

# Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralisation (MIH)

## An EAPD Policy Document

N.A. Lygidakis, F. Wong, B. Jälevik, A-M.Vierrou, S. Alaluusua, I. Espelid

### Abstract

**BACKGROUND:** The European Academy of Paediatric Dentistry (EAPD) has long recognised the necessity of promoting further research and knowledge regarding the dental defect described as molar-incisor-hypomineralisation (MIH). Following the establishment by EAPD of the defect diagnostic criteria in 2003, the publication of various papers and a whole issue assigned to the defect in the European Archives of Paediatric Dentistry (2008), an Interim Seminar and Workshop on MIH was organized in Helsinki in 2009. **RESULT:** The outcome of this event is the present consensus paper on the prevalence, diagnosis, aetiology and treatment for children and adolescents presenting with MIH. A clear diagnostic proposal and a treatment decision-making guide are presented together with suggestions on aetiology and guidance for future research. **CONCLUSION:** MIH is an important clinical problem that often concerns both the general dental and specialist paediatric dentists; the present 'best clinical practice guidance' aims to further help clinicians dealing with the condition.

### Introduction

The present best clinical practice guidance was produced as a result of an Interim Seminar and Workshop concerning MIH organized by the European Academy of Paediatric Dentistry in Helsinki in May 2009. A comprehensive search of the literature was undertaken and presented by the invited speakers and revealed that there are only a limited number of evidence based research papers on this topic. From the small number of studies and their shortcomings, as presented by the speakers, it was evident that a 'Guidelines Diagram (drawing)' according to SIGN Methodology is impossible to be made at this time. However, it was agreed by the speakers and the Workshop experts and participants, that there is still a great need to produce a guide for clinicians dealing with children presenting with MIH. As a result, it was agreed that a 'Best Clinical Practice Guidance' should be developed for topics that achieved consensus in the Workshop; these were prevalence, diagnosis and treatment. Concerning aetiology only comments, questions and suggestions arising from the discussion group are included.

### Prevalence

MIH is frequent in many populations throughout the world. However, a wide variation in defect prevalence (2.4 – 40.2 %) has been reported. Cross comparison of the results of various studies is difficult because of the use of different indices and criteria, examination variability, methods of recording and different age groups.

For future studies it is recommended that a study group should be population based. The size of the study group should be large enough to be representative for the studied population. If the study group is composed of more than one age group, the frequency of MIH should be reported for each age group separately. Calibration procedures should be uniform and validated to ensure that there is conformity of the reported results. Finally it was agreed that the best age for a cross sectional study is 8 year of age. However, an even better study would be a longitudinal one with examination at the age of 6, 8, 10, 12 and perhaps 14 years of age, to evaluate to what degree other teeth are also affected, connections between the defects, and clinical variability of the defects over time.

### Terminology and Diagnosis

Regarding the term used at present 'molar-incisor-hypomineralisation', demarcated opacities of the same type as in MIH have been observed on second primary molars, tips of permanent canine cusps, second permanent molars and the premolars. Therefore, it is a risk that the denomination MIH is misleading resulting possibly to an under-estimation of the defect. For the moment however it is preferable to keep the term MIH as it has been established by the EAPD criteria [Weerheijm et al, 2003] and is well known. It is recommended that for any future studies to record mineralisation defects on all teeth in order to be able to evaluate if more teeth than molars and incisor are affected. If it will be shown that MIH is not principally restricted to molars and incisors we have the opportunity to revise the name.

The following diagnostic criteria and clinical appearance of the defects have been agreed upon:

*Permanent first molars and incisors.* One to all four permanent first molars (FPM) shows hypomineralisation of the enamel. Simultaneously, the permanent incisors can be affected. To diagnose MIH, at least one FPM has to be affected. The

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Postal address: Dr N. A. Lygidakis. 2 Papadiamantopoulou Street, Athens 11528, Greece

Email: lygidakis@ath.forthnet.gr

defects can also be seen in second primary molars, incisors and the tip of the canines. Where there are more molars and incisors affected the more severe is the defect.

*Demarcated opacities.* The affected teeth show clearly demarcated opacities at the occlusal and buccal part of the crown. The defects vary in colour and size. The colour can be white, creamy or yellow to brownish. The defect can be negligible or comprise the major part of the crown. It is recommended that defects less than 1 mm are not to be reported.

