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REVIEW

Consensus on diagnosis and treatment of sudden hearing loss☆

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KEYWORDS
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Adverse effects

Abstract Idiopathic sudden sensorineural hearing loss is an unexplained unilateral hearing loss with onset over a period of less than 72 hours, without other known otological diseases.

We present a consensus on the diagnosis, treatment and follow-up of this disease, designed by AMORL, after a systematic review of the literature from 1966 to June 2010.

Diagnosis of sudden sensorineural hearing loss is based on mandatory otoscopy, acoumetry, tonal audiometry, speech audiometry, and tympanometry. After clinical diagnosis is settled, and before treatment is started, a full analysis should be done and an MRI should be requested later.

Treatment is based on systemic corticosteroids (orally in most cases), helped by intratympanic doses as rescue after treatment failures.

Follow-up should be done at day 7, with tonal and speech audiometries, and regularly at 15, 30, and 90 days after start of therapy, and after 12 months.

By consensus, results after treatment should be reported as absolute dBs recovered in pure tonal audiometry, as improvement in the recovery rate in unilateral cases, and as improvement in speech audiometry.

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PALABRAS CLAVE
Sordera súbita;
Acumetría;
Audiometría;
Corticoides;
Efectos secundarios

Consenso sobre el diagnóstico y tratamiento de la sordera súbita

Resumen La sordera súbita idiopática es aquella hipoacusia neurosensorial de inicio súbito, en menos de 72 horas, sin otros antecedentes otológicos previos.

Presentamos un consenso sobre el diagnóstico, tratamiento y seguimiento de la sordera súbita, surgido desde la Asociación Madrileña de ORL.

Mediante revisión sistemática de la literatura sobre sordera súbita, desde 1966 hasta junio de 2010, sobre los términos MESH "(acute or sudden) hearing loss", llegando a las siguientes sugerencias: en cuanto al diagnóstico, ante una sospecha clínica de sordera súbita, las pruebas diagnósticas que se consideran necesarias son: otoscopia, acumetría, audiometría tonal, audiometría verbal y timpanograma.

Una vez hecho el diagnóstico clínico de sordera súbita, antes de comenzar el tratamiento, se solicitará una batería analítica, debiendo completarse más tarde el estudio con RM de oído interno.

Se recomienda que el tratamiento de la sordera súbita esté basado fundamentalmente en los corticoides sistémicos, generalmente por vía oral, apoyados en los corticoides intratimpánicos como rescate.

Respecto al seguimiento, se realizará un control a la semana del inicio del mismo, incluyendo audiometría tonal y verbal, y a los 15, 30 y 90 días del diagnóstico, y 12 meses después.

Como consenso, el resultado de los tratamientos aplicados debería presentarse, tanto en cuanto a la cantidad de dBs recuperados en el umbral auditivo tonal como siguiendo la tasa de recuperación en los casos unilaterales, así como con parámetros de audiometría verbal.

Introduction

During the Third Congress of the Madrid ENT Association (AMORL), held in 2008, the Round Table on Sudden Deafness saw the need to create a consensus document on its diagnosis and treatment among hospitals in Madrid, as this is a controversial and relatively common condition. Moreover, various reviews on sudden deafness (SD) have recently been published in journals of general interest,[1,2] so it seems appropriate to review the subject, to propose unified criteria for this disease.

Regarding methodology, this consensus group, backed by AMORL, has conducted a systematic review of the literature published from 1966 to June 2010 on the terms MESH "(acute or sudden) hearing loss", collecting 4,180 articles in Spanish, English, German and French. It is striking that among so many publications, there are no clinical practice guidelines published on the subject, and very few randomized trials and meta-analysis. Through various meetings, the group evaluated the literature independently, highlighting the studies with the highest scientific quality, especially with regard to treatment.

Our study work attempted to collect and combine the experiences of various hospitals, and to systematically review the available scientific evidence to reach a common consensus about this pathology, in to the benefit of adult patients with sudden hearing loss.

Definition

By consensus, SD is considered as sensorineural or perceptual hearing loss cases with a sudden onset, within 72 hours, with a loss of over 30 dB, in at least 3 consecutive frequencies of tonal audiometry, without other prior otological history.[1-5] If a previous audiometry is available, this should be used as a baseline reference; if no previous audiometry is available, in unilateral cases the healthy contralateral ear should be used as a reference.

