Guideline for the Diagnosis and Management of Dentin Hypersensitivity

Chinese Stomatological Association Expert Committee on Dentin Hypersensitivity

Editor Comment

Dentin hypersensitivity (DH) is a common problem in dentistry; however there is limited data on the prevalence of it in China. On the other hand, many dentists in clinical practice have little knowledge on the diagnosis and management of DH. In oral health services, DH is continually misdiagnosed, missed in diagnosis and treated inadequately. Therefore, an expert committee on DH was set up under the Chinese Stomatological Association. A cross-sectional study on the prevalence of DH in urban areas of China and a questionnaire study on dentists‘ perceptions of dentin hypersensitivity in China was conducted. Based on the results from the epidemiological studies, a guideline on the diagnosis and management of DH was developed. The aim was to improve the ability of dentists in China to diagnose and control dentin hypersensitivity in their clinical practices.

Definition of DH

Through comprehensive reviews on the literature, with full consideration given to the consensus-based recommendations for the diagnosis and management of dentin hypersensitivity published by the Canadian Advisory Board[1]; and the definition proposed by Holland and others[2], the expert committee accepted the definition of DH as a “short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical, which cannot be ascribed to any other form of dental defect or disease”.

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The characteristics of DH can be understood by dental practitioners based on the diagnosis, management and treatment of the condition. This means conditions that present symptoms mimicking DH must be ruled out. This definition provides challenges for dentists given that they must consider other potential causes for pain associated with tooth sensitivity. In the diagnosis of DH, a differential diagnosis is essential as there are several other probable causes of similar symptoms. The exposed dentin can only be observed under the microscope while other dental diseases with symptoms of sensitivity, such as tooth fracture, decay or marginal leakage after filling can be observed clearly in the clinic. Patients with DH usually experience a short, sharp pain in response to the cold (the most common trigger), touch, evaporation, osmosis or chemical stimuli. The DH symptoms are often intermittent and sometimes self-healed. Now, there are specific and effective methods on the management and control of DH, which has been applied in oral health care services; and is different from those for other oral diseases, such as tooth fracture and marginal leakage after filling.

Dentin morphology change in anatomy

The difference between sensitive and non-sensitive dentin observed under the scanning electron microscope depends on the number of exposed tubules and the diameter of tubules. The number of open tubules per unit area in sensitive dentin is eight times more than the number of tubules in non-sensitive dentin. Also, the average diameter of tubules in sensitive dentin is almost twice that of tubules in non-sensitive dentin[3,4]. According to Poiseuille’s law[5], which states that fluid flow is proportional to the fourth power of the radius; diameter differences alone would indicate that the fluid flow in tubules of sensitive dentin should be 16 times greater than that of fluid in non-sensitive dentin. In addition, sensitive dentin has a thinner smear layer or the smear layer is removed, leading to exposure and opening of dentinal tubules.

Three conditions are predisposing factors for the development of DH, they are: 1) dentin must become exposed (lesion localisation) through either loss of enamel or gingival recession, 2) the dentin tubules must be open to both the oral cavity and the pulp (lesion initiation) and 3) stimuli, which includes thermal, evaporative, tactile, osmotic or chemical stimuli.

Causes and mechanisms of DH

Causes of DH

The exposure of tubules of dentin is normally caused by abrasion, attrition, erosion and possibly abfraction or their co-effects. Bruxism caused by malocclusion is considered as a critical risk factor for tooth abrasion.

Tooth brushing with qualified toothbrushes and toothpastes, using the correct method appears to cause no wear to enamel, while abrasion by some toothpaste during tooth brushing occurred on exposed cementum. Tooth brushing immediately after the consumption of acidic food or drinks might remove softer dentin. Therefore, a combination of erosion and abrasion exacerbates wear of teeth, causing the loss of enamel and exposing dentine.

Enamel erosion is another critical factor that causes the opening of dentinal tubules, which is characterised by acid-mediated surface softening that, if unchecked, will progress and lead to the irreversible loss of surface tissue, potentially exposing the underlying dentine. Extrinsic acidic sources include acidic drinks and foods, while intrinsic acidic sources refer to gastric acid from the stomach coming into contact with the teeth when people suffer from diseases such as gastroesophageal reflux disease and excessive vomiting. Enamel is particularly sensitive to the effect of acid, therefore tooth brushing of acid-softened or eroded enamel has a marked abrasive effect.

