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REVIEW ARTICLE

Panayiotopoulos Syndrome: A Benign Childhood Autonomic Epilepsy Frequently Imitating Encephalitis, Syncope, Migraine, Sleep Disorder, or Gastroenteritis

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ABSTRACT

BACKGROUND. Panayiotopoulos syndrome is a common idiopathic childhood-specific seizure disorder formally recognized by the International League Against Epilepsy. An expert consensus has defined Panayiotopoulos syndrome as "a benign age-related focal seizure disorder occurring in early and mid-childhood. It is characterized by seizures, often prolonged, with predominantly autonomic symptoms, and by an EEG [electroencephalogram] that shows shifting and/or multiple foci, often with occipital predominance."

OBJECTIVE. The purpose of this review is to provide guidance for appropriate diagnosis and management of Panayiotopoulos syndrome.

CLINICAL FEATURES. Autonomic epileptic seizures and autonomic status epilepticus are the cardinal manifestations of Panayiotopoulos syndrome. Autonomic seizures in Panayiotopoulos syndrome consist of episodes of disturbed autonomic function with emesis as the predominant symptom. Other autonomic manifestations include pallor (or, less often, flushing or cyanosis), mydriasis (or, less often, miosis), cardiorespiratory and thermoregulatory alterations, incontinence of urine and/or feces, hypersalivation, and modifications of intestinal motility. In approximately one fifth of the seizures the child becomes unresponsive and flaccid (ictal syncope) before or often without convulsions. Cardiorespiratory arrest is exceptional. More-conventional seizure symptoms often appear after the onset of autonomic manifestations. The child, who was initially fully conscious, becomes confused and unresponsive. Eyes turn to one side or gaze widely open. Only half of the seizures end with brief hemiconvulsions or generalized convulsions. Convulsive status epilepticus is extremely rare. Autonomic symptoms may be the only features of the seizures. Half of the seizures in Panayiotopoulos syndrome last for >30 minutes, thus constituting autonomic status epilepticus, which is the more common nonconvulsive status epilepticus in normal children. Two thirds of seizures occur during sleep.

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Key Words
PS, Panayiotopoulos syndrome

Abbreviations
EEG— electroencephalogram
PS—Panayiotopoulos syndrome
ILAE—International League Against Epilepsy

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EPIDEMIOLOGY. Panayiotopoulos syndrome probably affects 13% of children aged 3 to 6 years who have had 1 or more afebrile seizures and 6% of such children in the 1- to 15-year age group.

DIAGNOSTIC TESTS. An electroencephalogram is the only investigation with abnormal results, usually showing multiple spikes in various brain locations.

PATHOPHYSIOLOGY. Panayiotopoulos syndrome is probably the early-onset and Rolandic epilepsy the late-onset phenotype of a maturation-related benign childhood seizure-susceptibility syndrome. Ictal epileptic discharges in Panayiotopoulos syndrome, irrespective of their location at onset, activate autonomic disturbances and emesis, to which children are particularly vulnerable. The symptoms/sequence of autonomic seizures and autonomic status epilepticus in Panayiotopoulos syndrome are specific to childhood, and they do not occur in adults.

PROGNOSIS. Panayiotopoulos syndrome is remarkably benign in terms of seizure frequency and evolution. Autonomic status epilepticus imparts no residual neurologic deficit. The risk of epilepsy in adult life seems to be no higher than in the general population. However, autonomic seizures are potentially life-threatening in the rare context of cardiorespiratory arrest, an area in which additional study is required.

MISDIAGNOSIS. The clinical features of Panayiotopoulos syndrome are frequently mistaken as nonepileptic conditions such as acute encephalitis, syncope, migraine, cyclic vomiting syndrome, motion sickness, sleep disorder, or gastroenteritis. The consequence is avoidable misdiagnosis, high morbidity, and costly mismanagement.