However, some studies also consider as "probable SD" cases of sensorineural or perceptual hearing loss that affect only 2 or 3 frequencies, with losses of 10-20 dB, appearing in less than 12 hours, including deafness appreciated upon waking in the morning, which recover rapidly.[6,7]

Epidemiology

Since its first description in 1944,[8] the incidence of SD has been increasing steadily over the years, and currently represents 1.2% of the cases at ENT Emergency Units.[1,3,9] Looking at the literature on its epidemiology, its impact can be established in 5-20 cases per 100,000 inhabitants per year.[1,6,9,10] Recently, other authors have reported an even higher incidence, of up to 160 cases per 100,000 inhabitants per year,[11,12] especially in cases where there are national registers of SD, as in Japan.

Presumably, these differences are due to different inclusion criteria defined for sudden deafness and to the underdiagnosis of this entity at the primary healthcare level.[1,5,7,12]

Aetiopathogenesis and physiopathology

Three possible causes have been postulated for idiopathic SD.[1-7,10,13,14]:

Viral theory

This is the most documented theory, both anatomically-pathologically, with post-mortem findings related to infection by type 1 herpes simplex virus,[15] and by the frequent history of upper respiratory tract catarrh or its higher incidence at certain times of the year.[16] However, no specific serological profiles[17] or response to common antiviral treatment such as acyclovir have been reported.[15,18-24]

Theory of alteration in inner ear microcirculation

Some studies have found a certain genetic prothrombotic susceptibility,[25] while others have shown a higher incidence of SD in patients with cardiovascular risk,[26-32] especially with mitral prolapse[33,34] or antiphospholipid syndrome.[35] On the other hand, SD has been reported after transient ischemic events in the inner ear, such as during general anesthesia,[36,37] or confirmed after an episode of intralabyrinthine haemorrhage, objectified by MRI imaging.[38-40] It has also been reported that the frequency of stroke is greater in the 5 years after having suffered SD.[41] These alterations in the microcirculation of the inner ear have been the basis of treatments such as administration of low molecular weight heparins,[42] plasmapheresis for the cleaning of plasma LDL,[43-49] the use of inhaled carbogen[50-52] or hyperbaric oxygen chambers[53-62] and the administration of prostaglandins such as PGE1,[52,63,64] with mixed results.

Theory of immune-mediated disease

This theory is supported by pathological studies, spontaneous recoveries and the response to treatment with steroids.[1-7,14,65,66] However, in some patients with SD, there is no evidence of impaired immunity and the clinical evolution is not always compatible with an autoimmune case.[67,68] Moreover, it is common for SD cases initially classified as idiopathic to be diagnosed as a specific autoimmune disorder with the passage of time.[69]

Conversely, although it would not be an idiopathic SD, the "theory of cochlear membrane rupture" has been described,[70,71] due to a possible perilymph fistula, appearing in connection with physical exercise, barotrauma or a Valsalva manoeuvre. This theory could justify the spontaneous recovery of some patients, and for some authors it determines the indication for early exploratory tympanostomy to seal the rupture.[72,73]

Diagnosis

Faced with a clinical suspicion of SD, and before considering a possible treatment, the diagnostic tests required are:

In primary healthcare and ENT emergency services, the two tests required are otoscopy and acoumetry:

Otoscopy should be normal in both ears; however, the finding of a wax plug does not exclude a possible SD. The plug should be removed and test whether hearing becomes normal.[1,5,13]

Acoumetry (tuning forks) will give us a sensorineural pattern: positive Rinne in the diseased ear (Figures 1A and 1B) and Weber lateralised to the healthy ear (Figure 1C), allowing us to rule out causes of SD due to middle ear diseases: otitis media with effusion, etc. with a transmission pattern (negative Rinne in diseased ear and Weber towards the diseased ear).[1,5,74] However, in cases of severe SD, cophosis, there can be a false negative Rinne (the patient does not hear the tuning fork at all).

In outpatient ENT consultations, in addition to confirming the normal otoscopy and sensorineural acoumetry, it is necessary to carry out a tonal and verbal audiometry and a tympanogram

Tonal audiometry will determine the pure tone audiometry (PTA), taking the average dB threshold at frequencies 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA, which must be greater than 30 dB in bone conduction to confirm the diagnosis of SD.

Verbal audiometry will assess verbal comprehension, determining the verbal reception threshold (VRT) and the maximum discrimination (DMax).[75]

With these tests, we may conduct an initial diagnosis, and thus begin treatment; in addition, they will serve as baseline data to evaluate the response.

