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Research Article

Risk and protective factors associated with buccal soft tissue dehiscence around dental implants

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ABSTRACT

This review defines dehiscence, its classification and anatomical risk factor that makes implant undesirable. Many diagnostic methods to identify the risk/protective indicators of peri-implant soft tissue dehiscence (PISTD) in implants not suffering peri- implantitis.

1. INTRODUCTION

Dental implants have been used for centuries, with evidence tracing back more than 2,000 years to early civilizations in South and North America, as well as in regions of the Middle East and the Mediterranean [1]. The modern era of dental implantology, however, began around World War II, when Dr. Norman Goldberg, while serving in the army, explored the possibility of using metals for dental restoration, similar to how they were applied in replacing other body parts [1]. A major breakthrough came in 1957, when the Swedish orthopedic surgeon Per-Ingvar Brånemark discovered that bone tissue could integrate with titanium (Ti) and form a stable bond without rejection, marking a turning point in implant science [2][3]. Brånemark termed this phenomenon “osseointegration” and subsequently conducted numerous studies on both animal and human subjects [3]. Therefore, dental implants are considered the most reliable treatment option for replacing missing teeth, particularly in the esthetic zone. They offer superior functional and esthetic outcomes compared to other available treatment approaches [4]

Although dental implant placement can be associated with several complications, The success of implant therapy is assessed through the implant’s functional performance over time as well as the esthetic result and its long-term stability. Similar to native teeth, the stability of the peri-implant soft tissues plays a key role in achieving a satisfactory esthetic result [5]. A common challenge in this regard is the occurrence of soft-tissue dehiscence on the buccal surface, frequently observed following implant restorations [6]. Dehiscence is defined as the apical shifting of the peri-implant mucosal margin. Several terms have been used to describe this shift, including mucosal recession, midfacial recession, soft-tissue recession, and mucosal dehiscence. In contrast, gingival recession refers to the displacement of the gingival margin in an apical direction, extending beyond the cemento-enamel junction [7][8].

Unlike natural teeth, dental implants lack a standard anatomical reference point for Establishing the standard position of the mid-buccal mucosa, which has led to inconsistent definitions among clinicians. To evaluate soft-tissue dehiscence, A considerable number of authors have utilized the mucosal margin during final prosthesis delivery, as well as the implant or abutment surface becomes exposed and is considered as reference point.

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However, such approaches may lack accuracy as they disregard the gingival margin of neighboring or contralateral teeth. This can result in implant-supported crowns that appear excessively long and create an imbalance in the soft-tissue architecture compared to neighboring teeth, an issue highlighted in recent consensus recommendations for upcoming research [9].

Soft tissue dehiscence peri-implants can be characterized as a downward migration of the soft tissue margin of an Implant-retained prosthesis compared to the neighboring natural tooth, potentially involving exposure of the metallic implant surface. Unlike gingival recession in natural dentition, which has established classification systems that aid in treatment prognosis [10], predicting treatment outcomes for peri-implant soft-tissue dehiscence remains challenging. Compared to gingival recession, knowledge regarding its diagnosis and management around implants is still limited [11].

2. DEHISCENCE

Facial apical migration of peri-implant soft tissue has been termed mid-facial recession, mucosal recession, dehiscence, soft-tissue deficiency, or soft tissue defect. These Challenges may present as visible mucosal recession (apical displacement of the peri-implant mucosal margin), A faint grayish discoloration apparent transmucosally, and/or differences in the longitudinal measurement of the implant-supported restoration versus the adjacent dentition, may be observed. The term peri-implant soft tissue dehiscence/deficiency (PSTD) is regarded as the most accurate descriptor for these clinical presentations[12].