Gingival recession is another factor in the exposure of dentin. With gingival recession, the thin layer of cementum becomes easily abrasive, which causes the excessive exposure of dentinal tubules. There are many possible causes for gingival recession, such as inadequate brushing, brushing with hard toothbrushes, overaggressive brushing, self-inflicted injury, periodontal diseases and improper periodontal treatment procedures.

Overall, DH is developed due to the exposure of dentinal tubules by attrition, abrasion, erosion and abfraction. The process is chronic and there is no characteristic symptom in the early stages. Most patients with DH have their own risk factors, although there are some iatrogenic factors, such as dental scaling and periodontal treatments.

Mechanisms of DH

There are several mechanisms of DH: the dentinal receptor mechanism, the odontoblast transducer mechanism and the hydrodynamic theory. The widely accepted hydrodynamic theory was proposed by Brännström. According to this theory, with the presence of lesions and the opening of dentinal tubules into the oral environment, the fluid flow within dentinal tubules is increased or changed directionally by thermal, tactile or chemical stimuli near the exposed surface of the tubules. This alteration would lead to the stimulation of the A-ð fibers surrounding the odontoblasts, causing the pain sensation.

Diagnosis of DH

Diagnosis

The diagnosis of DH is based on the patients' medical history and their self perception of pain. Patients with DH usually experience a short, sharp pain in response to cold, touch, evaporation, osmosis or chemical stimuli.

In the clinical setting, objective measures of pain from an air blast, thermal stimuli or probing are commonly employed. While all of these might not be capable of replicating all types of DH, the outcomes of therapeutic intervention are sometimes applied in diagnosis.

Differential diagnosis

By definition, DH is a diagnosis of exclusion. Therefore, conditions that present symptoms mimicking DH, such as tooth fracture, fractured restorations, marginal leakage after filling, dental caries, pulpitis and so on, must be ruled out. It is necessary to differentiate or exclude these diseases:

- Tooth fracture and marginal leakage after filling can be clearly observed through amplifying devices or dye infiltration.
- Abrasion, erosion, and abfraction can lead to the loss of enamel and cementum, the exposure of dentinal tubules and can cause DH. Restoration is needed for severe tooth structure defects due to abrasion. In these situations, dentin hypersensitivity can be diagnosed with the diagnosis of abrasion, erosion or wedge-shaped defects.