*Enamel disintegration.* The degree of porosity of the hypomineralised opaque areas varies. Severely affected enamel subjected to masticatory forces soon breaks down, leading to unprotected dentine and rapid caries development.

*Atypical restorations.* FPM and incisors with restorations revealing similar extensions as MIH are recommended to be judged as affected.

*Tooth sensitivity.* The affected teeth may be reported by frequent as sensitive, ranging from a mild response to external stimuli to spontaneous hypersensitivity; these teeth are usually difficult to anaesthetize.

*Extracted teeth.* Extracted teeth can be defined as having MIH only in cases where there are notes in the records or demarcated opacities on the other FPM. Otherwise it is not possible to diagnose MIH.

*Recording the severity of the defects.* Severity should be recorded as *mild* or *severe* in order to help the clinician. In *mild* cases there are demarcated enamel opacities without enamel breakdown, occasional sensitivity to external stimuli e.g. air/water but not brushing and only mild aesthetic concerns on discolouration of the incisors. In *severe* cases there are demarcated enamel opacities with breakdown, caries, persistent/spontaneous hypersensitivity affecting function e.g. during brushing and finally strong aesthetic concerns that may have socio-psychological impact.

## Aetiology

### Comments and suggestions arising from the working group.

It is likely that MIH is not caused by one specific factor. Several harmful agents/conditions may act together and increase the risk of MIH occurring additively or even synergistically.

*Genetics.* At present it is not known whether there is a genetic component in the development of MIH that makes the individual more susceptible to the condition. From studies in rats it is now known that, for example, different strains of mice/rats have different susceptibility to develop fluorosis when exposed to the same levels of fluoride. In future there is a need to look more carefully into studies concerning twins. Comparison of twins, that are monozygotic (identical) or dizygotic (non-identical), may indicate the relative

importance of genetics. Clinically there should be suspicion concerning possible genetic components, as there remains a lack of evidence on this issue. Also statistically, there is a relatively high frequency of MIH in some child populations and twins are likely to have more problems in the neonatal period. In addition there is probably a lower prevalence of MIH in Asian children but there are no studies evaluating whether there is an ethnic component.

*Aetiological significance of the different localization and uneven distribution of enamel defects in teeth formed in the same time period.* There is no good explanation as to why enamel defects are more commonly found on the occlusal/buccal surface(s). Could it be that disturbing agents, such as amoxicillin, affect cells during a certain stage of development? Amoxicillin modifies immunological and inflammatory response of the host child in various illnesses. The altered response lasts longer than the actual antibiotic course. Change in the levels of certain growth factors expressed also by ameloblasts may interfere with enamel formation. There is also a question of whether there is a dosage/response relationship. Considering other potential toxic agents, as occurs in the use of chemotherapy, is the aetiology of MIH likely to be a question of dosage or duration of the effect during a critical time in tooth development?

Effects on developing ameloblasts may be important. A question arises as to whether ameloblasts never become mature cells when MIH occurs? There is also no clear answer to whether the enamel thickness of affected teeth is an important issue or not; studies using animal models might answer this question.

The susceptibility of individual children to illnesses at critical ages might explain why FPMs are more frequently affected than for example premolars or second primary molars. The same consideration might account for the different reactions and tolerance to medication at different ages. The order of which parts of FPMs develop defects (central-mesio-buccal-distal) might have an effect on susceptibility. But there is no clear answer why maxillary FPMs are more frequently affected than mandibular, if this is indeed true.

*Medical problems during pregnancy.* There is mostly circumstantial evidence for any relationship with MIH. An association can be found using combined variables, but not when just taking one of either of the problems into account. There is probably a synergy between various medical factors. Additionally there is need for more information regarding evidence accruing from special care patients. Pre-term children, when their ameloblasts are in a secretory stage, usually have hypoplasia and not hypomineralisation which is part of the maturation stage. From the present data available it is not possible to make a ranking list for which problems during pregnancy might be aetiological factors. However, the recently investigated effect of caesarean section vs. normal delivery should be further studied.