MANAGEMENT. Education about Panayiotopoulos syndrome is the cornerstone of management. Prophylactic treatment with antiepileptic medication may not be needed for most patients. Autonomic status epilepticus in the acute stage needs thorough evaluation; aggressive treatment may cause iatrogenic complications including cardiorespiratory arrest.

A MAJOR ADVANCE in epileptology has been the recognition of syndromes with distinct etiology, clinical and electroencephalographic (EEG) features, and prognosis, which define appropriate management.[1,2] A prime example of this is Rolandic epilepsy, which is now well recognized by pediatricians, who can convey its excellent prognosis to parents who worry about the possibility of "epilepsy." Rolandic epilepsy comprises ~15% of childhood epilepsy and has a peak onset at 7 to 8 years of age. Seizures mainly occur during sleep and comprise hemifacial sensorimotor symptoms, speech arrest, oropharyngolaryngeal movements, and hypersalivation. EEG shows frequent spikes in the centrotemporal brain regions.[1]

However, Rolandic epilepsy is not the only benign childhood epileptic syndrome. Panayiotopoulos syndrome (PS) is a common and benign childhood autonomic epilepsy that has recently attracted significant interest beyond epileptologists and has been highlighted in editorials of major medical journals,[3–5] documented in >800 patients worldwide,[6–18] and officially recognized by the International League Against Epilepsy (ILAE).[2] In a recent expert consensus PS was defined as "a benign age-related focal seizure disorder occurring in early and mid-childhood. It is characterized by seizures, often prolonged, with predominantly autonomic symptoms, and by an EEG that shows shifting and/or multiple foci, often with occipital predominance."[16]

Autonomic seizures and autonomic status epilepticus with ictal emesis are the cardinal manifestations of PS. Autonomic seizures are epileptic attacks that start or entirely manifest with ictal autonomic disturbances that may be objective, subjective, or both. Autonomic status epilepticus is an autonomic seizure that lasts for >30 minutes.[17,19,20] Epileptic discharges in PS, irrespective of their location at onset, activate autonomic disturbances and emesis, to which children are particularly vulnerable.[16,17,19] This childhood-related susceptibility to autonomic disorders is illustrated by cyclic vomiting syndrome and motion sickness, which are nonepileptic conditions in children.[16,17,19]

The ictal clinical features of PS are unusual for epileptic seizures and frequently imitate nonepileptic conditions such as acute encephalitis, syncope, migraine, cyclic vomiting syndrome, motion sickness, sleep disorder, and gastroenteritis. The consequence is avoidable misdiagnosis, high morbidity, and costly mismanagement.[13,17,18]

PS is probably the early onset and Rolandic epilepsy, the late-onset phenotype of a maturation-related benign childhood seizure-susceptibility syndrome.[16,17,19] Idiopathic childhood occipital epilepsy (Gastaut type) is another syndrome recognized by the ILAE in the same category as PS and Rolandic epilepsy.[2,18,21] Idiopathic childhood occipital epilepsy is rare and of unpredictable prognosis. It manifests with brief, frequent, and diurnal purely occipital seizures. Elementary visual hallucinations and blindness are the predominant ictal symptoms. Postictal migrainous type of headache is common.[18]

Table 1 shows the key features of PS compared with Rolandic epilepsy and idiopathic childhood occipital epilepsy.[16–18]

HISTORICAL ASPECTS

This syndrome was described by Panayiotopoulos through a 30-year prospective study detailed in his monographs.[17,22] In doing so, Panayiotopoulos also described autonomic seizures and autonomic status epilepticus specific to childhood.

