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Review

Hearing impairment and osteogenesis imperfecta: Literature review

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ABSTRACT

The goal is to clarify the epidemiology of hearing loss in patients with osteogenesis imperfecta (OI), so as to improve management. A literature review analyzed data from 15 patient series. Hearing loss prevalence in OI varied widely, from 2% to 94.1%. Typically, hearing loss was conductive in young patients and sensorineural in older patients. Prevalence increased with age, but after 50 years the increase was slight, and seldom became total. Hearing loss was usually bilateral, but not necessarily symmetrical. There were no correlations between type of mutation (*COL1A1* or *COL1A2*), prevalence, type or severity of hearing loss, or age of symptom onset; there was intra-familial variability. There was also no correlation between mutated gene, type of mutation and auditory phenotype. Frequency, type and severity of hearing loss were unrelated to other clinical parameters. Hearing loss prevalence depended on type of OI: higher in type I and lower in type IV. Incidence of otitis media was higher in children with OI, related to the associated craniofacial dysmorphia. Hearing screening before 5 years of age with long-term follow-up are recommended.

1. Introduction

Osteogenesis imperfecta (OI) or Lobstein's disease is a form of congenital osteoporosis. Prevalence is between 1/10,000 and 1/20,000 in France [1].

In 90% of cases, OI implicates a heterozygous autosomal dominant mutation of *COL1A1* or *COL1A2* gene; these genes code for type-1 collagen chains α1 and α2, respectively. Collagen is the main component of the extracellular matrix in bone, tendon, sclera, and the auditory and cardiovascular systems. Type-1 collagen synthesis defect may be quantitative or qualitative, conditioning clinical severity [2,3].

In 1979, Sillence et al. published a classification in 4 phenotypes according to severity: moderate (type I), lethal (type II), severe (type III), and moderate-to-severe (type IV) [4]. Recent discovery of other mutations, notably autosomal recessive, extended the classification to I to XI [2,5,6]. There is phenotypic and genotypic variability. No correlations have been established between specific mutations and clinical signs [7,8].

The present update was based on a PubMed literature review of hearing loss epidemiology in OI for the period January 1, 1950 to March 30, 2018. Search was restricted to articles in French or English (Fig. 1).

2. Description

2.1. Temporal bone histology

Histologic study of the temporal bone in OI patients finds lesions due to type I collagen defect [9–13]: ossification defect, demineralization and otosclerotic lesions. Microfractures may be found in the malleus, stapes and otic capsule. Otosclerotic lesions may induce stapes footplate fixation to the oval window and obliteration of the round window. In OI, otosclerotic lesions are more extensive than in otospongiosis. These histologic abnormalities may concern all types of hearing loss: conductive, sensorineural and mixed.

2.2. Temporal bone radiologic aspects

CT finds progressive bone demineralization with age [14]. The bone labyrinth is thickened, narrowing the middle-ear cavity and covering the stapes footplate and round and oval windows [15]. Swinnen et al. reported that OI patients with conduction or mixed hearing loss showed lower trabecular volumetric bone mineral density than in normal hearing or pure sensorineural hearing loss [16].

Temporal bone CT in OI patients with hearing loss shows pathologic lesions with reduced bone density associated with sites of elevated metabolic activity on MRI [16,17]. The extent of the hypodense areas on CT corresponds to the type of hearing loss: conduction hearing loss is associated with lesions of the fissula