2.1 Biological width around implant:

The theory of biological width in natural teeth was first introduced in 1961 [13]. It was described as a 2 mm dimension consisting of approximately 0.97 mm of junctional epithelium and 1.07 mm of connective tissue, measured between the base of the gingival sulcus and the alveolar crest [14]. The junctional epithelium is a non-keratinized stratified squamous epithelium, while the connective tissue is primarily composed of transseptal and dentogingival fibers, mainly collagenous in nature. These collagen fibers include dentoperiosteal and circular fibers, which insert into both the radicular cementum and alveolar bone. The gingival segment of the dento-gingival junction adheres to the tooth surface via the basement membrane zone and hemidesmosomal connections [15]. The biological width serves an essential protective role, acting as a barrier that shields the tooth from external stimuli. In 1991, Berglundh et al. reported a similar phenomenon surrounding dental implants, suggesting the existence of a biological width in implant dentistry [16]. However, unlike the biological width around natural teeth, that around implants differs in its concept, development, remodeling, dimensions, structure, and function. In implant sites, biological width is generally described as a 3–4 mm zone, extending from the top of the peri-implant mucosa to the first point of bone-to-implant contact. Its structure includes the sulcular epithelium, junctional epithelium, and the connective tissue situated between the epithelium and the bone-implant interface. The basement membrane zone also exerts a critical function in maintaining molecular integrity at this interface. Functionally, the biological width acts as a biological barrier, contributing to the stability and reconstruction of peri-implant soft and hard tissues and influencing clinical outcomes in implant therapy. Numerous studies have examined its concept, development, structure, remodeling process, dimensional characteristics, and clinical significance. (As shown in the figure 1)

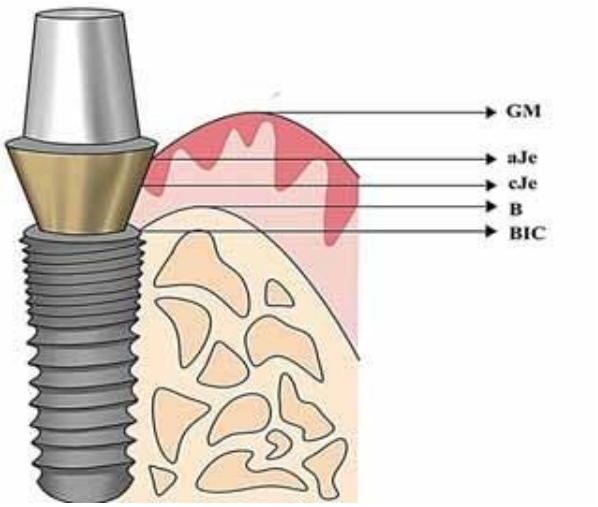

Fig. 1. Schematic of the biological width around a dental implant. GM: gingival margin; cJE: coronal junctional epithelium; aJE: apical junctional epithelium; B: top of the bony crest; BIC: first bone-to-implant contact. Sulcular epithelium: GM–cJE; Junctional epithelium: cJE–aJE; Connective tissue: aJE–BIC; Biological width: GM–BIC.

2.2 Classification of Dehiscence:

Zucchelli et al. (2019) proposed a classification system for peri-implant soft tissue dehiscence (PSTD) [17]. The system is according to the position of soft tissue margin relative to marginal gingiva of the adjacent natural tooth and the profile of the implant-supported crown:

- Class I:
  The soft tissue margin is positioned esthetically, in alignment with gingival margin of the adjacent native tooth. In such cases, the underlying abutment or implant may be discernible via the mucosa, and/or there may be a deficiency in keratinized tissue or mucosal thickness (figures 2A–C).
- Class II:
  The soft tissue margin is positioned toward the apex relative to the optimal gingival level of the corresponding native tooth. Nevertheless, the contour of the implant-supported restoration stays within (more palatally) the hypothetical curve joining the profiles of the Neighboring teeth at the level of the soft tissue margin (figures 2D–F).
- Class III and IV:
  In both categories, the soft tissue margin is situated toward the apex relative to the optimal gingival level, and the profile of the implant restoration lies beyond (more facially) the hypothetical curve connecting the adjacent teeth. Under such circumstances, the crown of implant must remove. In Class III, the implant head is located within (more palatally or aligned with) the straight hypothetical line joining the profiles of the Neighboring teeth at gingival margin level (figure 3). In Class IV, the implant head is positioned beyond (more facial to) this imaginary line (figure 4).
  Additionally, each class (except Class I, where subgroup "c" cannot be detected clinically) can be further Categorized according to the height of the interdental papilla:
  1. Both papillae tips are ≥ 3 mm coronally to the optimal soft tissue margin of the implant-supported crown (figure 5A).
  2. Minimum of one papilla tip is < 3 mm coronally to the optimal soft tissue margin (figure 5B).
  3. Minimum of one papilla tip is at the same level or apically to the optimal soft tissue margin (figure 5C).