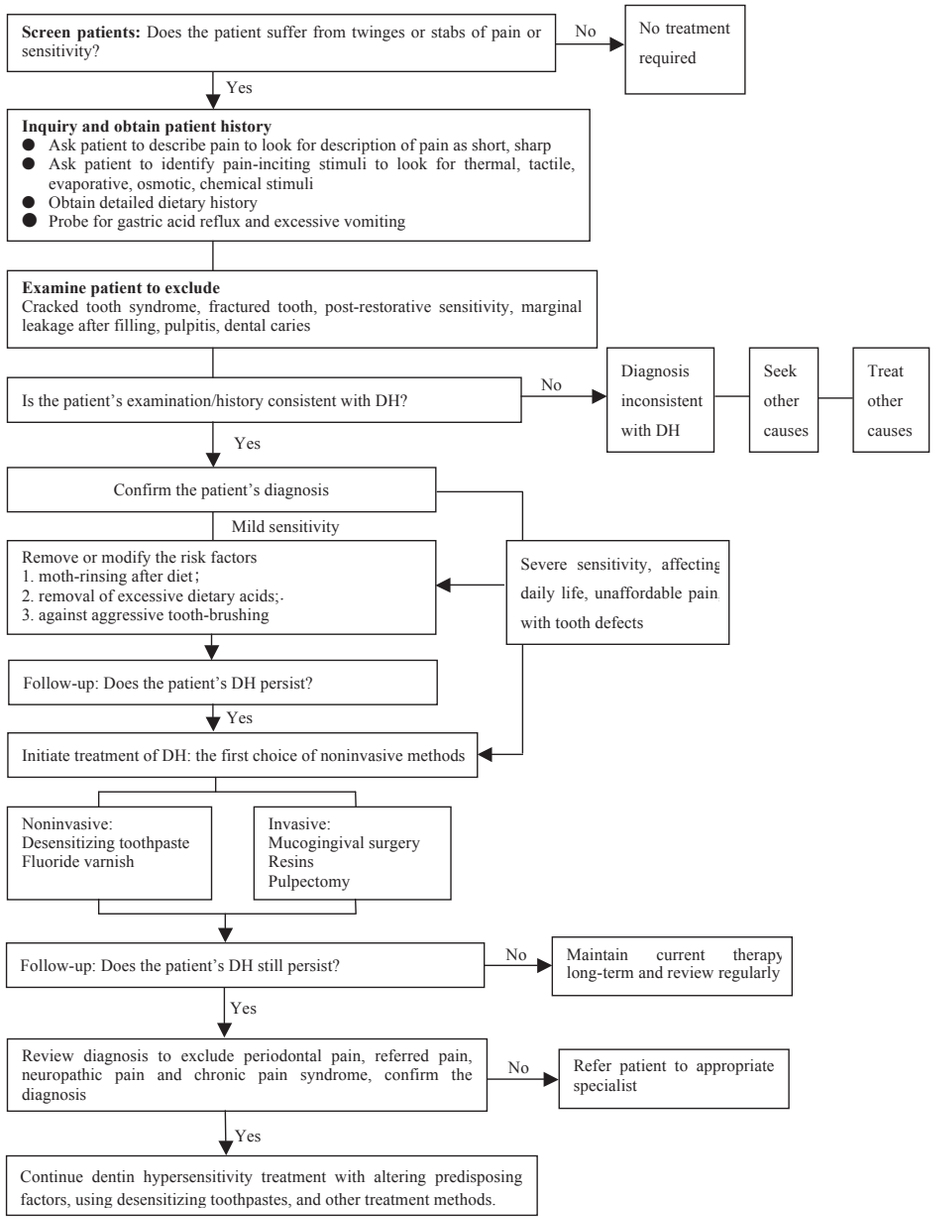

Fig 1 Algorithm for the diagnosis and management of dentin hypersensitivity.

- The symptom of DH is a short, sharp pain in short duration; it is not aggravated with time or stimulated by chewing pressure.

Prevention of DH

Predisposing factors and causes of DH should be removed or modified. It is suggested it is important: 1) to establish the habit of mouth-rinsing after diet; 2) to reduce the intake of acidic food or drinks; 3) not to brush teeth immediately after consuming acidic food and drinks, but to brush after 1 h; 4) to use qualified toothbrushes, with the correct method of tooth brushing, and not by brushing with excessive pressure; 5) to see a dentist whenever periodontal problems, bruxism, excessive tooth wear and associated conditions occur; 6) for those who suffer from diseases which lead to endogenous acids to see a doctor.

Treatment of DH

Treatment principles

The treatment principles are reducing fluid flow in the tubules and/or blocking the nerve response in the pulp[6]:

- Potassium nitrate or potassium chloride is commonly used as desensitising agents to interrupt neural activation and pain transmission.
- Fluid flow can be reduced by a variety of physical and chemical agents that induce a smear layer or block the tubules. Tubule-blocking agents include resins, glass-ionomer cements and bonding agents, strontium chloride or acetate, aluminum, potassium or ferric oxalates, silica- or calcium-containing materials and protein precipitants.

Treatment strategy and methods

The treatment methods of DH can be classified as invasive and non-invasive treatments. The priority consideration should be non-invasive treatment methods[7]:

- The first choice: daily use of desensitising toothpastes should be considered and recommended as a non-invasive, inexpensive, efficacious first line of treatment, which can be used by patients at home. Potassium compounds such as potassium nitrate or potassium chloride is commonly used as active ingredients in desensitising toothpastes.
- The treatment strategy of DH is based on the patient's complications:
  1) In the case of mild to moderate sensitivity, after initiating at-home tooth brushing with desensitising toothpastes, fluoride varnish with high concentrations of fluoride, glass-ionomer cements, resin sealants or bonding resins can be applied.
  2) In the case of severe sensitivity with manifestations of tooth defects, resin restorations can be applied.
- Others: DH might have occurred during some dental treatment procedures or after dental treatments; such as tooth scaling, root planning, periodontal surgery and tooth whitening. In order to reduce the patients' anxiety, detailed explanations, precautions and desensitising treatments should be provided before or after dental treatments.
- Follow-up: for patients with DH, follow-up is necessary. If DH still persists after using desensitising toothpastes for 4 to 8 weeks, the diagnosis should be reviewed in order to exclude other diseases (Fig 1). Invasive treatments such as mucogingival surgery, resin restorations or even pulpectomy should be considered.

Research needed

It is important to conduct long-term follow-up studies; randomised, placebo-controlled and double-blinded studies would be most preferable. The mechanisms of dentin hypersensitivity should be explored and more effective therapies should be developed.

Education

The committee suggested the Chinese Stomatological Association should organise continuous education projects on DH to improve the clinical capabilities of dentists. The education will focus on the risk factors, differentiate diagnosis and treatment of DH.

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