The audiological examination may be selectively extended to include supraliminal tonal audiometry, otoacoustic emissions or auditory potentials (BAEP, ASSR) or to include vestibular, caloric, and VEMP tests.

It is advisable to supplement the diagnostic process with:

Stapedial reflex, including the Metz test, to rule out cochlear recruitment and Ménière's disease.[76]

Series of analyses,[66,68,77-79] whose extraction should be performed prior to treatment, including at least the following parameters: blood count, erythrocyte sedimentation rate (ESR), luetic serology (VDRL and FTAabs) and antinuclear antibodies (ANA), according to prior systematic reviews.[66,77] It may also be useful to request immune phenotype by CD4+ and CD8+ lymphocyte subpopulations, and the CD45RO+ and CD45RA+ isoforms.[66,80]

MRI of the inner ear with gadolinium to exclude retrocochlear pathology or show intracochlear haemorrhage.[81-86]

For a more extensive review on the diagnosis process for SD, we recommend reviewing the work of Chau et al[14] and Nosrati-Zarenoe et al.[83]

Differential diagnosis

Sudden deafness can be a symptom of many diseases, both as their debut form and during the course of their evolution. The most difficult part is determining the causal relationship in each individual patient.

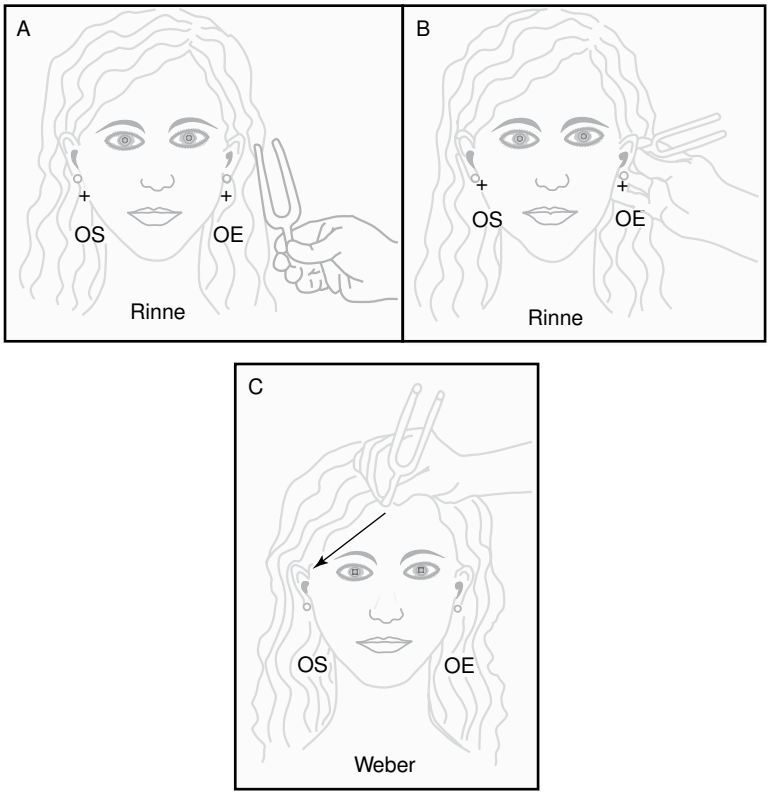

Figure 1 Acoumetry in sudden deafness: A: Rinne applying the tuning fork to the ear to explore the air pathway. B: Rinne applying the tuning fork on the mastoid bone to explore bone conduction. C: Weber, perceiving bone conduction to the healthy ear. OS: healthy ear, OE: diseased ear.

Table 1 Causes of sudden deafness

Cochlear
Inflammatory: virus, bacteria, spirochetes (syphilis)
Traumatic
Vascular
Haematological (anaemia, stroke, alterations of circulation)
Immune-mediated disease (Cogan, scleroderma, ulcerative colitis, sarcoidosis), vasculitis
Endolymphatic *hydrops*, including Ménière's disease
Metabolic diseases (diabetes)
Bone diseases of the otic capsule (metastases, myeloma, histiocytosis X)
Ototoxic agents
Retrocochlear
Meningitis
Multiple sclerosis
Friederich ataxia
Amyotrophic lateral sclerosis
Vogt-Koyanagi-Harada syndrome
Xeroderma pigmentosum
Posterior fossa tumours (acoustic neurinoma)
Central deafness
Idiopathic

In Table 1, we present the relationship of pathology and anatomical location,[1-7] with the aim of reducing the cases which are labelled as idiopathic SD.

