Secondary Dystonia—Clinical Clues and Syndromic Associations

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Background: Dystonia is a hyperkinetic movement disorder defined by involuntary sustained muscle spasms and unusual postures. Etiologically, dystonic syndromes can be broadly divided into primary and secondary forms, dystonia-plus syndromes and heredodegenerative forms. In particular, diagnosis of secondary dystonic syndromes can be challenging in view of the variety of causes. Purpose: The purpose of this article is to highlight some clinical clues and syndromic associations as well as investigational findings which may be helpful in the approach to a patient with suspected secondary dystonia. Methods: We outline characteristic clinical and neuroimaging findings which may be directive in the diagnostic process of dystonia patients and facilitate making the correct diagnosis, thus allowing initiating the best treatment. Results: Secondary causes of dystonia include, among others, strategic brain lesions of various origins, metabolic disease, neurodegenerative conditions, and previous exposure to drugs or toxins. Presence of clinical signs including prominent oromandibular involvement, eye movement disorders, retinitis pigmentosa, deafness, peripheral neuropathy, parkinsonism or progressive dementia should alert the clinician to consider a secondary cause. Strategic lesions within the basal ganglia, but also within the brainstem, cerebellum or cortical areas may underlie dystonia and should thus be excluded. Conclusions: When thorough clinical examination reveals features atypical of primary dystonia, syndromic associations may help the clinician to narrow down the list of differential diagnosis. Directive investigations like neuroimaging may confirm the clinical suspicion.

Key Words: Chorea, Brain infarction, Anterior cerebral artery.

Introduction and Methods

Dystonia is a hyperkinetic movement disorder characterized by involuntary sustained muscle spasms causing in twisting movements and abnormal posturing of one or multiple body parts. Dystonia syndromes can be classified by onset age, distribution and etiology. With respect to the latter, dystonic syndromes can be divided into primary and secondary forms, dystonia-plus syndromes and heredodegenerative forms. While numerous authors have reviewed the primary dystonias in recent years, highlighting the proceedings into genetics and other aspects of the primary dystonias, less emphasis has been given to the secondary forms, albeit exigent.

In this article we chose to focus on secondary dystonias, in particular, because their diagnosis can be challenging. Causes are multifold and include brain lesions of various nature, previous exposure to drugs (in particular dopamine antagonists) or toxins, metabolic conditions and neurodegeneration. Fortunately, combination of clinical features (“syndromic associations”) may serve as red flag towards certain syndromes and investigations may demonstrate characteristic findings which may confirm the diagnosis or be directive in the diagnostic process and facilitate making the correct diagnosis and thus allow initiating the ideal treatment.

In this article we point out some clinical clues and syndromic associations which may be helpful in the approach to a patient.
Results

Prevalence of secondary dystonia

How common is secondary dystonia? Prevalence data on secondary dystonia are limited. A Brazilian study of 122 patients with a dystonic syndrome, 46 (38%) were found to have a symptomatic form. Among these, the most frequent causes were tardive dystonia (35%) and perinatal cerebral injury (30%). Other causes included stroke (13%), encephalitis (6.5%) and Wilson’s disease (4%). Causes were more common in certain age groups: Younger patients tended to have had perinatal cerebral injury or encephalitis preceeding their dystonia. In older patients stroke and exposure to drugs (tardive dystonia) were more common. In a recent study by Wenning et al. of 16 elderly patients with dystonia, ten (62.5%) were classified as having a secondary form of dystonia. Of these, eight were drug induced, highlighting the importance of this etiology. This is in line with a large study of more than 3,000 dystonia patients, 29% of which had a secondary form:tardive dystonia was the leading cause.

However, in addition to tardive dystonia, there are a number of other secondary and heredodegenerative disorders which can cause dystonia as a predominant feature or as part of a syndrome. Certain clinical clues may help to narrow down the list of differential diagnoses and focus the investigations accordingly (Table 1).

Clinical clues

Dystonia with prominent oro-bulbar involvement

Some primary dystonias there may have laryngeal involvement, for example DYT4 (“whispering dystonia”, the underlying gene remains unknown), DYT6 (due to mutations in the THAP1 gene, as very recently identified), DYT12 (“rapid-onset dystonia parkinsonism”) and DYT17 (recessively inherited, linked to the chromosome 20, gene not yet identified) dystonia. However, overall, prominent oro-lingual-buccal dystonia is uncommon in primary dystonia and a secondary or heredodegenerative form should be considered, particularly, when severe (Table 1). In particular pervious neuroleptic intake, but also certain genetic disorders such as PKAN (pantothenate kinase associated neurodegeneration, previously known as Hallervorden-Spatz disease) due to mutations of the PANK2 gene, neuroacanthocytosis, neuroferritinopathy and Lesch-Nyhan syndrome are high on the list of differentials. With the exception of neuroferritinopathy (autosomal dominant inheritance) they follow autosomal recessive inheritance and family history may thus be negative for a similar disorder, but there may be a history of consanguinity.

