%)	2 (4.4%)	14 (17.5%)	9 (6.9%)	
Psychiatric	16 (6.3%)	1 (2.2%)	3 (3.8%)	12 (9.2%)	
Other	11 (4.3%)	3 (6.7%)	3 (3.8%)	5 (3.8%)	
Specialist consultation	173 (67%)	32 (71%)	56 (70%)	85 (64.9%)	NS (0.63)
Neuroimaging					0.0001
Not done	202 (78.9%)	16 (35.6%)	66 (82.5%)	120 (91.6%)	
CT	8 (3.1%)	5 (11.1%)	1 (1.3%)	2 (1.5%)	
MRI	35 (13.7%)	20 (44.4%)	8 (10.0%)	7 (5.3%)	
CT plus MRI	11 (4.3%)	4 (8.9%)	5 (6.3%)	2 (1.5%)	
Outcome					
Discharged	168 (65.6%)	6 (13.3%)	56 (70.0%)	106 (80.9%)	0.0001
Hospitalised	88 (34.4%)	39 (86.7%)	24 (30.0%)	25 (19.1%)	
Length of hospital stay (days; mean±SD (median))	9.1±13.1 (6)	14±18.2 (10)	5.2±3.8 (4.5)	5.2±3.8 (4)	0.006

The different frequencies are compared by means of X^2 test.
MD, movement disorder; NS, not significant.

the study settings, referring mainly to child neurology services. Our study represents the first large, multicentre case series on AHMDs collected in pED.

The range of disorders causing AHMD is broad and the first diagnostic pitfall is the correct classification of the MD.[3] In fact, it can be difficult in children (especially in younger patients), also because of the overlapping features of different MDs.

The frequency of the different AHMDs in our series varied from previously reported data, likely because of the different setting of recruitment and the different inclusion criteria.

With regard to conditions causing AHMDs, NPDs were the most common aetiology, probably because of the inclusions of tic disorders in this subgroup, different from the previous studies where tics were excluded.[7 8]

Consequently, NPDs prevailed among patients aged 3–12 years, reflecting the high prevalence of tics during primary school.[10]

The different inclusion criteria are the consequence of the different setting of recruitment and explain the different composition of our series from those previously described.

IDs represented the second subgroup of AHMD causes. Particularly, SC represented the most common condition. Despite its lowering incidence in developed countries, SC remains the most common cause of chorea in children worldwide.[11 12] Consistent with published data,[11 13] girls were more often affected (F:M=1.6:1). The remarkable frequency of cardiac involvement highlights the importance of its prompt exclusion in any patient with acute-onset chorea.[6] Of note, in 5 out of 36 SC cases, tics were the presentation symptom on admission to pED. In fact, differential diagnosis between SC and tic disorders related to streptococcal infections can be difficult, particularly at onset.[14] On one hand, many patients with SC also have tics or psychological symptoms[6]; on the other hand, clinical distinction between

Table 2 Clinical presentation and final diagnosis of patients described in this study, compared with those reported by Dale *et al* and Goraya

	Dale *et al*[8]	Goraya[7]	Present study (2017)
Patients	52	92	256
Setting	Paediatric movement disorders service	Paediatric neurology service	Paediatric emergency department
Age	2 months to 15 years	5 days to 15 years	2 months to 17 years
Gender (M:F, ratio)	21:31; 0.67	63:29; 2.17	149:107; 1.39
Presenting AMD	**Hyperkinetic (n=59)**	**Hyperkinetic (n=92)**	**Hyperkinetic (n=256)**
	Tics (NC) Chorea (n=20) Dystonia (n=17) Tremors (n=12) Myoclonus (n=10)	Choreoathetosis (n=20) Dystonia (n=21) Tremors (n=15) Myoclonus (n=25) Tics (n=2) Tetany (n=5) Tetanus (n=2)	Tics (n=114) Chorea (n=35) Tremors (n=53) Dystonia (n=27) Myoclonus (n=16) Stereotypies (n=11)
	Hypokinetic (n=10)	**Hypokinetic (n=3)**	**Hypokinetic**
	Parkinsonism (n=10)	Parkinsonism (n=3)	NC
Aetiologies	**Inflammatory (n=22)**	**Inflammatory (n=32)**	**Inflammatory (n=45)**
	NMDA-R encephalitis (n=5) OMS (n=4) SC (n=3) SLE (n=3) ANE (n=3) Other encephalitis (n=3)	Encephalitis (n=11) OMS (n=7) SC (n=6) ADEM (n=3) Tetanus (n=3) Postinfectious tics (n=2) NMDA-R encephalitis (n=1)	AE including NMDA-R (n=3) SC (n=36) PANDAS (n=4) OMS (n=2)
	Non-inflammatory (n=18)	**Non-inflammatory (n=56)**	**Non-inflammatory (n=80)**
	Drug induced (n=7) Postpump chorea (n=5) Metabolic (n=3) Vascular (n=2)	Metabolic/nutritional (n=25) Physiological (n=17) Drug/toxins (n=4) Vascular (n=1) Traumatic brain injury (n=2) Benign paroxysmal torticollis (n=2) PAID (n=2) Cryptogenic (n=2) Ataxia-telangiectasia (n=1)	Primary dystonia (n=10) Physiological and essential tremors (n=30) Metabolic diseases (n=4) Undiagnosed myoclonus (n=6) Drug/toxins (n=7) PNED* (n=22) Cerebral tumour (n=1, pilocytic astrocytoma)
	Psychogenic (n=12)	**Psychogenic (n=4)**	**Neuropsychiatric (n=131)**
			Tic disorders (n=110) Isolated stereotypies (n=5) Psychogenic (n=16)