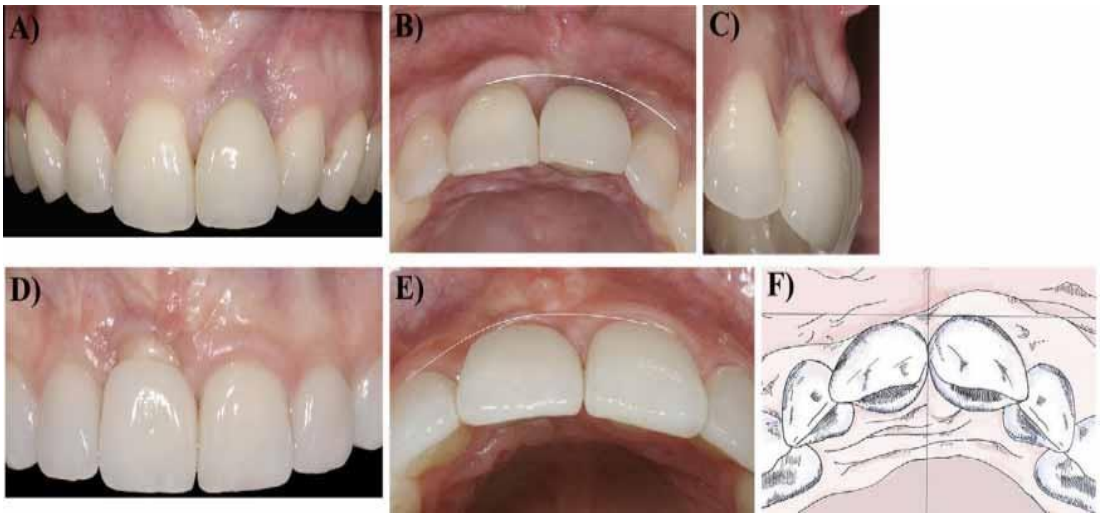

Fig. 2. PSTD Class I A, frontal; B, occlusal; C, lateral views show an esthetically correct soft tissue margin aligned with the natural tooth, though the abutment or implant color is shown via the mucosa. PSTD Class II D, frontal; E, occlusal; F, schematic features a soft tissue margin positioned apically relative to the optimal gingival margin, with the crown profile within the imaginary curve connecting neighboring teeth at the soft tissue margin height.