It is compulsory to make a correct differential diagnosis between SD and other entities with a similar presentation, such as catarrhal ototubaritis, barotrauma, etc.[1,5] This requires an acoumetry and otoscopy.

Clinical forms

Among the presentation forms, we can find the following audiometric curves of sensorineural hearing loss (SNHL), with different prognostic implications[87] (Figure 2), since those affecting lower tones usually show a better response to treatment.

Moreover, there are three atypical forms of SD.[88]

Paediatric forms

In the event of traumatic injury, however slight, we must investigate a dilated vestibular aqueduct syndrome, through an ear CT scan.[89,90]

Bilateral forms

In these cases, it is common to find elevated ANA in the case of autoimmune forms, such as sarcoidosis, multiple sclerosis, Crohn's disease, Cogan syndrome, immune-mediated inner

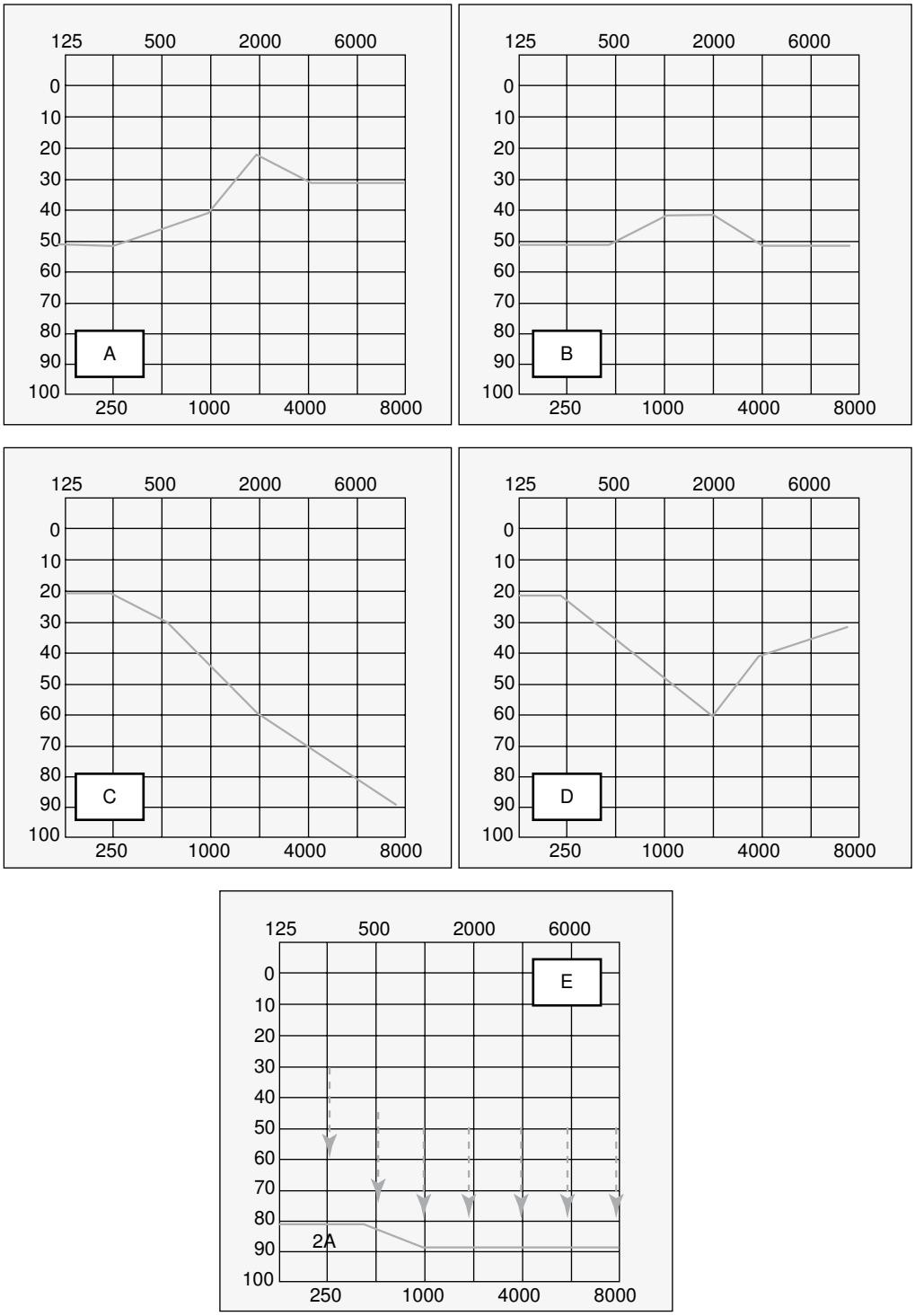

Figure 2 Forms of presentation of sudden deafness. A: Sudden deafness with low frequency SNHL (better prognosis). B: Sudden deafness with pantonal SNHL. C: Sudden deafness with SNHL at high frequencies. D: Sudden deafness with SNHL at medium frequencies. E: Sudden deafness with residual hearing (cophosis).

ear disease, etc. Other possible causes of bilateral SD are infectious (infectious mononucleosis, syphilis, meningitis, HIV infection, etc.), neoplastic (malignant lymphomatosis, carcinomatous meningitis, leukaemia, etc.) or vascular (intracranial aneurysm, hydrocephalus, stroke, periarteritis nodosa, history of spinal anaesthesia and so on).

Evolving forms

These are cases of SD as a presentation symptom of fluctuating sensorineural hearing loss or Ménière's disease, especially when it affects low frequencies.[83,91,92]

Table 2 Prognostic factors in sudden deafness

Prognosis	Audiometry		Symptoms			Start of treatment	Age
Worse	SNH in high and medium frequencies	Higher auditory affectation (and cophosis)	Vertigo	Tinnitus	Headache	Late	Elderly
Better	SNH in low and pantonal frequencies	Lower auditory affectation	Absent	Absent	Absent	Early	Young

However, when it affects mostly high tones, SD can be the first symptom of an acoustic neurinoma,[81,82] even when hearing is restored completely.

Prognosis

The natural history of SD is variable, since its causes are multiple. Some patients recover completely without medical intervention, usually during the first three days (spontaneous recovery) and generally do not see a doctor.[93-95] Others improve slowly over a period of 1 to 2 weeks, with reports of improvement or spontaneous recovery in up to 65%of cases in the most classical series of the 70s and 80s.[96-99] However, most patients do not recover their hearing without treatment, and up to 10%of patients experience a worsening of their hearing over time, despite the introduction of adequate treatment.[100]