His cohort consisted of 900 children and adult pa-

TABLE 1 PS Versus Rolandic Epilepsy and Idiopathic Childhood Occipital Epilepsy

	PS	Rolandic Epilepsy	Idiopathic Childhood Occipital Epilepsy
Prevalence in children aged 1–15 y with nonfebrile seizures, %	6	15	<1
Mean age at onset (range), y	4–5 (1–15)	7–8 (1–15)	10–11 (4–17)
Male/female ratio	1	1.5	1
Main seizure symptoms at onset	Emesis and/or other autonomic disturbances	Facial sensorimotor, speech arrest	Elementary visual hallucinations or blindness
Common duration of seizures	>9 min	1–3 min	Seconds to <1 min
Total No. of seizures in most patients (% single)	1–5 (30)	1–10 (20)	Hundreds (0)
Circadian distribution (%)	Nocturnal (64)	Nocturnal (70)	Diurnal (100)
Focal nonconvulsive status epilepticus (>30 min), %	40	Exceptional	Exceptional
Febrile convulsions, %	17	10–20	Unknown
Common interictal EEG spike location	Multifocal	Centrotemporal	Occipital
Onset of ictal EEG	Posterior or anterior brain regions	Lower part of pre and post central gyrus	Occipital
Prognosis	Excellent	Excellent	Unpredictable: 70% remit

tients who had had 1 or more afebrile seizures. For 414 of them, onset of seizures was between the ages of 1 and 15 years.[23,24] Autonomic seizures, commonly associated with ictal vomiting, occurred in 28 patients, all of whom were children. Half of the autonomic seizures lasted for several hours, thus constituting autonomic status epilepticus. Of these 28 children, 25 were otherwise normal and comprised the cohort of what is now known as PS, and the other 3 had cerebral pathology (symptomatic cases).

From the EEG point of view, despite similar clinical features and prognosis, the 25 patients with normal development were grouped in 2 subsets:

1. The first group consisted of 16 patients with predominantly occipital spikes alone or in combination with spikes in other anatomic brain locations. This subset of patients with PS has attracted more attention,[24] hence the name "early-onset benign childhood occipital epilepsy (Panayiotopoulos type)" proposed by the ILAE.[2]
2. The second group of 9 children had extraoccipital spikes (5 patients), brief generalized discharges (1 patient), or a normal EEG (3 patients). This subset of children had similar seizures and as good a prognosis as the first group with occipital spikes.[23,25] It is now recognized that these patients comprise a significant proportion of patients with PS.[16] Hence, descriptive terms such as "occipital epilepsy" or "epilepsy with occipital spikes" do not represent the true spectrum of PS.[10–12,16]

Retrospectively, another 11 of the 414 children were identified with probable or atypical PS.[17]

PREVALENCE

PS has high prevalence. It affects ~13% of children aged 3 to 6 years who have had 1 or more afebrile seizures and 6% of such children in the 1- to 15-year age group.[17,18] In the general population, 2 to 3 in 1000 children may be affected.[17,18] These figures may be higher if cases that are currently considered to have atypical features are included.[17,18]

AGE AT ONSET AND GENDER

Age at onset is between 1 and 14 years and at peak between 4 and 5 years; 76% start between 3 and 6 years.[17,18] Boys and girls of all races are equally affected.

CLINICAL MANIFESTATIONS

Autonomic seizures and autonomic status epilepticus are the principal clinical features of PS.[6–18,23] In a typical presentation of PS, the child is fully conscious, is able to speak, is aware, complains that "I feel sick" or "I want to be sick," looks pale, and vomits.[6–13,16–18,23] Two thirds of the seizures start in sleep. The child either wakes up with similar complaints or is found vomiting, conscious, confused, or unresponsive.

Emesis and Other Autonomic Ictal Manifestations

The full emetic triad (nausea, retching, vomiting) culminates in vomiting in 74% of the seizures[17]; in others, only nausea or retching occur, and in a few, emesis may not be apparent.[6–13,16–18,23] Other autonomic manifestations may occur concurrently or appear later in the course of the ictus. These include pallor (or, less often, flushing or cyanosis), mydriasis (or, less often, miosis), cardiorespiratory and thermoregulatory alterations, incontinence of urine and/or feces, hypersalivation, and modifications of intestinal motility.[16–18] Headache or, more often, cephalic auras and behavioral disturbances may occur, particularly at onset.