*The time span for the postnatal medical problems that may cause MIH.* Most of the tooth crown of a FPM is matured by the age of 3 years, but maybe changes can also occur after that. There is still the question whether there is a possibility for an aetiological agent to have an impact up to the age of 5 or even 6 years of age. Also there might be some cases where mineralisation starts before birth and others that start after birth. Events during the first year of life appear to be more important, however, as the early mineralisation stage of tooth development might be the one more susceptible to aetiological factors causing MIH. There is also some evidence that severity of MIH may correlate to the time of onset of the aetiological factor.

A further area of interest is the relationship of MIH to the condition in permanent incisors, usually in the maxilla. Reports on MIH, which only included assessment of permanent incisors, are not reliable with respect to the diagnosis of MIH because dental trauma could be a confounding factor for the aetiology.

*Ranking the importance of different postnatal aetiological factors.* Ranking the importance of different postnatal aetiological factors is not possible on the basis of the present scientific knowledge. Postnatal problems/diseases during the first year of life are clearly more common in children with MIH than in those without. However, the use of a common or uniform terminology is important here. Because of differences in terminology there are problems with the validity of existing reports. As an interested group, paediatric dental scientists have not yet decided how to measure and record both the related illnesses and the dental defects in the best way. To overcome these problems two independent investigators should be involved in any study and should always be calibrated and come to the same conclusion.

Concerning the possible postnatal aetiological factors, the following areas of investigation should be considered. The antibiotic amoxicillin has been shown, in an animal model, to disturb enamel formation. In humans however it is not possible to be sure if childhood illness/fever or the treatment itself is the causative factor or if both are involved. It is also known that chickenpox affects ectodermal cells. Fever also needs to be considered as it might have a synergistic effect with other conditions or aetiological factors.

In order to have definitive answers to the questions noted above, aetiological factors should be statistically evaluated in a more appropriate way, e.g. using logistic regression models. Finally it has been suggested to hypothesise that MIH enamel defects result from a multifactorial process going wrong, not as a single cause but rather a 'process approach'.

*Is there enough evidence so prescribers of antibiotics should be warned about a possible side – effect?* Potential prescribers of antibiotics should be made aware that there might be a problem here. Of course when there is a need for antibiotics

they should be prescribed. A concern is that there is about a 2-fold increase in risk of MIH if amoxicillin is used in the first year of life. However, based on archeological material it is known that MIH occurred long before amoxicillin was introduced, indicating that even if amoxicillin is involved, it explains only part of the MIH cases.

*What type of research is needed to explore more about the aetiology of MIH?* There are a number of areas of further research needed. These are:

- Studies on aetiology should always start with a hypothesis based on clinical findings and then these should be tested with laboratory studies. Animal studies and basic research is needed to test these hypotheses. Positive findings are, however, difficult to verify in clinical terms. More extensive testing is needed on teeth and more than one laboratory should be involved to confirm findings. Finally there is need for agreement on protocols for clinical and laboratory studies.
- There have been some reports for particular localized environmental agents to be related to MIH, but there is still lack of any clear correlation. These localized environmental issues are gaining enough interest at the moment, because consciousness over environmental concerns nowadays is increasing and the pharmaceutical industry is beginning to act differently.
- The pharmaceutical industries do not test the effect of drugs on tooth development; therefore it might be helpful to test new drugs using an animal model (in rodents) or cell cultures.
- Research studies looking at genetic factors on twins could further explore aetiology. In addition research should focus on whether socio-economic background and ethnicity is a factor in MIH aetiology.
- It would also help to look into older age groups, examining canines and even premolars. To achieve this proposal it might be advisable to look at, for example, 12-year-old children.
- Finally future studies should have prospective study designs and in case of evaluating treatment outcome they should use case control studies. A common framework, such as questionnaires, data sheets for collecting data, etc, should be used.