Table 2 describes the signs of good and bad prognosis.[100-102]

The most commonly accepted factors of poor prognosis in SD in the literature are the following:

Advanced age of the patient.

Cardiovascular risk factors (arterial hypertension, etc.).

Exposure to noise trauma.

Intensity of the initial loss: the greater the degree of loss, the worse the prognosis for recovery of hearing function.

Type of audiometric curve: pantonal or high frequency deafness cases have a lower percentage of recovery.

Healthy ear auditory function: when the contralateral ear suffers a different pathology, the SD of the affected ear has a poorer prognosis for recovery.

Associated symptoms: the presence of vestibular symptoms represents a greater involvement of the entire labyrinth, and has a worse prognosis.

Promptly initiated treatment: the sooner the process is treated, the greater the chances of recovery.

Speed of onset of clinical improvement: the earlier the onset of the improvement of clinical symptoms, the better the functional outcome of the SD.

Treatment

Treatment of SD is very controversial, due to the absence of solid evidence to clearly endorse any of the options raised.[103] The doses employed are also very variable.[104,105]

Traditional general measures such as bed rest or diets with a restricted salt intake have not demonstrated effectiveness, so hospital admission to maintain bed rest is debatable. For this reason, there is no agreement on the need for a first phase of hospital treatment for 4 to 7 days, followed by outpatient treatment.[1,2]

Despite the excess of existing literature on SD, there are only a few randomized double-blind controlled trials, which have been reviewed by Cochrane[104] and summarised in a meta-analysis.[106] The most outstanding include the classic work of Wilson et al,[107] who compared oral steroids versus placebo in 1980, laying the foundation for their use (OR 3.22; 1.18-8.76). However, the group of Cinamon et al[50] found no significant effect with steroids (0.89; 0.1-7.86).

With regard to antiviral therapy, although antiviral agents should theoretically have a positive effect on SD, randomized clinical trials conducted by Stokross et al in 1998,[20] Tucci et al in 2002[21] and Westerlaken et al in 2003[23] were unable to find statistically-significant differences between antiviral agents and placebo.

