



Review Article

Vertigo in children; a narrative review of the various causes and their management

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ABSTRACT

Vertigo is not an uncommon symptom in children, but often the treating doctors are unsure of the diagnosis and the management of these cases. This narrative review of the literature discusses the brief etiopathology, the clinical manifestations and the management algorithm of most of the conditions causing vertigo in children. The relevant information has been condensed into a table for the perusal of the readers, which would assist in the appropriate management of these children.

1. Introduction

The etiopathology of vertigo among children differs from that in adults. Not only that 'vertigo in children' has peculiarly different differential diagnoses but also, the affected children respond relatively well to the treatment, and they recover quicker than the adults. However, pediatricians, physicians, otorhinolaryngologists and neurologists often find it difficult to establish the appropriate diagnosis in these cases. The inability of the affected children to explain the characteristics of the experienced symptoms may preclude the diagnosis making, especially in very young children. In addition, the vestibular tests, both clinical and neurophysiological, are not uniformly reliable in the younger patients. However, the critical factor for the delay in diagnosis or the misdiagnosis is the lack of awareness about the nuances in symptomatology and management algorithms among the treating clinicians. This narrative review of the literature discusses the brief aetiopathology, the clinical manifestations, and the management algorithms of most of the conditions causing vertigo in children. The objective is to familiarize the readers with these seemingly complex disorders and about the current protocols in their management. The detailed accounts into the various causes of the vertigo in children, including the rarer ones have been discussed, and a master table depicting the characteristics of these conditions has been included.

2. Differential diagnosis of vertigo in children

2.1. Benign paroxysmal vertigo of childhood

Benign paroxysmal vertigo (BPV) is one of the most common causes of vertigo in children. It was first described by Basser in 1964 and was

thought to be a variant of vestibular neuritis [1]. Though the exact etiology is not yet precisely known, it is considered as a variant or an equivalent of a pediatric migraine [2,3]. Transient ischemia of vestibular nuclei and/or vestibular pathway has been proposed to be the cause for BPV [4]. It has no relation or association with the benign paroxysmal positional vertigo, which is uncommon in childhood [5]. Clinically, it is characterized by recurrent brief episodic attacks of vertigo of few minutes, occurring without warning, and resolving spontaneously in otherwise healthy children [3]. Though the BPV has been reported in children aged between two and 12 years, the majority of the affected children are of less than four years of age [6]. Girls are affected more commonly than boys [2]. Many authors have reported the family history of a migraine in at least half of the BPV patients in their series [4,5,7,8]. Clinical examination and investigations would be essentially normal in BPV cases, without any hearing or vestibular deficit. However, even in those with normal hearing, abnormal 'auditory brainstem response' has been reported in 66% (n = 56) of the BPV children [9]. Some authors have reported altered caloric test and 'vestibular evoked myogenic potentials' in the affected children [10]. Rarely even cochlear signs may develop over time [11]. Since BPV is a self-limiting disorder, the management includes reassuring the affected children and their parents. In general, vertigo in children is not so bothersome, is associated with less vegetative symptoms, and the affected children tend to return to normal day to day activities much more quickly as compared to adults [12]. BPV was thought to resolve entirely without any long-term sequel based on initial reports by Lindskog (n = 19) [13]. However, later studies have reported the development of a migraine in more than 35% of BPV children [6–8].

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2.2. Vestibular migraine

Though Kayan and Hood described the condition for the first time in 1984 [14], the term Vestibular Migraine (VM) was coined by Dieterich and Brandt in 1999 [15] and was supported by others [16]. VM is the most common cause of vertigo in children [17–20]. As per the recent review (n = 2726), VM constitutes 24% of all the causes of vertigo in children [21]. However, VM is the disease of older children, and the BPV remains the commoner causes of giddiness in children up to the age of six years [22] BPV is rarely seen in children older than ten years, and VM is rare in children younger than ten years [17,23]. The pathogenesis of VM is not yet clear [24]. The genetic susceptibility of the individual leading to his/her enhanced sensory excitability seems to be responsible for the VM [25].

Clinical manifestation of VM includes recurrent vestibular symptoms temporally associated with a migraine. Vertigo in VM may last anytime between 5 min and 72 h [25]. Lempert gave the diagnostic criteria for VM [26], and later the same was modified and adopted by international classification of headache society [27]. In VM, vertigo can occur before, during or after a migraine headache [28] and vice versa [29]. Affected children may show sensitivity to light and noise [30]. In-between the vertigo episodes, a detailed neuro-otological examination would usually be within the normal limits, though, in some of the affected children it may reveal ocular motor abnormalities and canal paresis with or without the sensorineural hearing loss [25,28,30–32]. Canal paresis has been reported in up to one-third of cases [17]. However, the neurophysiological tests show inconsistent results in VM and may not be required for diagnosis [25,32].