Eye movement disorders

Patients with primary dystonia have normal eye movements. In fact, presence of an eye movement disorder hints towards a secondary form of dystonia.

Eye movement dysfunction may be in the form of supranuclear gaze palsy. Patients with a supranuclear gaze palsy may complain of difficulties going downstairs because of limited downward gaze which is usually more affected than upward gaze. Presence is limited to a few dystonic conditions, most importantly progressive supranuclear palsy (PSP), where parkinsonism is the prominent and patients are elderly. Furthermore, supranuclear palsy may be present in Niemann-Pick type C and PKAN, both inherited autosomal recessively. In Niemann-Pick type C, a neurovisceral lipid storage disorder, supranuclear gaze palsy is present in 75% of adult-onset cases and a presenting sign in 8% of cases.

Dystonia and retinitis pigmentosa

The presence of retinitis pigmentosa in the context of dystonia narrows down the list of differential diagnoses. Most important differential diagnoses in this context are PKAN (or the allelic disorder referred to as HARP syndrome characterized by hypoprebetalipoproteinemia, acanthocytosis, retinitis pigmentosa, and pallidal degeneration), GM2 gangliosidosis and metachromatic leukodystrophy.

Dystonia and deafness

The combination of dystonia and deafness is characteristic of the Mohr-Tranebjerg syndrome due to a new mutations in the DDP1 gene. Other mitochondrial disorders may produce a complex phenotype which may involve additional visual problems (blindness) or heart problems.

Another complex disorder with phenotypic similarities to mitochondrial disease is the Woodhouse-Sakati syndrome. For this, only recently the underlying cause was identified and effects the C2orf37 gene, encoding a nucleolar protein. This rare autosomal recessive disorder presents with hypogonadism, alopecia, diabetes mellitus, mental retardation, and extrapyramidal features. As for other genetic disorders, there is phenotypic and genetic variability.

Dystonia and peripheral neuropathy

Presence of neuropathy is not a feature of primary dysto-

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Table 1. Clinical features suggestive of secondary dystonia

- Sudden onset and/or rapid progression
- Hemidystonia
- Cranial onset in childhood
- Restriction to focal or segmental dystonia in patients with childhood-onset
- Adult-onset of leg dystonia
- Progression to generalized dystonia of adult-onset dystonia
- Prominent oro-bulbar involvement
- Other neurological or systemic signs (except tremor)
ataxia can also be seen in some of the autosomal dominant D).25 Dystonia may then present as axial dystonia and bleph-ple system atrophy (MSA) or corticobasal degeneration (CB-
towards an atypical parkinsonian syndrome like PSP, multi-
dopa-responsive dystonia or Wilson’s disease. On the other
conditions may have superimposed parkinsonism as seen in
combination of young-onset dystonia and parkinsonism is
autosomal recessive disorders of nigro-striatal-pallidal-py-
ataxia, dystonia and peripheral neuropathy may be present.
for example, in a study of 70 ataxia telangiectasia pa-
patients, dystonia was present in 55 and peripheral neuropathy
A less common cause is the young-onset vari-
ataxia telangiectasia 17,18 and their differential diagnoses.19
where ataxia, dystonia and peripheral neuropathy may be pre-
patients, this can be a helpful clue towards the
diagnosis. A further clue may be enlargement of the liver
and spleen (present in about one 30-90% of cases)11,21 and, in
children, neonatal jaundice (present in half of the patients).
Notably, the combination of dystonia with ataxia and
ataxia can also be seen in some of the autosomal dominant
spinocerebellar ataxias,22 e.g. SCA 3.23
Finally, the combination of peripheral neuropathy, pro-
gressive dystonia and ataxia, as well as cognitive decline is
seen in metachromatic leukodystrophy caused by mutations
in the arylsulfatase A gene cause.24

**Dystonia and parkinsonism**

In addition to pure dystonic and pure parkinsonian syn-
dromes, there are overlap syndromes. On one hand dystonic
conditions may have superimposed parkinsonism as seen in
dopa-responsive dystonia or Wilson’s disease. On the other
hand, dystonia may be a seen in (or even be the presenting
feature of) various parkinsonian disorders.

While dystonia is uncommon in drug-naive patients with
idiopathic Parkinson’s disease, its presence may be a red flag
towards an atypical parkinsonian syndrome like PSP; multi-
ple system atrophy (MSA) or corticobasal degeneration (CB-
D).25 Dystonia may then present as axial dystonia and bleph-
aroas (levator inhibition) causing the starring expres-
sion associated with PSP; antecollis and facial dystonia in
MSA; or the dystonic arm posture seen in CBD.