Other widely used treatments, on the basis of vascular aetiology, such as vasodilators, carbogen or hyperbaric oxygen have been reviewed recently in a meta-analysis[55] and by Cochrane.[60] However, there were no findings of significant effectiveness in SD.

Consequently, having reviewed the literature, once SD has been diagnosed, this consensus group advises the following therapeutic scheme (Figure 3), based mainly on systemic corticosteroids,[50,51,102,107-113] supported by intratympanic corticosteroids as rescue, with a large volume of recent publications in this respect:[114-151]

- If the diagnosis was possible within 30 days of onset of symptoms, the treatment should be with oral steroids for 1 month, with 3 main options[152,153]:
  - Prednisone (Prednisone Alonga®, Dacortin®), 1 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 80 mg x 5 days, 60 mg x 5 days, 40 mg x 5 days, 20 mg x 5 days, 10 mg x 5 days, 5 mg x 5 days).
  - Methylprednisolone (Urbason®), 1 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 80 mg x 5 days, 60 mg x 5 days, 40 mg x 5 days, 20 mg x 5 days, 10 mg x 5 days, 5 mg x 5 days).
  - Deflazacort (Dezacor®, Zamene®), a similar decreasing pattern,[a] 1.5 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 120 mg x

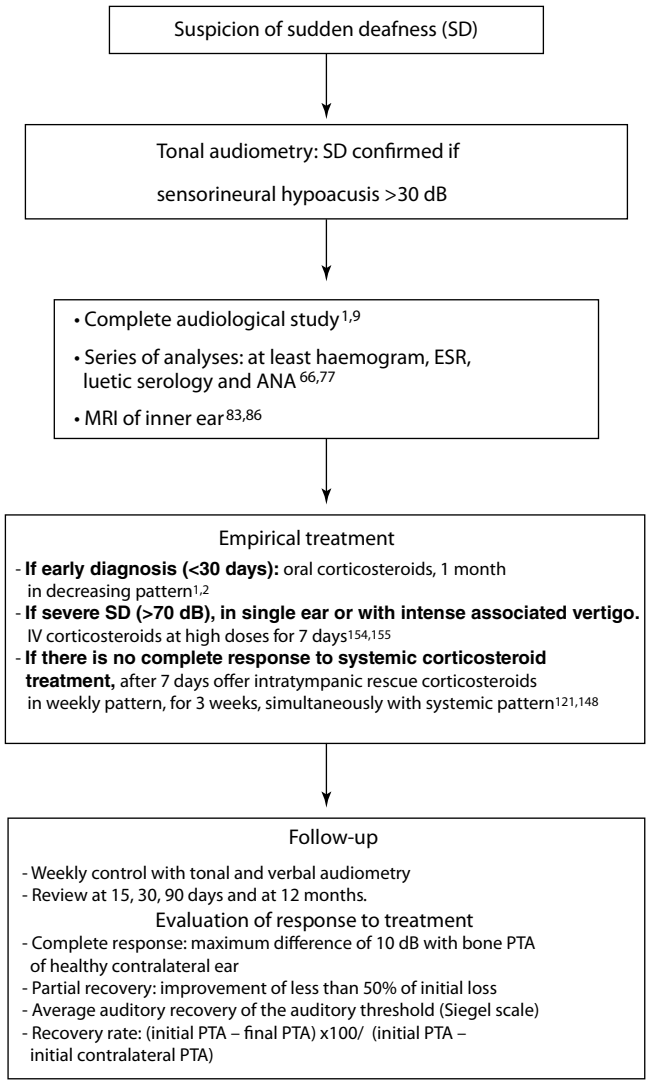

Figure 3 Diagnostic and therapeutic algorithm for sudden deafness.

5 days, 90 mg x 5 days, 60 mg x 5 days, 30 mg x 5 days, 15 mg x 5 days).

- If SD is severe (> 70 dB), in a single ear or with severe associated vertigo (suspected vestibular neuritis), treatment should be offered with intravenous corticosteroids for 7 days, in a day hospital regime or through hospital admission, with a dose of 500 mg methylprednisolone per day, slowly passing to one dose in serum in 30 minutes.[154,155] Subsequently, the oral regime described above should be reintroduced.
- If there is contraindication for the use of systemic corticosteroids, or if there is no response to treatment with systemic corticosteroids, oral or intravenous, after 7 days of its establishment, a rescue treatment with intratympanic corticosteroids should be offered,[121,148] with 1 dose weekly for 3 weeks in outpatient ENT consultation (and maintaining the decreasing oral pattern), with two commonly used guidelines (after topical anaesthesia with phenol on the tympanic membrane, with a no. 22 Abocath needle)[150]:
  - Methylprednisolone, 0.9 cc of a 40 mg vial, mixed with 1%lidocaine at 0.1 ml.
  – Dexamethasone, 0.9 cc of an 8 mg vial.