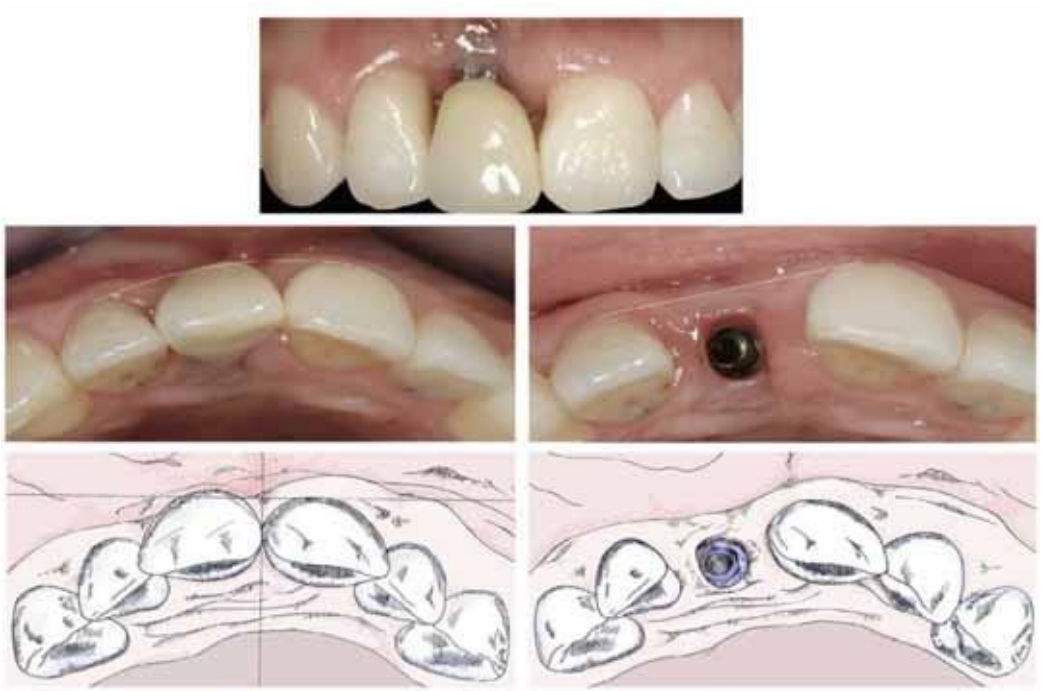

Fig. 3. Frontal (top), occlusal (middle), and schematic views (bottom, before and after crown removal) of PSTD Class III. This class is defined by a soft tissue margin positioned apically relative to the ideal gingival margin, with the crown profile extending beyond the imaginary curve connecting neighboring teeth at the soft tissue margin height. After crown removal, the implant head aligns within the straight imaginary line joining the profiles of the neighboring teeth at the gingival margin height .

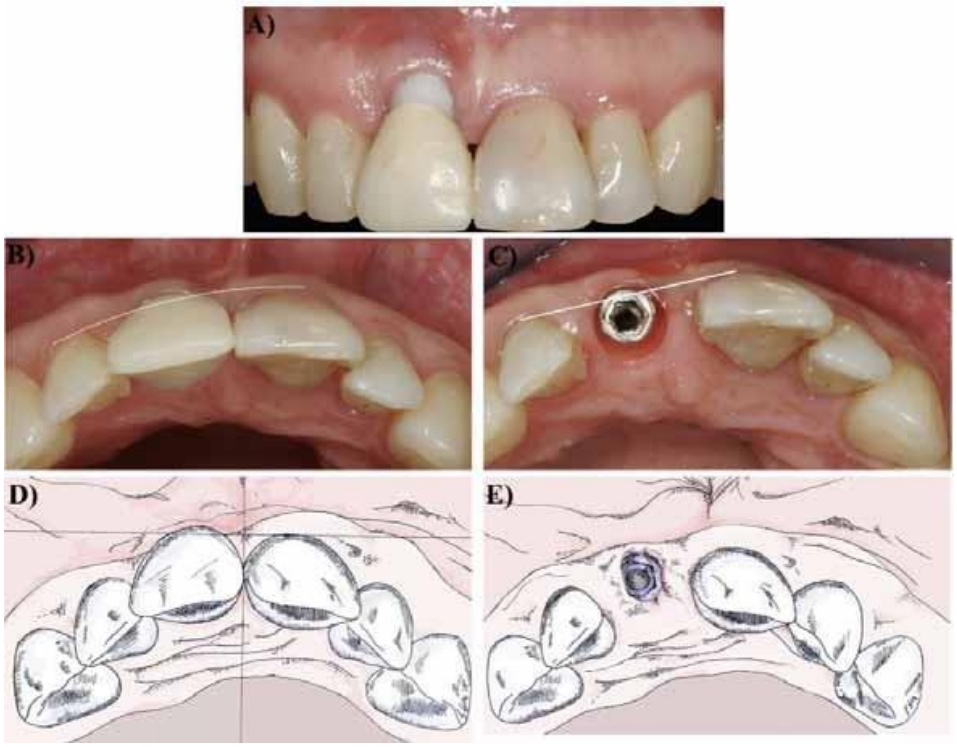

Fig. 4. Frontal (A), occlusal (B and C), and schematic views (D and E, before and after crown removal) of PSTD Class IV. Similar to Class III, the soft tissue margin is positioned toward the apex. After crown removal, however, the implant head lies outside the straight line connecting the profiles of adjacent teeth at the gingival margin level.