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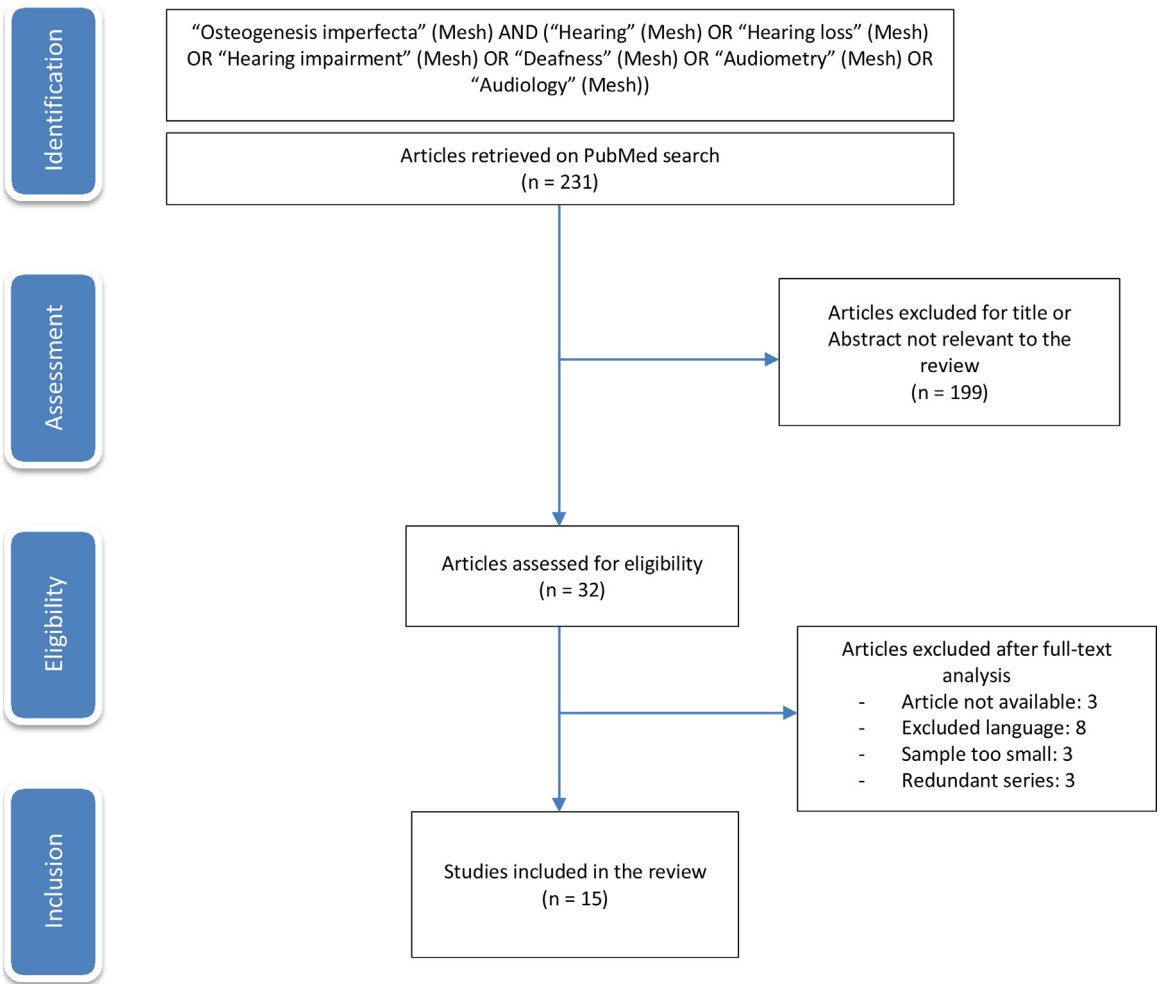

Fig. 1. Search strategy flowchart.

ante fenestram and round and oval windows; mixed hearing loss is associated with the same lesions plus retrofenestral lesions [17]. Hearing loss severity correlates positively with tumoral bone damage [17–19].

In the otic capsule, MRI finds pericochlear lesions with soft-tissue hypersignal and enhancement on contrast medium injection [14,20].

2.3. Hearing loss

2.3.1. General considerations

Hearing loss associated with OI was first reported by Adair-Dighton in 1912 [21]. In 1918, Van der Hoeve and de Kleyn further associated blue sclera in a triad that came to be named for them [22]. Patients may present conduction, sensorineural or mixed hearing loss, which is usually bilateral.

The prevalence of hearing loss in OI patients varies greatly between reports, from 2% to 94.1% (Table 1), due to differences in definition and in methodology: for example, some authors exclude cases of conduction hearing loss associated with seromucous otitis [30,31].

Rates of hearing loss in OI increase with age, plateauing around 40 years [30,32,37–39]. Typically, hearing loss is conductive in younger and sensorineural in older patients [7,8,23–35], although mixed or sensorineural hearing loss is found at all ages [8,25,26]. After 50 years of age, there is little increase in incidence and progression to total hearing loss is rare [28]. Hearing loss is most often mixed in older adults [16,26,31,33,39,30]. Severity varies with age and type of hearing loss [32].

Prevalence of hearing loss depends on the type of OI: highest in type I and lowest in type IV [28,30,37,38]. Hearing loss is more often sensorineural in type I than type IV [29].