During systemic steroid treatment, either orally or intravenously, there should be gastro-duodenal prophylaxis with proton pump inhibitors (such as omeprazole) at doses of 40 mg/day, for 1 month.

In patients over 65 years, if glucocorticoid treatment is prolonged beyond 15 days, it will be necessary to associate vitamin D (800 IU/day) and calcium (800-1,000 mg/day) as a preventive regimen of bone loss and osteoporosis (requesting a bone density test is optional).[156,157]

Moreover, given the relative frequency of adverse effects associated to standard treatments for SD,[158-162] this consensus group suggests the use of specific informed consent, especially when it uses intravenous corticosteroids at high doses (validated informed consent, which is available from the authors).

In cases of suspected vascular aetiology (known cardiovascular risk factors, etc.), it may be associated with vasodilators,[42,163-167] such as nimodipine, intravenously (5-15 cc in 500 ml of saline solution, passing slowly, every 8 hours), or such as oral trimetazidine (especially if the patient is already in treatment for hypertension, 1 comp/8 h for 1 month), and then refer the patient to the Internal Medicine Department for evaluation and possible antiplatelet therapy.

- If the diagnosis is late, between 30 days and 90 days after the onset of symptoms, treatment should be with oral corticosteroids for 1 month, following the previous scheme.
- If the diagnosis is very late, more than 90 days from the onset of symptoms, treatment should be discussed individually.

Follow-up

Once the treatment has been established, there should be a control after the first week, including tonal and verbal audiometry, to assess tolerance to treatment and its results:

- If the patient has fully recovered hearing and presents acusia, the prescribed scheme should be followed (oral corticosteroids in decreasing doses for one month).
- If the patient has regained partial hearing, with an improvement of less than 15 dB, the prescribed scheme should be followed (oral corticosteroids in decreasing doses for one month), and simultaneous intratympanic treatment may be recommended individually.
- If hearing has deteriorated, intravenous rescue treatment will be recommended.

[a]Please note the equivalence between the two steroids in terms of anti-inflammatory effect: 4 mg methylprednisolone equals 6 mg deflazacort.

Table 3 Criteria for hearing improvement in SD, based on the average hearing threshold

Response	Average auditory recovery
Full recovery	Until obtaining a final PTA <25 dB, or independently of dB gained
Partial improvement	Improvement >15 dB, but final PTA between 25-45 dB
Slight improvement	Improvement >15 dB, but final PTA >45 dB
No improvement	Improvement <15 dB

(Taking the threshold average at 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA) according to Siegel.[172]

Table 4 Criteria for hearing improvement in SD, based on the average hearing threshold

Response	Average auditory recovery
Full recovery	Until obtaining a PTA <20 dB, or until the threshold of the contralateral healthy ear
Good improvement	Improvement >30 dB
Slight improvement	Improvement 10-30 dB
No improvement	Improvement <10 dB

(Taking the threshold average at 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA). Criteria established in 1981 by the Sudden Deafness Research Committee of the Ministry of Health and Welfare, Japan.[173-175]

Control should be performed, including tonal and verbal audiometry, at 15, 30 and 90 days of diagnosis.[167-170] However, to rule out autoimmune hearing loss or Ménière's disease, it would be advisable to monitor patients until 12 months after diagnosis, especially in cases of SD affecting low frequencies.[89,171]

Evaluation of results

After treatment, recovery would be complete if a maximum difference of 10 dB is reached with the bone PTA of the healthy contralateral ear. Partial response is considered when there is an improvement of less than 50% of the initial loss.

For most authors, successful treatment of SD is defined arbitrarily, as an average auditory recovery of the auditory threshold (taking the average of the threshold at 0.25, 0.5, 1, 2, 4, and 8kHz as PTA) of more than 30 dB, or following gradual schemes as proposed in the U.S. by Siegel in the 70s (Table 3),[172] by the Japanese Committee on Sudden Deafness in the 80s (Table 4),[173-175] or by the Swedish National Register.[176]

Other authors use more stringent criteria such as the recovery rate, described in publications in the 70s. This rate is established taking into account the degree of recovery of the diseased ear with respect to the healthy ear, so it only applies in cases with a normal contralateral hearing (<25 dB), according to the following ratio:

$$\frac{\text{Rate of recovery}}{\dfrac{\text{Initial PTA} - \text{Final PTA}}{\text{Initial PTA} - \text{Contralateral Initial PTA}}} \times 100$$

Moreover, it is essential to confirm the improvement in verbal comprehension through serial verbal audiometry, determining the verbal reception threshold (VRT) and the maximum discrimination (DMax or SDS).