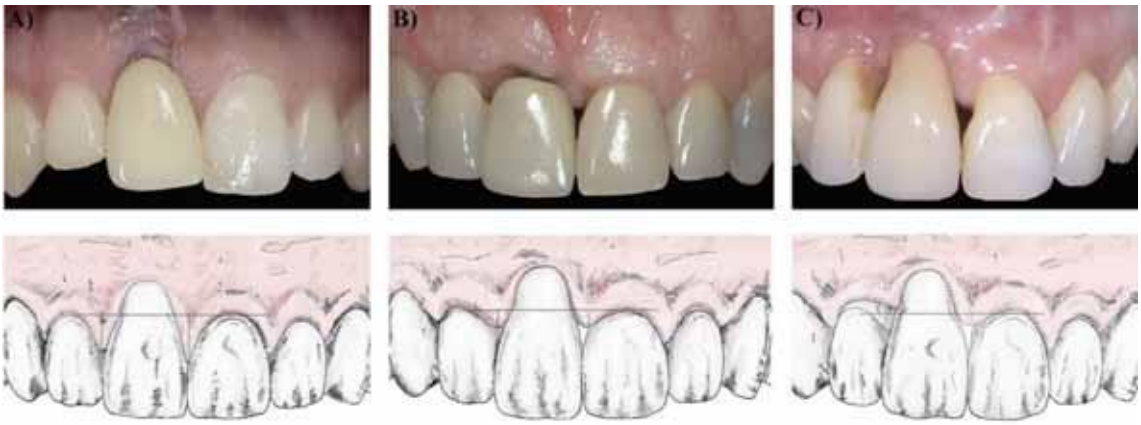

Fig. 5. Subclasses of peri-implant soft tissue dehiscence/deficiency (PSTD) according to papilla level. A) Subclass a: Both papillae tips are ≥3 mm coronally to the optimal margin of soft tissue of the crown. Subclass b: At least one papilla tip is located <3 mm coronally to this margin.

- Risk Factors of Soft Tissue Dehiscence
  The causes of buccal soft-tissue dehiscence in implant restorations remain a subject of debate. However, several risk factors were suggested to be responsible for such undesirable outcome and these include:
- Anatomic Risk Factors

2.3 Thin tissue biotype:

A thin soft-tissue biotype has been recognized as a significant predisposing factor for mucosal recession. In their review, Chen and Buser noted that immediate implant placement in areas characterized by a thin biotype demonstrated a greater incidence of mucosal recession exceeding 1 mm, when compared with sites presenting a thick biotype [7]. Similarly, Nisapakultorn et al. found that soft-tissue biotype was an essential parameter influencing the buccal marginal mucosal level [18]. Their findings indicated that a thin peri-implant biotype substantially elevated the incidence of mucosal recession (odds ratio = 18.8). Furthermore, in this cross-sectional study, sites with a thin biotype demonstrated a significantly greater mean soft-tissue recession compared with thick biotype sites (1.4 mm vs. 0.4 mm). Consequently, sites characterized by a thin tissue biotype should be regarded as being at greater risk of mucosal recession compared with sites with a thick biotype.