### Treatment approaches

The available treatment modalities for teeth with MIH are extensive, ranging from prevention, restoration, to extraction. The decision on which treatment should be used is complex and is dependent upon on a number of factors. The commonly identified factors are the severity of the condition, the patient's dental age and the child/parent's social background and expectation. A diagrammatic summary of possible factors interacting for each treatment modality according to the severity of the condition at a particular dental age, is shown

**Figure 1.** Diagrammatic summary of severity-age related to tooth defects for MIH.

		Dental Age		
		Early Mixed	Late mixed	Full permanent
Level of Severity	Mild	Prevention		
		Adhesive + sealant for restoration		
			Composite restoration	
		Microabrasion, bleach + sealant for anterior		
	Severe	Prevention & symptom control		
		Adhesive + sealant for posterior		
		Microabrasion, bleach + sealant for anterior		
		Glass ionomer restoration		
			Composite restoration	
		Performed metal crown		
		Orthodontic extraction		
			Cast restoration	

in Figure 1. For example, prevention is very important at an early developmental age because the defective tooth is more likely to have caries and post-eruptive breakdown due to its increased porosity. However, in later development stages, although it is still important, the tooth becomes more mature and if prevention works in an earlier phase of development and the tooth remains intact, the relative importance of prevention becomes less comparative to the necessity of restorative treatment. As the growth of a child is continuous, the dental age in Figure 1 is in reality a continuum rather than in discrete stages of development. The relative amplitude indicates the appropriateness of the particular treatment modality as the treatment of choice. Finally, this guide should be used in conjunction with other factors such as a child's behavioural management and presence of other anomalies.

**Prevention.** It is important and sensible to start approaching the affected children and their parents with appropriate dietary advice. Toothpaste with a fluoride level of at least 1,000 ppm F should be recommended [Willmot et al., 2008]. Recently, Casein Phosphopeptide-Amorphous Calcium Phosphate (CPP-ACP), which provides a super saturated environment of calcium and phosphate on enamel surface, has been shown to enhance re-mineralisation. Although still controversial as to clinical effectiveness, its recommendation in the form of toothpaste or sugar free chewing gum may benefit those patients who complain of mild pain to external stimuli [Shen et al., 2001, Azarpazhooch and Limeback, 2008]. For patients with spontaneous hypersensitivity, professional application of fluoride varnish (e.g. Duraphat 22,600ppm F) and possibly 0.4% stannous fluoride gel may be helpful.

This modality has great importance in early post-eruptive stage when the defective tooth is more vulnerable to breakdown and caries attack. In late post-eruptive stage, if it is still intact, the tooth should have matured and should only need routine maintenance of good oral hygiene without any additional measures.

**Adhesive and fissure sealant (FS) for the posterior teeth.** FS is an obvious choice to protect the affected, without breakdown permanent molar. However, poor retention rates cast doubt on its efficacy for MIH molars [Kotsanos et al. 2005]. Mathu-Muju and Wright [2006] suggested that a 60 second pre-treatment application with 5 % sodium hypochlorite may remove the surface enamel proteins to enhance etching pattern created by 35% phosphoric acid; there are however no clinical or laboratory studies to support this claim. A recent long-term clinical study has shown that higher retention rates could be achieved if a 5th generation bonding adhesive is applied prior to FS application [Lygidakis et al., 2009]. This may be due to deeper penetration of the adhesive into the porous MIH enamel because of its lower viscosity, and/or its ability to bind the residual enamel protein.

FS are important before breakdown occurs and when the tooth is fully erupted and moisture control is adequate. For

partially erupted molars with inadequate moisture control, glass ionomer cements (GIC) can be considered as an interim treatment option with a view of replacement with a resin-based FS, due to the poor retention rate, GIC. In late post-eruptive stage, FS may need to be re-applied due to wear. However, as the tooth would have matured, its efficacy is decreased.

**Microabrasion, bleach and sealant for anterior teeth.** Aesthetic concern is common for any child with MIH incisors. Yellow or brownish-yellow defects are of full thickness whilst those that are creamy-yellow or whitish-creamy are less porous and variable in depth [Jälevick and Noren, 2000]. As a result the former defects may occasionally respond to bleaching with carbamide peroxide [Fayle, 2003] and the latter to microabrasion with 18% hydrochloric acid or 37.5% phosphoric acid and abrasive paste [Wray and Welbury 2001; Wong and Winter 2002]. More pronounced enamel defects might be dealt with by combining the two methods [Sundfeld et al., 2007]. However, bleaching for young children may induce hypersensitivity, mucosal irritation and enamel surface alterations [Joiner 2006], whilst microabrasion may result in loss of enamel [Sapir and Shapira, 2007]. A recent etch-bleach-seal technique with acceptable clinical results has been suggested by Wright [2002] involving: a) 60 seconds etch with 37% phosphoric acid; b) bleach with 5% sodium hypochlorite for 5-10 min, c) re-etch and application of FS over the surface to occlude the porosities. The infiltration of the clear FS may be enough to change the reflective index of the defective enamel to create an acceptable appearance.