*PNED: benign myoclonus of infancy n=8, hypnic myoclonus n=1, jitteriness n=1, paroxysmal tonic upgaze n=1, Sandifer syndrome n=2, febrile myoclonus n=1, paroxysmal shuddering n=1, head nodding n=1, unspecified PNED n=6.
ADEM, acute disseminated encephalomyelitis; AE, autoimmune encephalitis; AMD, acute movement disorder; ANE, acute necrotising encephalopathy; NC, not collected; NMDA-R, N-methyl-D-aspartate receptor; OMS, opsoclonus-myoclonus syndrome; PAID, paroxysmal autonomic instability with dystonia; PANDAS, paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections; PNED, paroxysmal non-epileptic disorder; SC, Sydenham's chorea; SLE, systemic lupus erythematosus.

chorea and complex motor tics could be difficult in an emergency setting. Consequently, SC should always be considered in children presenting with acute-onset tics.

Of note, among 114 children presenting with acute-onset tics, only four fulfilled PANDAS diagnostic criteria. This finding suggests that PANDAS is an uncommon cause of acute-onset tics, according to previous reports.[15] Comprehensively, IDs represented the most concerning forms, requiring significantly longer hospitalisations and a wider use of neuroimaging.

Referring to NIDs, drug adverse reactions were a rare cause of AHMDs, in contrast with adult-based studies.[5] Neuroleptic drugs are a leading cause of iatrogenic MDs, with an increased risk in younger patients.[16 17] We encountered just one case of neuroleptic-related MD, probably because of the wider use of second-generation antipsychotics, which show a better tolerability profile, in the last years.[18] Similarly, although dystonic reactions have been frequently reported in children taking metoclopramide,[19] we found just one case of metoclopramide-induced dystonia. This low incidence in our series could be explained by the significant reduction of metoclopramide use in childhood since 2004, when in Italy its prescription was not recommended under the age of 16.

In contrast with literature data,[7 8] in our cohort no cases of postpump chorea were observed. This is obvious considering that postpump chorea only occurs in hospitalised patients undergoing cardiopulmonary bypass.[20]

The present study suffers from some limitations. First, AHMD diagnosis has been made by emergency physicians, who are not expected to be experienced in AHMD assessment. Although neurologist consultation was requested for a non-negligible group of patients (40%), the robustness of clinical diagnosis could have been partially limited. Other major limitations reside in the retrospective nature of the study and its exposure to some selection biases. In fact, some AHMDs could have been misdiagnosed, underestimating the prevalence of these conditions. Conversely, their prevalence could have been overestimated at a tertiary care level. Finally, some data (eg, time before symptoms onset) have not been collected because they were lacking from ED records. These factors are expected to partially limit the validity of our conclusions.

CONCLUSION

To our knowledge, this is the first study analysing AHMD presentation in pEDs, producing the most representative cohort available so far.

We highlighted the importance of NPDs as a leading cause of AHMD in children; conversely, the spread of atypical neuroleptics could explain the low prevalence of antipsychotic-induced AHMDs. Finally, IDs represent the most demanding forms in terms of diagnostic and therapeutic efforts. Given that most of the inflammatory AHMDs result from treatable conditions, a high level of suspicion is required to early recognise these potentially harmful disorders. Moreover, differentiating SC and tic disorders, especially secondary to streptococcal infections, can prove challenging, particularly at onset.

In conclusion, AHMDs are still a diagnostic challenge, especially in children, and further prospective studies are needed to provide robust evidence to guide their management.

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