Parkinsonism associated with dystonia is furthermore char-
acteristic of genetic forms of parkinsonism which often have
young-onset and recessive inheritance (for review see).26,27
One example is parkinsonism related to mutations in
the Parkin gene where dystonia may be present intermittently, as
so-called exercise-induced paroxysmal foot dystonia, and
this may precede signs of parkinsonism by some years.28 The
combination of young-onset dystonia and parkinsonism is
also seen in other neurodegenerative diseases like the rare
autosomal recessive disorders of nigro-striatal-pallidal-py-
ramidal syndrome referred to as Kufor-Rakeb disease and
PLA2G6-associated neurodegeneration (PARK14).29 (also
see review by Schneider, et al.26)

Most frequent, however, is dystonia in the context of par-
kinsonism as complication of dopaminergic treatment, for
example as peak-dose dystonia, diphasic dystonia and off-
dystonia.28

**Dystonia with progressive dementia**

Progressive dementia is not a feature of the primary dys-
tonias like the young-onset DYT1-related dystonia or the
adult-onset sporadic forms. Progressive dementia is, however,
one of the core features of Huntington’s disease and the
Huntington disease-look like syndromes (including HDL4/
SCA17), as well as neuroacanthocytosis and PKAN. In-
deed, chorea is the main movement disorder, however, pro-
minent dystonia can occur. A further condition to consider
is Creutzfeldt-Jakob disease, a rare neurodegenerative dis-
ee characterized by rapidly progressive dementia, mutism,
ataxia and extrapyramidal and pyramidal involvement.30 The
movement disorder is typically characterized by focal or
generalized myoclonus (present in 80-100% of cases), but
dystonia occurs and may rarely be a presenting sign.30-32
Dystonia in Creutzfeldt-Jakob disease is then usually unilat-
eral and distally but may become generalized in later disease
stages.30 Over all, disease course is rather progressive and
this should alert the clinician to this diagnosis. Furthermore,
HIV encephalopathy is a cause of dementia: and dystonia
has also been observed.33

Finally, dementia may also be a symptom in the complex
autosomal recessive dystonia parkinsonian syndromes men-
tioned above.26

**Clues from neuroimaging**

Neuroimaging can reveal patterns characteristic of certain
conditions. Strategic lesions in the basal ganglia, brainstem,
cerebellum or cortical areas (parietal and frontal) may result
in dystonia34-41 and there may be a relationship between the
distribution of dystonia and the localization of the lesion.
For example, it has been proposed that thalamic lesions are
more likely to result in hand dystonia. However, not all basal
ganglia lesions necessarily result in neurological symptoms
or signs. Brainstem lesions on the other hand have been as-
associated with cranial dystonias such as blepharospasm, and
putaminal lesions were found in patients with hemidystonia
or limb dystonia. In hemidystonia, lesions are often unilat-
eral, contralaterally to the dystonia.

Of course, the list of causes underlying the lesion is long
and includes tumors, trauma, bleeding, inflammation, atroph-
ic changes in the context of neurodegeneration or accumula-
tion of metals (such as iron, copper, manganese etc.).

How imaging can facilitate the diagnostic work-up we
will be discussed in more detail as an example for basal
ganglia disorders with metal deposition. The basal ganglia host high concentrations of metals which act as cofactors for metabolic activity including iron, copper and manganese. In the case of excessive metal accumulation, this may cause dysfunction and disease.

Metal deposition can be detected by neuroimaging on CT (e.g. copper) or MRI (e.g. iron). In recent years, in particular, iron deposition has received growing attention and a new term referring to these disorders, “syndromes of neurodegeneration with brain iron accumulation” (NBIA), has been coined. This group entails the condition of PKAN (Neurodegeneration with brain iron accumulation) (NBIA), has been proposed that presence of globus pallidus calcifications only requires further elaboration when the patient is younger than 40 years of age. In addition all patients, no matter of age, where other basal ganglia or brain areas are affected should be further investigated. The differential diagnosis is wide and includes metabolic, infectious, toxin-induced and degenerative causes. Among the metabolic disorders, idiopathic or surgical hypoparathyroidism is probably the most common cause of symmetric basal ganglia calcification, and dystonia as presenting feature may occur. Infections (including congenital forms) by toxplasmosis, rubella, cytomegaly, herpes and HIV may result in basal ganglia damage with calcifications and secondary dystonia. Following carbon monoxide poisoning movement disorders including dystonia may develop as a part of delayed encephalopathy and imaging may reveal basal ganglia calcifications. Neurodegenerative causes include Wilson’s disease.

Last but not least, familial causes of basal ganglia calcifications have been recognized, also referred to as striopallidodentate calcinosis or Fahr’s disease and dystonia has been observed.

Conclusions

While presence of tremor is compatible with a diagnosis of primary dystonia, there are other clinical features which point away from this diagnosis, including eye movement disorders, retinitis pigmentosa or peripheral neuropathy, to name a few. In these syndromic associations, some of which have been outlined in this article, can be useful and help the clinician to narrow down the list of differential diagnosis. Investigations such as a peripheral blood smear to screen for neuroacanthocytosis or neuroimaging may help to reach at the correct diagnosis.

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