There are no correlations between type of mutation (*COL1A1* or *COL1A2*) and prevalence, type or severity of hearing loss or age at onset; there is intra-familial variability [8,28,40]. There are also no correlations between the gene in question, type of mutation and auditory phenotype [7].

The frequency, type and severity of hearing loss are unrelated to other clinical features, such as number of fractures or blue sclera. Gender is unrelated to prevalence or type of OI [24,26].

Patients with conduction or mixed hearing loss show lower bone mineral density, with microfractures and bone remodeling implicated in footplate fixation, compared to patients with sensorineural hearing loss or no auditory involvement [16].

The type of mutation (*COL1A1* or *COL1A2*) is unrelated to progression of hearing loss, whether conductive, mixed or sensorineural. It may be a factor for progression in sensorineural but not mixed or conduction hearing loss [8]. Mixed or conduction hearing loss is more frequent than sensorineural hearing loss, with earlier onset and more rapid progression.

In mixed hearing loss, the sensorineural component, following initial conduction hearing loss, is due to pathologic bone remodeling and demineralization affecting inner ear structures. Pure sensorineural hearing loss is due to cochlear microfractures,

Table 1
Osteogenesis imperfecta and hearing loss.

Authors	Date	*n*	Definition of hearing loss	Patient population	Sex ratio	Mean age(years)	% hearing loss	% conduction hearing loss	% mixed hearing loss	% sensorineural hearing loss
Ting and Zachari, [23]	2012	36	Yes	Type I: 26 (72%) Type III: 1 (3%) Type IV: 3 (8%) Other: 6 (17%)	18:18	14.6 (3–19)	19.4[a] 11.1[b]	13.9[a] 5.6[b]	0[a] 0[b]	2.8[a] 2.8[b]
Swinnen et al., [24]	2012	182	Yes	Type I: 152 Type III: 4 Type IV: 26	88:94	30.2 (3–89)	52.2 44.5 bilateral 7.7 unilateral	8.5	37.8	11.6
Pillion and Shapir, [25]	2008	41	Yes	Type I. III. IV. V	15:26	26.5 (2–68)	62	21	29	12
Hartikka et al. [7]	2004	49	Yes	Type I: 36 (77%) Type III: 4 (8%) Type IV: 8 (16%) Other: 1 (2%)	19:30	36.4 (12–59)	65.3 49.0 bilateral 16.3 unilateral	18.4	28.6	13.3
Kuurila et al. [26]	2002	133	Yes	Type I: 96 (72%) Type III: 5 (4%) Type IV: 27 (20%) Other: 5 (4%)	50:83	40.8 (17–81)	57.9 47.4 bilateral 10.5 unilateral	4.5	17.3	15.1
Imani et al. [27]	2003	22	Yes	Type I: 12 (54.5%) Type III: 3 (14%) Type IV: 1 (4.5%) Other: 6 (27%)	8:14	9.6 (3–19)	77.3	63.6 54.5 SMO	0	13.6
Paterson et al. [28]	2001	1.394	No	Type I: 855 Type III: 207 Type IV 275 Other: 56	ND[c]	10 20 30 40 50 60 70	2 14 31 42 50 54 55	ND	ND	ND
Kuurila et al. [29]	2000	45	Yes	Type I: 28 (62%) Type III: 3 (7%) Type IV: 10 (22%) Other: 4 (9%)	19:26	10 (4–16)	6.7	4.4	0	2.2
Garretsen et al. [30]	1997	142	Yes	Type I with hypoacusis	ND	27 (4–87)	78	1	51	26
Stewart and O'Reilly, [31]	1989	56	Yes	OI[d] congenita: 10 (18%) OI tarda: 46 (82%)	24:32	10–60	58.5 49.1 bilateral 9.4 unilateral	30.4	34.8	26.1
Pedersen et al. [32]	1984	201	Yes	OI congenita: 43OI tarda: 156ND: 2	90:111	ND	50	12	27	11
Shapiro et al. [33]	1982	55	Yes	OI	ND	2–64 <30 years >30 years	 49 94.1	4	ND	 4; CHOI: 47 11; CHOI: 47
Cox and Simmons, [34]	1982	30	Yes	OI–5 families	13:17	20 (5–67)	36.7	20	13.3	3.3
Riedner et al. [35]	1980	70	Yes	13 OI families	ND	ND	41	20	32	38
Quisling et al. [36]	1979	160	No	Descendants of OI patient	ND	ND	47	18.2	3.6	14.5

[a] Before seromucous otitis.
[b] After seromucous otitis.
[c] ND = No Data.
[d] Osteogenesis imperfecta.

retrocochlear otosclerosis and atrophy of the stria vascularis and hair cells. The specific audiometric profile was named "CHOI" (CHaracteristic Osteogenesis Imperfecta) by Shapiro et al., who defined it as mild high-frequency sensorineural hearing loss with onset in the 6000–8000 Hz band, progressing to lower frequencies over time [28,33].