The treatment guidelines for VM are extrapolated from that of migraine and those from studies on the adult population. There are no studies done in children, and even in adults, open randomized controlled trials are scarce. Triptans and calcium channel blockers have been tried for treatment as well as prophylaxis of VM with the satisfactory results [16,25,33]. Tricyclics, cyproheptadine, topiramate, and gabapentin have also been found to be beneficial in children with VM [34,35]. However, the American Academy of Neurology guidelines has expressed skepticism about giving pharmacotherapy as prophylaxis for a migraine in children and adolescents citing the lack of supportive evidence [36]. Currently, the first line of therapy for these children is non-pharmacological. Vestibular rehabilitation has been shown to be helpful in controlling the vertigo episodes in VM [37]. Non-pharmacological modalities like hydration, behavioral measures, sleep hygiene, exercises, balanced diet, avoiding triggering food items, cognitive behavioral therapy and biofeedback have been found to be helpful not only as the first-line therapy but also for preventing the migraine, as well as the VM [33,38].

2.3. Otitis media

Otitis media (OM) is nothing but inflammation of the middle ear. It can contribute to vertigo in children primarily via non-suppurative secretory otitis media (also known as middle ear effusion) or via acute or chronic suppurative otitis media. Majority of these children would also have hearing loss. Grace et al. first quantified the association of non-suppurative otitis media (NSOM) with disequilibrium among children in 1990 [39]. 22% of NSOM children (n = 154) had some sort of vestibular disturbance, and 85% of these symptomatic children had complete resolution with grommet insertion [39]. Objective tests in children with chronic middle ear effusion (n = 136), aged four to nine years, showed the abnormal vestibular results in 58% as compared to 4% in healthy children (n = 74) [40]. After the placement of ventilation tube, vestibular symptoms resolved in 96% of these patients [40]. Many other authors have reported the similar balance disturbance in children having otitis media with effusion, and also have shown symptomatic relief along with the normalization of vestibular tests by myringotomy and ventilation tube placement in almost all of the

affected children [41–43]. However, many of these children have an increased likelihood of developing dizziness in adulthood, compared to those children without NSOM [44].

Suppurative otitis media can lead to giddiness by affecting the ipsilateral labyrinthine function both in acute suppurative otitis media [45] or in chronic suppurative otitis media [46]. In chronic suppurative otitis media with cholesteatoma, the stapes destruction in children has been reported to be significantly more common than in adults [47]. However, as in adults, favorable outcomes, both terms of hearing as well as control of vertigo have been reported by complete removal of cholesteatoma matrix even in children [48].

2.4. Vestibular neuritis

Vestibular neuritis (VN) is among one of the common causes of giddiness in children and constitute 16% of all the giddy pediatric patients [17]. Commonly seen in children older than five years of age and adolescents [49]. History of preceding upper respiratory infection is seen in nearly half the children [50,51]. The superior vestibular nerve is thought to be commonly inflamed following a viral infection. Affected children would complain of sudden severe rotatory vertigo lasting from few hours to few days, associated with nausea and vomiting, but no hearing loss. On examination, they would demonstrate hypofunction of the affected labyrinthine canal, both clinically as well as in caloric test [17,49]. Pharmacotherapy with steroids and vestibular rehabilitation has been advocated even in children, whereas the role of antiviral therapy in VN remains controversial. However, the younger children have been shown to have complete recovery in canal functioning, compared to the adolescents [49]. The recovery of canal paresis in children has been shown to be in proportion to clinical (symptomatic) recovery [50], contrary to disproportionate recovery seen in adults [52]. Moreover, the recovery from canal paresis is more likely in children than in adults, and the overall prognosis of VN in children seems to be better than that in adults [51].

2.5. Trauma

Post-traumatic dizziness without the hearing loss in children could be due to a labyrinthine concussion, whiplash syndrome, basilar artery migraine, vertiginous seizures, or non-specific giddiness [53]. The temporal bone fracture would lead to inner ear disruption and may result in vestibular dysfunction as well as hearing loss, with or without facial nerve weakness and cerebrospinal fluid leak. Perilymphatic fistula can occur even without evident temporal bone fracture and is usually associated with fluctuating hearing loss [54,55]. Though the surgical repair of fistula can relieve vertigo completely, the hearing loss may not recover in these cases [54–56]. The other cause of post-traumatic vertigo with hearing loss could be ossicular disruption with or without tympanic membrane perforation [57]. Following trauma, children tend to exhibit abnormal results to vestibular tests in nearly half of the cases, even though the symptomatic children may be very less [58]. Children with congenital inner ear anomalies like Mondini's malformation, enlarged vestibular aqueduct, and genetic disorders like CHARGE syndrome are predisposed to have giddiness along with hearing loss even after a trivial trauma [19]. The prognosis of vestibular function recovery in post-traumatic cases is variable and unpredictable.

2.6. Meniere's disease

Meniere's disease (MD) is rare in children and constitute less than 3% of all MD cases [59]. Among all the causes of vertigo in children, MD is diagnosed in 1.5–4% of children [24,60,61]. First described in children by Crowe [62]. Etiopathologically, MD is attributed to the endolymphatic hydrops. Clinically, fluctuating hearing loss, episodic vertigo, ear fullness and tinnitus are the predominant symptoms. The audiometric examination may reveal low-frequency hearing loss [60].

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