Dentine Hypersensitivity

It is described clinically as an: exaggerated response to the application of a non-noxious stimulus over the exposed dentine regardless of its location.

It is completely different than sensitivity that results from periodontal problems or periodontal treatment.

It is characterized by short, sharp pain arising from exposed dentine in response to stimuli, typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other dental defect or pathology.

Usually, the cold stimulus appears to be the strongest and causes the greatest problem to those troubled by "DH".

Dentine sensitivity or Dentine hypersensitivity are used interchangeably, however terms like root DS or root DH are currently used to describe a different entity which is related to periodontal disease or periodontal treatment.

Dentine Hypersensitivity is a distinct clinical entity and invites the clinicians to consider a differential diagnosis since other conditions may have similar symptoms but require different management strategies.

DD:

1. cracked tooth syndrome
2. fractured restorations
3. chipped or fractured teeth
4. dental caries
5. post-restorative sensitivity
6. atypical facial odontalgia
7. hypo-plastic enamel
8. congenitally open CEJ
9. improperly insulated metallic restoration

All of these can present in a similar way to Dentine Hypersensitivity.

Prevalence:

It depends on the type of study done and the population being studied, so it is a wide range, it ranges between >4% to <8% this variation is related to the variation in the study methodology and variation in clinical examination procedure, generally speaking studies that rely on questionnaires usually result in higher incidence of Dentine Hypersensitivity because people tend to exaggerate, and studies that rely on clinical examination result in lower incidence of Dentine Hypersensitivity less than 15%.

Dentine Hypersensitivity can affect patients at any age but mainly 20-50 years and the peak is at the end of 30s.

You will find higher incidence in females; this reflects their overall health awareness.

This Dentine Hypersensitivity is most commonly reported from Buccal and cervical zones of permanent teeth

Intraoral distribution mostly in premolars and canines > incisors and molars.

The anatomy of dentine will help understand this phenomenon

- Dentine is made up of straw-like tubules (DT).
- The tubules are filled with a fluid resembling the composition of plasma.
- When the dentinal tubules are patent a constant outward flow of fluids is observed.

This important in dentine bonding, especially when you use a single component bonding, there's always a way of contamination even under rubber dam, these contaminants are coming from the tubules, so ideally you should place the restoration quickly after applying the bonding agent especially with single-component bond.

Dentine is covered and protected by enamel and cementum, and itself it's a vital tissue (dentine pulp complex) as they're considered as one tissue and the vitality of dentine is related to the vitality of the pulp.

Although they're histologically different, their origin embryologically is the same, the ectomesenchyme. Pulp is integrally connected to dentine so any damage that affects one will affect the other and vise versa.

- Dentine is continually formed throughout life, by the odontoblasts which have their processes at the pulpal third of the dentinal tubules.
- Irregular atubular dentine or irritation dentine is produced at the pulp-dentine border when dentine is traumatized.
- Irritation dentine formation seems to be deficient in some individuals.
- The permeability of dentine is consequently reduced, due to the occlusion of the tubules.
- Precipitation of mineral deposits in the dentinal tubules and coverage of the exposed dentine surface with calculus can occlude the dentinal tubules and this is why patients who undergo periodontal treatment suffer from hypersensitivity and this is a different case because it will disappear in matter of days\weeks.

- The dental pulp is richly innervated by extensively branched nerve fibers, which form a dense network in the subodontoblast region, known as the plexus of Raschkow.
- Nerve fibers from the subodontoblast nerve plexus were found extending into dentinal tubules for 100μm remaining in close contact with the odontoblast process.
- Pain is the only sensation carried from the pulp to the conscious level.