During adolescence, conduction hearing loss develops, due to abnormal bone remodeling affecting the oval window and causing footplate fixation.

In OI, hearing loss sets in earlier than in case of otospongiosis-related otosclerosis, and progresses toward mixed hearing loss related to more severe and extensive pericochlear involvement [13,38,30,40,41].

2.3.2. Pediatric series

Hearing loss incidence in pediatric series is variable: 25.4% for 4–19 year-olds according to Pedersen [32], 15.4% for 10–19 year-olds according to Stewart and O'Reilly [31], 54.9% for 4–19 year-olds according to Garretsen [30], and 38.5% for 10–19 year-olds according to Garretsen and Cremers [42] (these studies excluded seromucous otitis).

Imani et al. included hearing loss associated with seromucous otitis and reported 63.6% prevalence of conduction hearing loss, which was a much higher rate than in the above series [27].

Ting et al. reported a series of OI patients treated by bisphosphonates for at least 2 years [23]. Bisphosphonates are recommended in moderate-to-severe OI, inhibiting bone resorption [43]. After treatment for seromucous otitis, Ting et al. reported a much lower rate of hearing loss than in any previous series, and suggested that bisphosphonates hinder the progression of hearing loss in OI by reducing the risk of ossicle fracture and improving middle and inner ear structure ossification.

French national diagnostic and treatment guidelines for auditory screening in children with OI recommend performing an audiogram before the age of 5 years, repeated on average every 3 years or more frequently in case of onset of hearing loss.

2.4. Hearing loss management

Management depends on the type and severity of hearing loss.

2.4.1. Conduction and mixed hearing loss

2.4.1.1. Seromucous otitis. Incidence of seromucous otitis seems to be higher in children with OI, related to associated craniofacial dysmorphia [25,27]. Treatment is the same as in the general population.

2.4.1.2. Ossicular chain. The ossicular chain may be jeopardized in OI. In conduction and mixed hearing loss, surgical exploration may find fractures in thin, fragile ossicles, fixation of a thickened or obliterated footplate, and/or thickened hypervascularized mucosa [41,44–51].

Hearing aid rehabilitation should be proposed. Stapedotomy is an option in conduction hearing loss; surgical success is particularly related to preoperative Rinne test [46,47]: 88% of operated patients show a result less than 10 dB [47]. This is, however, less than in stapedotomy series in otospongiosis [39]. Hearing deterioration after stapedotomy is more frequent in OI, with rates as high as 9% of patients [42,52].

Implant bone anchorage is an option when stapedotomy is not performed or fails, although osteoporosis may impair stability [53–55]. A middle-ear implant is another possibility [48].

Patients with poor bone mineral density are at risk of conduction hearing loss [16]. Bisphosphonates are widely used in OI, to treat demineralization and reduce fracture rates [56,57]. Correcting osteoporosis in childhood could prevent hearing loss [23].

2.4.2. Sensorineural hearing loss

Hearing aid rehabilitation is indicated for mild to severe hearing loss. In more severe cases, cochlear implantation is an option [58–63]. Results are as good as in other hearing loss etiologies. Facial nerve stimulation by the intracochlear electrodes is a frequent complication, due to otic capsule demineralization [59,60,62–64].

3. Conclusion

Hearing loss is more frequent in OI than in the general population. It is usually bilateral, unrelated to gender, and with onset around the age of 20 years, although onset may also be as early as 4–5 years. Prevalence increases with age. Classically, it is initially conductive, progressing with age toward mixed or sensorineural hearing loss, although sensorineural hearing loss may also be found in younger subjects. Risk is greater in type I than type III or IV OI.

Very early screening and long-term follow-up are indispensable.

Disclosure of interest

The authors declare that they have no competing interest.

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