An unusual and important ictal feature of PS is that in at least one fifth of the seizures the child becomes unresponsive and flaccid (syncopal-like attacks or ictal syncope) before or without convulsions.[16–19] Ictal syncope is sometimes the sole clinical event, without other mani-

festations recognizable as seizures. Brief apnea and cardiac asystole may be common but are usually mild. Cardiorespiratory arrest is exceptional, probably occurring in up to 1 per 200 individuals with PS.[16,17,26]

Conventional Seizure Symptoms

More-conventional seizure symptoms often appear after the onset of emesis or other autonomic manifestations.[6–13,16–18,23] The child, who was initially fully conscious, gradually or suddenly becomes confused and unresponsive, although (rarely) consciousness may be preserved (6%).[17] Eyes and often the head turn to one side (60%) or eyes gaze widely open (12%).[17] Only half of the seizures (40%) end with brief hemiconvulsions or generalized convulsions.[6–13,16–18,23] Convulsive status epilepticus is extremely rare.

Duration of Seizures and Autonomic Status Epilepticus

The duration of the seizures is usually longer than 10 minutes.[16–19] Half (44%) of them last from 30 minutes to many hours, constituting autonomic status epilepticus.[6–13,16–18,23] The same child may have brief or prolonged seizures with marked or inconspicuous autonomic manifestations. Even after the most severe and lengthy seizures, the patient is normal after a few hours' sleep.

INVESTIGATIONS

PS affects children with normal development. The EEG, as with Rolandic epilepsy, is the most useful diagnostic test (Fig 1). The results of all other investigations are normal. Brain MRI may be needed for children suspected of symptomatic autonomic seizures caused by structural brain pathology.

The EEG in PS commonly (90%) reveals multifocal functional spikes that are accentuated by sleep.[7,10–13,16–18,23] The term "functional spikes" refers to transient focal EEG abnormalities of sharp waves that occur in children with or without epileptic seizures and disappear in the late teenage years.[27] They are exemplified by the centrotemporal spikes of Rolandic epilepsy.[27] In PS, functional spikes appear in many brain locations, often shifting from one to another region in series of EEGs. Occipital spikes predominate, but they do not occur in one third of the patients. EEGs of some children with typical clinical features of PS may be identical to those of Rolandic epilepsy.[10] Results of a single routine EEG may be normal in 10% of patients, which should prompt a request for a sleep EEG. The frequency, location, and persistence of spikes do not determine clinical manifestations, duration, severity and frequency of seizures, or prognosis. The clinical seizure manifestations are roughly the same irrespective of EEG-spike localization.

The multifocal nature of epileptogenicity in PS has also been documented with dipole analysis[15] and magnetoencephalography.[14,28] The latter revealed that the main epileptogenic areas are along the parietal-occipital, the calcarine, or the central (Rolandic) sulci.[14]

Ictal video-EEG has unequivocally documented the epileptic nature of the autonomic manifestations in PS.[7,29–31] These may start long after the onset of the electrical ictal discharge and manifest with tachycardia, breathing irregularities, coughing, or emesis, which would be impossible to consider as seizure events without EEG. Other recognizable conventional seizure symptoms such as convulsions appear later in the ictal phase or may not appear at all. The electrical onset of the ictal EEG paroxysms is more often posterior than anterior, with right or left lateralization.[7,29–31]

PROGNOSIS

PS is remarkably benign despite the high incidence of autonomic status epilepticus.[6–13,16–18,23,25] One quarter of patients have a single seizure only and half of the patients have 2 to 5 seizures. The remaining quarter have >6 or sometimes very frequent seizures. Remission often occurs within 1 to 2 years after onset. Autonomic status epilepticus imparts no residual neurologic deficit. Atypical evolution of PS with the development of absences, drop attacks, and epilepsy with continuous spike and wave during slow-wave sleep is extremely rare.[32,33]

One fifth of children with PS develop Rolandic (13%) and, less often, occipital seizures during childhood and the early teen years.[16–18] These are also age related and remit before the age of 16 years. The risk of epilepsy in adult life seems to be no higher than in the general population, although there is a need for more studies with long-term follow-up.[16,17]