Theories explaining dentine hypersensitivity:

1. **Direct innervations theory**
   The nerve endings that go from the pulp to the dentine (plexus of raschkow) extend into the surface of dentine and are directly stimulated when the dentine is exposed which initiates an action potential that makes the patient feel pain.
   Shortcomings of this theory:
   - Lack of evidence that the outer dentine is innervated
   - Developmental studies have shown that the plexus of Rashkow and intratubular nerves do not establish themselves until the tooth has erupted while newly erupted tooth is sensitive and the plexus is not established yet.
   - Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin.
2. **Odontoblast receptor theory**
   It's been said that free nerve endings and odontoblasts extend into the dentinal tubules, odontoblasts and their processes play a role in the evocation of "DH" whereby, the stimuli received by the dentine surface would be passed along to the odontoblasts where a synapse with a nerve fiber would transmit the sensory stimulus.
   Shortcomings of this theory:
   - Such a synaptic link between odontoblast processes and nerve fibers has never been detected.
   - Odontoblast processes extend only up to one third into the tubules and nerve structures are only observed up to 100μm into the tubules.
   - odontoblasts are matrix forming cells and hence they are not considered to be excitable cells.
   - HD is found to be present even when the odontoblast cell-layer is disturbed.
   - Pain-producing substances such as do not elicit pain when applied to dentine in contrast to when they are placed on the pulp.
   - Osmotic sugar solutions on the other hand, cause pain when applied to the dentine surface. This finding provides a link to the other theory of "DH".
3. **Hydrodynamic theory**
   This theory suggests that the pulpal nerve endings are indirectly stimulated by a change in fluid flow within the dentinal tubules. If the movement of fluids was large enough to cause a stimulus.

What causes the change in fluid flow within the dentinal tubules?

- Hypertonic solutions of sucrose extracted more fluid of the dentine than the normal constant fluid flow out of the patent tubules.
- So why does sugar cause pain but salt foods doesn't?
  Because the amount of sugar in sweets is very much more than the amount of salt in a salty food, so it's the concentration.
- A similar outward fluid flow was demonstrated when a cold stimulus was applied to the dentine surface.
- Stimuli, such as cold, which cause fluid flow away from the pulp produce more rapid and greater pulp nerve responses than those, such as heat, which cause an inward flow. This would explain the rapid and severe response to cold stimuli compared to the dull response to heat.
- It could also be related to the difference in temperature.
- An air blast causes evaporation of fluid from the outer part of the tubules allowing an increased outward fluid flow.
- When several hundred tubules are involved in such a process, a relatively large change in fluid flow is generated sufficiently large enough to create a movement of the pulpal tissue fluid in the subodontoblast region, activating the sensory nerve endings of the highly innervated plexus of Raschkow not if only one or two tubules are open.

  So for this process to happen we need 2 things:
  1. Large number of patent dentinal tubules.
  2. A stimulus that will cause the outward movement of the dentinal fluids.

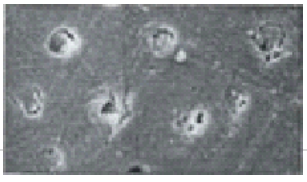
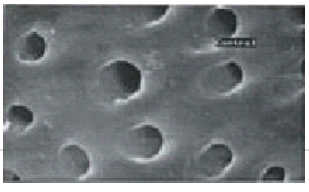
If we look at studies that explain this:

- There's a study by (Absi et al. 1987) that states that the amount of dentinal tubules patent to the pulp of dentine hypersensitive teeth was increased eight folds as compared to non-sensitive dentine.

The diameter of the open tubules of the sensitive teeth was approximately double that of non-sensitive teeth.

It is the width of the tubules that is particularly relevant since fluid flow is proportional to the fourth power of the tubule radius; doubling the tubule diameter results in 16-fold increase in the flow rate.

- Another study by Yoshiyama et al. (1989) found using SEM and TEM that 81% of the lumen of dentinal tubules in naturally de-sensitized areas were occluded with crystals and/or dense materials. In hypersensitive teeth, only 15% of the dentinal tubules were occluded.

Most evidence supports the hydrodynamic theory. Therefore, the apparent treatment of "DH" would be:

- The partial or total obliteration of the patent dentinal tubules or
- The desensitization of the pulpal nerve endings adjacent to the sub-odontoblast layer.