These modalities are important around the time of the late mixed dentition when patients usually start to express their concern on mild discolourations. This conservative approach should be used as the first line of treatment before more invasive treatment such as resin restorations/veneers or crowns that may create problems, resulting from the large pulp size and immature gingival contours in young incisors.

**Cavity Design.** Two empirical approaches to where the margins of the restoration should extend have been proposed: a) removal of all defective enamel is reached [William et al., 2006; Mathu-Maju and Wright, 2006]; and b) removal only the porous enamel, until resistance to the bur or to probe is felt [Lygidakis et al., 2003; Fayle, 2003]. The first approach provides sound enamel for bonding but excessive tooth tissue is removed. The second approach is less invasive, but the margins may have a high risk of breakdown due to defective bonding. Nevertheless, adhesive restorative design should be used in all cases as amalgam is not recommended because of its poor performance [Kotsanos et al., 2005; Mejare et al., 2005].

**Glass ionomer cement restorations.** This includes conventional GIC, Resin Modified GICs and Polyacid modified composite resins. These materials have adhesive capability

to both enamel and dentine. One main advantage is their long-term fluoride release and their hydrophilicity for usage in conditions with inadequate moisture control. However, because of their poorer mechanical properties, they are not recommended to be used in stress bearing areas in MIH molars but can only be used as an intermediate restoration. For cavities involving large areas of dentine, GIC has been proposed to be used as a sub-layer under the composite restoration [Mathu-Maju and Wright, 2006].

This modality of treatment is important in early post-eruptive stages because it can be used as an intermediate treatment in less-than-ideal conditions of moisture control. In late post-eruptive stage it might help as a sub-layer beneath composite restorations.

*Composite resin restorations.* Composite resin material was shown to have much longer-term stability compared with other restorative materials in MIH teeth, with a median survival rate of 5.2 years [Mejare et al, 2005] and a success rate of 74%-100% [Lygidakis et al., 2003; Kotsanos et al., 2005] during a 4-year follow-up period. Self-etching adhesive (SEA) was found to have better bond strength to MIH affected enamel than all-etch single-bottle adhesive (SBA) in a laboratory study [William et al., 2006]. This was attributed to the omission of rinsing, thus eliminating any interference of residual water on the bond and to the presence of both micromechanical and chemical bonds between hydroxyapatite and SEA. Alternatively the hydrophilic properties of acetone included in some other SBA systems, may play the same role for eliminating the residual water from the etched enamel surface [Lygidakis et al., 2009]. For incisors, composites can be used with opaque resin for direct veneers in deep lesions to achieve a more satisfactory aesthetic result [Fayle, 2003; Weerheijm, 2004].

This modality of treatment becomes more important as a child grows because of this material's proven survival rate. However, studies with longer follow-up are needed especially on comparison of marginal placement in cavity design.

*Preformed metal crowns (PMC).* These have been recommended previously as a treatment option to provide full coverage of defective molars [AAPD, 2008]. It prevents further tooth loss, controls sensitivity, establishes correct interproximal and proper occlusal contacts, is not costly and requires little time to prepare and insert [William et al., 2006]. High success rates were reported [Zagdwon et al., 2003; Kotsanos et al., 2005] with a follow-up period of 2 and 5 years respectively. However, severe reduction of dental tissues may be needed for the insertion of the crowns. It has been suggested that the 'Hall' technique for PMC [Innes et al. 2006] with no tooth reduction could be used, but there are no reports on its long term efficacy and greater occlusal problems might be expected.

This modality of treatment can be used from early to late post-eruptive stages for MIH molars with breakdown,

especially on those that do not have enough tooth structure to support composite restorations. However, clinical studies are only limited to short term and longer-term studies are now needed to investigate PMC efficacy.