Although the syndrome is benign in terms of its evolution, autonomic seizures are potentially life-threatening in the rare context of cardiorespiratory arrest.[16,17,26] This is an area in which additional study is required, although it is reassuring that normal children with epilepsy do not have an increased risk of death compared with the general population.[34]

MISDIAGNOSIS

Despite its dramatic and lengthy manifestations, PS is often misdiagnosed.[9,13,17,19,31] The main reason for this is that emetic and other autonomic manifestations are not recognized as seizure events. Brief ictal autonomic symptoms may suggest nonepileptic conditions such as atypical migraine, gastroenteritis, motion sickness, syncope, or sleep disorder. Prolonged and severe attacks may simulate acute cerebral insults such as encephalitis or intoxication. The consequence of misdiagnosis is avoidable morbidity, erroneous treatments, and costly hospital admissions.[9,13,17,19,31]

It should also be stressed that 10% to 20% of children with autonomic seizures of similar presentation to PS suffer from focal or diffuse brain lesions.[17,19,23] These patients can usually be differentiated, because they have

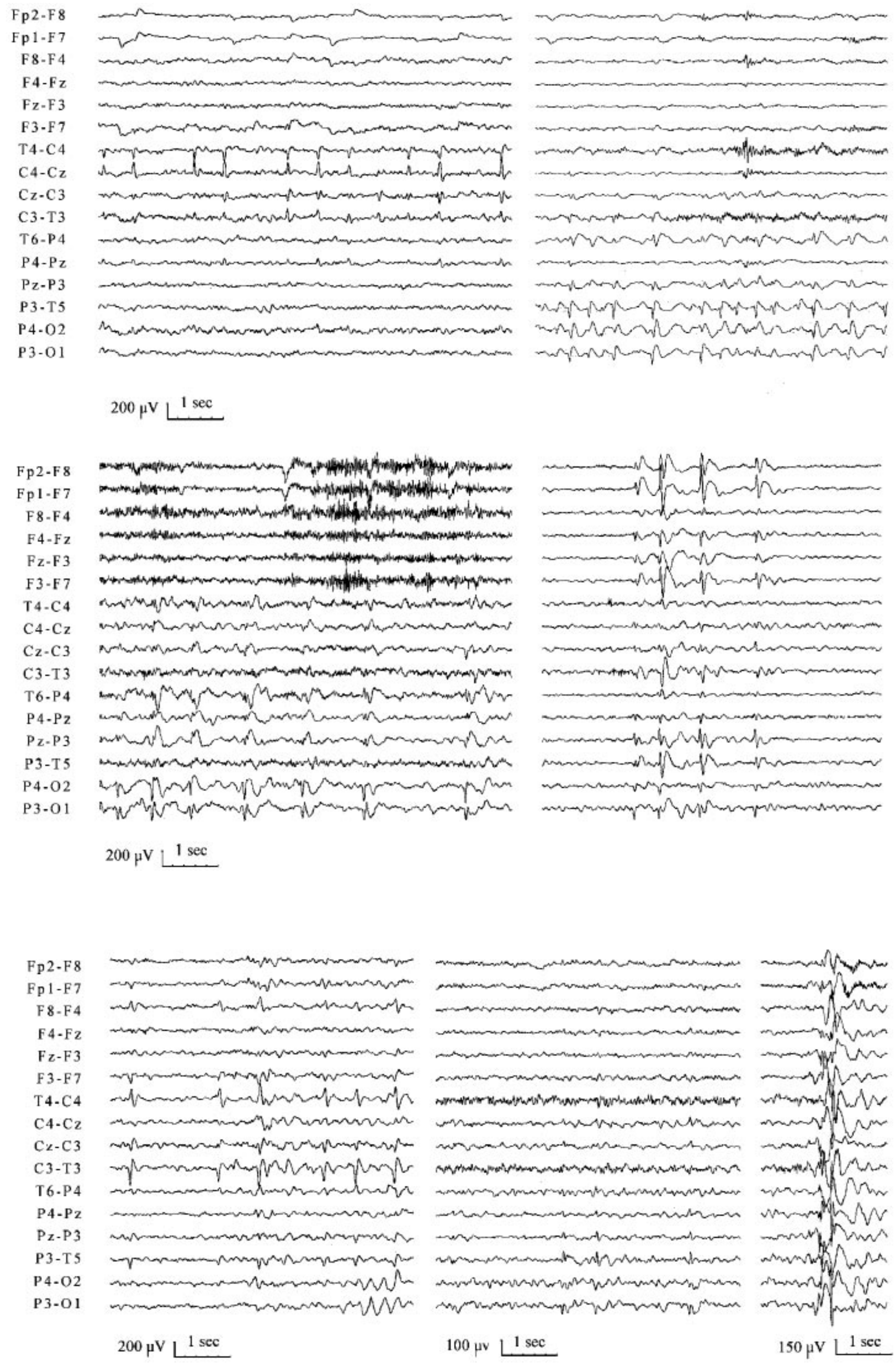

FIGURE 1
Top left, Typical EEG in Rolandic epilepsy with clusters of spikes localized in the central (C3 and C4) electrodes. Top right, Typical EEG in idiopathic childhood occipital epilepsy with occipital paroxysms (O2 and O1 electrodes). Middle and bottom, EEG variability in 5 patients with PS. EEGs show clusters of occipital spikes (middle left), repetitive paroxysms of diffuse but mainly bifrontal spikes (middle right), paroxysms of bitemporal spikes (bottom left), small occipital spikes (bottom middle), and brief generalized discharges (bottom right). An EEG may show single and infrequent spikes or be normal, but no such examples are illustrated here.

abnormal neurologic signs and brain imaging. They commonly have additional nonautonomic seizures that continue into adult life.

PS may also be misdiagnosed as Rolandic epilepsy because of clinical and EEG features that sometimes overlap. Febrile seizures may be diagnosed when seizures coincide with fever either inadvertently or as the result of ictal thermoregulatory changes. However, this has no adverse prognostic implications, because Rolandic and febrile seizures are also benign and age related.