Predisposing factors:

First: **Enamel loss and tooth wear**

This happens as a result of loss of enamel and/or root denudation with exposure of underlying dentine. Enamel loss may result from:

- Attrition
- Abrasion (this is the most prevalent and causes the most DH because it's related to tooth brushing)
- Erosion: Extrinsic, Intrinsic
- Abfraction

Note regarding diet

- Dentine exposure to dietary fluids like red and white wine, citrus fruit juices, apple juice and yogurt for five minutes was sufficient to remove smear layer and open up large numbers of dentinal tubules.
- Loss of smear layer is known to enhance "DH".

Therefore, dietary information can be of value to detect excessive use of acid containing dietary fluids.

Second: gingival recession:

Gingival recession and what follows of root surface exposure allow more rapid and extensive exposure of dentinal tubules because the cementum layer overlying the root surface is thin and easily removed.

Clinical evidence indicates that gingival recession accounts for a much greater dentine area exposure than cervical enamel loss so it's more important and more significant than enamel loss.

- Periodontal disease.
- Periodontal treatment particularly surgical treatment.
- Chronic trauma; Tooth brushing.

Third: Pulpal inflammation:

- Some researchers suggested that there's an inflammatory response in the pulp tissue subjacent to hypersensitive dentine because there are open or patent dentinal tubules that will allow bacteria to penetrate into the pulp and cause inflammation.

- And some studies have shown some inflammation in areas of hypersensitive dentine and this inflammation can increase the sensitivity of nerve fibers and cause pain.
- Open DT allow bacterial and bacterial toxins to induce an inflammatory reaction.

Fourth: Plaque and tooth brushing:

It was suggested that tooth brushing behavior played a certain role in determining the distribution of the dentine hypersensitive teeth whereby,

- Canines and 1st premolars had the greatest recession and hypersensitivity, while revealing the lowest buccal plaque scores which is common sense because of brushing technique.
- DH is primarily found on the buccal surfaces, which have better accessibility to brushing.
- The distribution of plaque was lower on the left side, where hypersensitivity and recession were more pronounced reflecting an increased brushing on the left side in right handed individuals.
- A traumatic brushing is suggested to be a contributing factor to the occurrence of “DH”.