As a consensus, the result of treatments applied should be presented in both forms, with respect to the dB recovered in PTA (Siegel scale), and following the recovery rate in unilateral cases, as well as with speech audiometry parameters such as VRT and DMax, as postulated in the most recent studies.[101,104,177]

Conclusions

Sudden deafness (SD) is a perceptual or sensorineural hearing loss with sudden onset, within 72 hours, with a loss of over 30 dB, in at least 3 consecutive frequencies in a tone audiometry, with no other previous otological history. It is an entity of increasing prevalence, affecting 5-20 cases per 100,000 inhabitants per year, and its diagnosis requires a greater involvement of the primary healthcare network.

Faced with a clinical suspicion of SD, and before considering possible treatment, diagnostic tests required in the field of primary healthcare and emergency ENT would be: normal otoscopy and acoumetry, showing a sensorineural pattern (positive Rinne in diseased ear and Weber lateralised to the healthy ear). In ENT outpatient consultations: it would also be necessary to obtain tonal audiometry, determine hearing threshold, administer verbal audiometry, find the verbal reception threshold and the maximum discrimination and perform a tympanogram.

Once the clinical diagnosis of SD has been carried out, before starting treatment; a series of analyses should be requested; their extraction should be performed prior to treatment and should include at least the following parameters: blood count, erythrocyte sedimentation rate, luetic serology and antinuclear antibodies. The study should be completed subsequently with an MRI scan of the inner ear.

Treatment of SD is very controversial; however, as consensus, once SD has been diagnosed, we can recommend treatment based primarily on systemic corticosteroids, usually taken orally, supported by intratympanic corticosteroids as rescue. Moreover, given the relative frequency of adverse effects associated with standard treatments of SD, this consensus group suggests the use of a specific informed consent.

With respect to follow-up, once the treatment has been established, it should be checked after one week, including tonal and verbal audiometry, to assess tolerance to treatment and its results, and at 15, 30 and 90 days of diagnosis. It would be advisable to monitor patients until 12 months after diagnosis.

As a consensus, the result of treatments applied to patients with SD should be presented both in terms of the number of dB recovered in the tonal auditory threshold and

Table 5 Sudden Hearing Loss Registry: initial data

Case	Initial
Hospital	
Date of SD	
Gender	
Date of birth	
Age	
General history	AHT, DM, etc.
ENT history	Single ear, otorrhea sequel
Side	Right, left
Recurrence	Yes/No
Tinnitus	Yes / No
Hyperacusis	Yes / No
Vertigo	Yes / No
Worst PTA	Arithmetical mean of frequencies
Best PTA	0.5, 1, 2, 4 y 8 KHz
Worst URV	
Best URV	
Worst DMax	
Best DMax	
Type of curve	Low/Pantonal/High/Medium/ Cophosis
Degree of deafness	Slight/Moderate/Severe

Table 6 Register of sudden deafness: evolution data

		Worst ear	Best ear
7 days	Worst PTA		
	Best PTA		
	Worst URV		
	Best URV		
	Worst DMax		
	Best DMax		
30 days	Worst PTA		
	Best PTA		
	Worst URV		
	Best URV		
	Worst DMax		
	Best DMax		
90 days	Worst PTA		
	Best PTA		
	Worst URV		
	Best URV		
	Worst DMax		
	Best DMax		

Recovery in mean dB and recovery rate.
Aetiological study: MRI, analyses, etc.

following the recovery rate in unilateral cases, as well as with verbal audiometry parameters.

Sudden deafness registry

Following the initiatives of other countries such as Sweden[176] or Japan,[173] it would be advisable to create a register of SD at our hospitals, acting as a unit within the Community of Madrid or in those communities deeming it appropriate.

To do this, there is a minimum set of data to be collected for each patient (Table 5, Table 6) and sent to the community register, by e-mail to SEORL (e-mail can be sent to the authors).

Conflict of interest

The authors declare no conflict of interest.

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