Clinically, PS is entirely different from idiopathic childhood occipital epilepsy (Gastaut type)[21] despite sometimes-similar EEG abnormalities of occipital spikes (see Table 1).[16–18]

MANAGEMENT

There have been no randomized, controlled studies or official guidelines regarding diagnostic procedures and management of PS. Panayiotopoulos extrapolated appropriate modifications[19] of the current recommendations regarding febrile seizures.[35]

Education about the nature and prognosis of PS is the cornerstone of correct management. The traumatizing, sometimes long-lasting effect on caregivers, even of febrile seizures,[35] is predictably worse for autonomic seizures that may last for many hours, especially when compounded by physicians' uncertainty regarding diagnosis, management, and prognosis.[36] Supportive family management includes education about PS and specific instructions about emergency procedures for possible subsequent seizures.

Autonomic status epilepticus in the acute stage needs thorough evaluation for proper diagnosis and assessment of the neurologic/autonomic state of the child. Benzodiazepines, intravenous or in rectal or buccal preparations, are commonly used to terminate this nonconvulsive status epilepticus.[16] Aggressive treatment should be avoided because of the risk of iatrogenic complications including cardiorespiratory arrest. Early treatment of rectal or buccal benzodiazepines, given by the parents is more effective than late emergency treatment.

Prophylactic treatment with antiepileptic medication may not be needed for most of the patients with PS.[17,19] An expert consensus statement concluded that such treatment was probably best reserved for children whose seizures were unusually frequent, distressing, or otherwise significantly interfering with the child's life.[16] There is no evidence of superiority of monotherapy with any particular antiepileptic drug. Most authors prefer carbamazepine,[19] although this drug may sometimes worsen seizures in a child with PS.[37] Recommendations in this area may change through randomized, controlled studies, the introduction of new antiepileptic drugs, and improved understanding of the risk of cardiorespiratory arrest.

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