Clinical management of Dentine Hypersensitivity

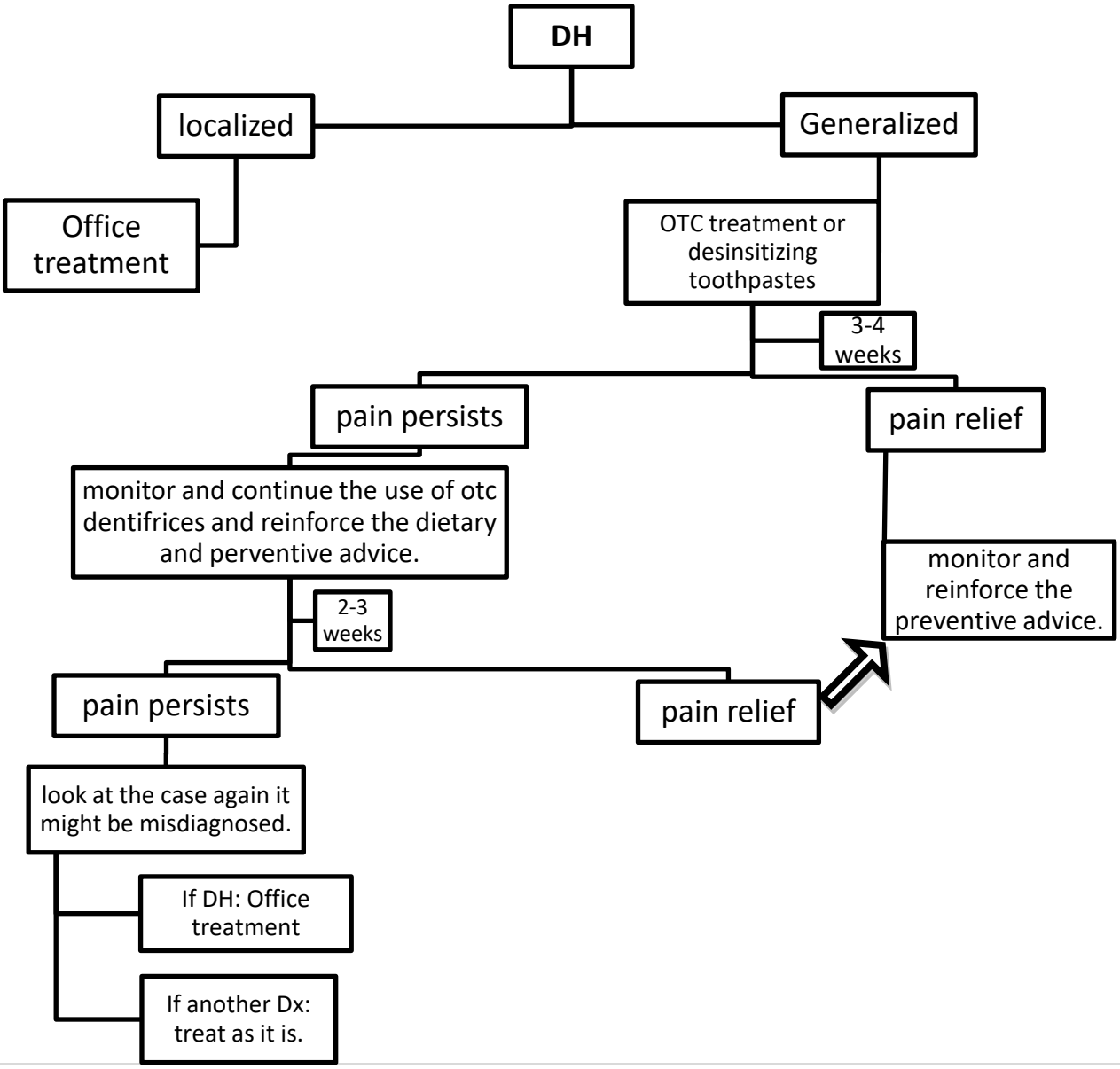
- Prevention is very important, and preventing this can only be debated by considering the probable aetiological factors.
- There is a need for greater professional and public awareness, through education, of the causes, effects and prevention of tooth wear and gingival recession.
- Management of patients suffering from “DH” should be based on a correct diagnosis by the dentist, who should be aware of other clinical conditions which are similar in their presenting features such as cracked tooth syndrome, fractured restorations, chipped teeth, dental caries, post-restorative sensitivity.
- Tooth brushing: Because incorrect tooth brushing appears to be an aetiological factor in “DH”, instructions in proper brushing technique can prevent further loss of dentine and the resulting “DH”.
  Should be avoided after consuming acidic foods and drinks since brushing in combination with acid decalcification of superficial tooth structure is capable of accelerating tooth structure loss and opening dentinal tubules causing more sensitivity.
  patients should be advised to rinse their mouths after consuming acidic drinks and wait at least 30 minutes before brushing.
- Dietary counseling is an important factor for the management of “DH”.
  Reduce the quantity and frequency of acid intake.
  Drink something neutral or alkaline after consuming acids such as water or milk.
  Avoid acids as a snack just before bedtime or during the night.

In case of intrinsic erosion, rinsing with liquid antacid or water after vomiting or regurgitation is advised.

Treatment:

If you diagnose the case with dentine hypersensitivity, follow these steps for treatment:

1. Remove the predisposing factor. And manage any local factors such as a fractured tooth.
2. Recommend a proper tooth brushing technique.
3. Initiate treatment by recommending a desensitizing agent at home or professionally at the office.
   So a patient walks into your clinic complaining of dentine hypersensitivity, first thing you do is check if there's any local factors you can manage, if not and the dx was confirmed as DH:



Agents for dentine hypersensitivity treatment were designed to be used:

- By the dentist in the office setting.
- By the patients themselves.