*Cast Restoration.* This approach includes adhesive metal copings, full coverage metal or tooth-coloured crowns for molars and porcelain veneers or crowns for incisors. Generally, for young children full coverage crowns are not recommended because of the large pulp size, short crown height, and difficulties in obtaining a good impression for subgingival crown margins [Koch and Garcia-Godoy, 2000]. Adhesive metal copings, usually made of nickel chrome alloy, are less destructive and have good short-term success rate over 2 years follow-up [Zagdwon et al. 2003]. Porcelain veneers for incisors could give good aesthetic results but should only be used in mature teeth. They are not recommended for teeth in early post-eruptive stage because of the continuous eruption exposing the crown margins, apart from the difficulties mentioned earlier.

This modality of treatment is not applicable for treatment for teeth in early post-eruptive stage because composite or PMC could give similar results and protection. In late post-eruptive stage, the cast restoration has the potential of being more durable but there is no long-term study to prove it.

*Extraction and orthodontic management.* Any extraction of a FPM should only be carried out with due consideration of the possible orthodontic complications. If the orthodontic condition were favourable, the ideal dental age for extracting the defective FPM would be 8.5-9 years of age [Williams and Gowans, 2003] in order to allow the second permanent molars to drift into the FPM position establishing an acceptable occlusion [Jälevick and Moller, 2007]. However, later extraction at the age of 10.5 years could also give acceptable results [Mejare et al., 2005]. When a lower FPM is extracted, compensating extraction of the upper FPM should be considered to allow mesial tilting of the second permanent molar; similarly a balancing extraction of the contralateral molar/premolar for avoiding a middle line shift should be investigated, particularly in crowded cases [Williams and Gowans, 2003].

This modality of treatment should be considered in late mixed dentition when radiographically the second premolar is in the crypt of the second primary molar and the second permanent molar's bifurcation starts to form. Too early extraction will result the second premolar drifting distally, inhibiting the second permanent molar's eruption into the FPM's space. Late extraction has less chance for spontaneous closure, resulting in excess residual space between the second premolar and second permanent molar, especially in the lower jaw.

**Children with MIH.**

Undoubtable a holistic approach should be taken into account formulating a treatment plan. It has been shown that children with MIH receive much more dental treatment that unaffected children [Jälevick and Klingberg, 2002, Kotsanos et al., 2005]. The porous exposed subsurface enamel and the dentine may promote bacterial penetration into the dentine resulting in chronic inflammation of the pulp, and difficulties in obtaining adequate local analgesia [Jälevick and Klingberg, 2002; Fagrell et al 2008]. Thus the child may be more anxious about treatment, needing considerable behavioural management. Hence, ideal treatment options may not be possible and alternative treatment plans may be needed.

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- I. Prevalence and diagnosis:** Speaker: B. Jälevik, Moderator: A-M. Vierrou, Invited experts: E. Berdouses (Greece), P. Fleming (Ireland), I. Skrinjaric (Croatia), U. Schiffner (Germany), L. Kiselnikova (Russia), R. Kosem (Slovenia), T. Leisebach (Switzerland), B. Kargul (Turkey), K. Jans (Belgium), R. Wogelius (Denmark), C. Tardieu (France), M. Elfrink (Netherlands), M. Raadal (Norway), E. Paiva (Portugal), R. Luca (Romania).
- II. Aetiology:** Speaker: S. Alalussua, Moderator: I. Espelid, experts: K.J. Toumba (UK), L. Esmark (Denmark), S. Pizzi (Italy), V. Zivojinovic (Serbia), G. Klingberg (Sweden), I. Cukovic-Bagic (Croatia), C. Butz (UK), J. Kühnisch (Germany), K. Gerreth (Poland), S. Sokolova (Russia), J. Fearne (UK).
- III. Treatment:** Speaker: N. Lygidakis, Moderator: F. Wong, experts: N. Krämer (Germany), M. Curzon (UK), R. Welbury (UK), W. Fenlon (Ireland), K. Emerich (Poland), L.Ferreira (Portugal), K. Kavvadia (Greece), E. O'Sullivan (UK), H. Alapulli (Finland), M. Pantelidou (Cyprus), K. Arapostathis (Greece), A. O'Connell (Ireland), A. Caputo (Italy), M. Ivanovic (Serbia), K. Kosir (Slovenia), C. Ullbro (Sweden), J. Roberts (UK), R. Steffen (Switzerland), V. Burkle (Austria).

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