Apparently, both approaches have failed to be completely effective in all case

Just the use of desensitizing toothpaste and proper OHI and dietary counseling for 2 weeks has shown to improve the case in most of the patients, while some patients don't feel any improvement at all even after office treatment.

Desensitizing agents have been classified according to mode of action, in-office or at home (OTC) use, chemical or physical properties, reversible or irreversible procedures.

They include:

1. Dentifrices (toothpaste).
2. Gels.
3. Mouth rinses.
4. Resin cements
5. Bonding agents
6. Varnishes
7. Primers
8. Periodontal grafting
9. Laser
10. Rct
11. Extraction of the offending tooth
12. Gene therapy and hypnosis. (These are very extreme and are not used clinically)

Desensitizing substances can be classified by their mechanism of action, currently used agents act by:

- ✓ Blocking the dentinal tubules through coating mechanisms.
- ✓ Altering the tubular contents through coagulation, protein precipitation or creation of insoluble calcium complexes.
- ✓ Direct interference with sensory nerve activity.

<u>Desensitizing substances</u>

1. **Sodium fluoride**
   Very common, available as Duraphat; thick, viscous, yellow gel, rich in fluoride and you can apply it in office directly at the sensitive areas.

At home-use available as tooth paste or gels.

Mechanism of action:

- Precipitated fluoride compounds might block the DT mechanically
- Biomedical blocking of neural transmission by fluoride in the organic matrix of dentine.
- Professional application of sodium fluoride varnish (Duraphate) and the home use of strontium chloride tooth paste (Sensodyne) was more effective in reducing "DH" than the use of the desensitizing tooth paste alone.

2. **Potassium nitrate**
    - Incorporated in tooth pastes and used in gel forms.
    - Mechanism of action:
      Formation of a calcified barrier blocking the dentinal tubules.
3. **Stannous fluoride**
    - Incorporated in tooth pastes and used in gel forms.
    - Mechanism of action:
      Formation of a calcified barrier blocking the dentinal tubules.
4. **Sodium monofluorophosphate**
    - Incorporated in tooth pastes in combination with formalin, strontium acetate and with potassium nitrate.
    - Mechanism of action:
      Unclear mechanism of action; might interact with hydroxyapitite.
5. **Sodium citrate**
    - Not very effective.
    - Incorporated in tooth pastes.
6. **Oxalates**
    - An experimental material.
    - found in some toothpastes
    - Mechanism of action:
      Reduce dentine permeability (tubular occluding properties).
      Inhibitory effect of potassium on nerve activity.
7. **Resins and adhesives**
    - Resins have been reported to seal exposed dentinal tubules and to provide immediate blockage of transmission of pain-producing stimuli to pulpal nerves.
    - The use of acids to condition dentine prior to impregnation with resins is still controversial issue.
    - Glass ionomer cements have also been recommended to seal dentinal tubules. The material is hydrophilic, has good mechanical strength and is adhesive which allows its placement without mechanical tooth preparation.

- Some types of **chewing gum** contain Sodium fluoride.
- Some **mouth rinses** contain potassium citrate or potassium nitrate.
- **Periodontal grafting** is done if it's a localized problem, either it is accompanied by another problem like a localized gum recession and it's aesthetically unpleasing, or in some cases when it's an area where there's a local area with exposed dentine the use of a periodontal graft to cover it has been suggested. The mechanism of action is not really understood but it's very efficient. It is more applicable when you have localized areas only.
- When everything has failed to solve the problem **RCT** could be done,
- **Extraction** is also applicable in very severe cases.
- Few people have suggested **hypnosis and gene therapy** but this is very extreme.

"Far and away the best prize that life has to offer is the chance to work hard at work worth doing."

- Theodore Roosevelt

Good luck.

Seema Salam Daradkeh.