- Thick versus thin gingival architecture:
  Treatment planning for dental implant placement should include an evaluation of the periodontal biotype at both the proposed implant site and the adjacent teeth [19]. Siebert and Lindhe later classified gingival biotypes into “thick–flat” and “thin–scalloped” categories. Gingival tissue thicker than 2 mm is considered a thick biotype, while tissue less than 1.5 mm is classified as thin [20] Previous studies indicate that thick periodontal biotypes are more common, representing approximately 85% of the population, whereas thin scalloped biotypes account for around 15% [21]. Thick biotypes are typically correlated with improved periodontal health, as the tissue tends to be denser, fibrotic, and accompanied by a wide area of attached gingiva. Surgically, thick biotypes correspond to a flatter and thicker underlying osseous architecture. Thick gingiva may exhibit low or high scalloping. Patients with thick–flat biotypes typically have short papillae, whereas thick–scalloped biotypes tend to show longer papillae, which may predispose to greater papilla loss [22]. Additionally, thick biotypes are characterized by a flat soft tissue and bone morphology, denser and more fibrotic soft tissue, an ample amount of attached masticatory mucosa, and a greater resistance to acute trauma, and a tendency to develop periodontal pockets and infrabony defects in response to disease. Contact areas of adjacent teeth are larger in both the faciolingual and incisogingival dimensions[23].(As shown in the figure 6)

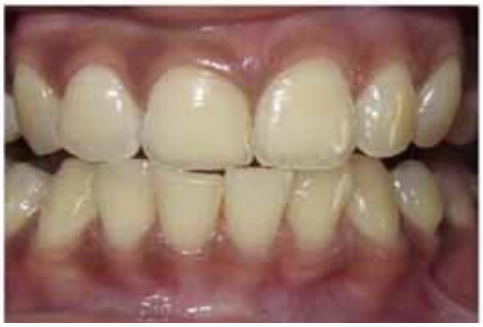

Fig. 6. Thick periodontal biotype.

Thin gingival biotypes are delicate, sharply scalloped, and exhibiting translucency (figure 7). The soft tissue is fragile with a limited zone of attached gingiva, and the supporting bone is exhibits diminished thickness, sometimes with fenestrations or dehiscence covering the labial roots [23]. Patients with thin–scalloped biotypes are considered at higher risk due to their impaired soft tissue reaction after surgical or restorative interventions [21][24]. Gingival thickness can influence treatment outcomes, likely owing to variations in blood supply of underlying bone and susceptibility to resorption. Individuals with thin biotypes are more prone to gingival or periodontal diseases, and post-extraction remodeling often leads to more pronounced alveolar bone resorption, particularly in the apical and lingual regions [25]. Therefore, atraumatic tooth extraction and maintenance of alveolar plates are critical when the area is intended for implant insertion. If impairment of the alveolar structure is anticipated, ridge augmentation protocols should be considered.

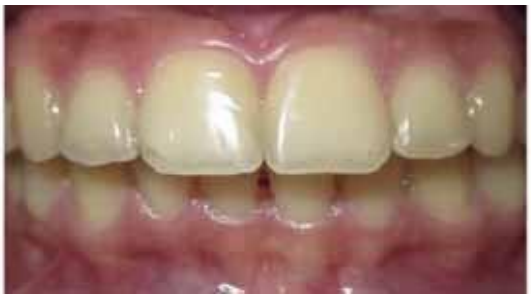

Fig. 7. Thin periodontal biotype

3. KERATINIZATION:

The stability of dental implants may be significantly influenced by the degree of keratinization of the surrounding soft tissues [26]. In a ten-year prospective comparative study, Roccuzzo et al. (2016) reported that implants lacking surrounding keratinized tissue are more susceptible to plaque accumulation and mucosal recession compared with those enveloped by keratinized tissue. The findings of a systematic review and meta-analysis on the role of keratinized mucosa in implant status indicate that insufficient keratinized mucosa surrounding endosseous implants correlates with increased mucosal recession, plaque accumulation, tissue inflammation, and loss of attachment [27]. Conversely, Bengazi et al. assessed tissue margins around implants following the placement of fixed dental prostheses and noted that the absence of keratinized mucosa and increased soft tissue displacement at time of bridge installation were poor predictors of soft-tissue recession over a two-year follow up [6]. Even with certain discrepancies, new evidence supports the existence of buccal keratinized tissue as an important determinant for improving plaque reduction and reducing mucosal recession around implant, although the optimal width of keratinized tissue has yet to be clearly defined. (As shown in the figure 8)

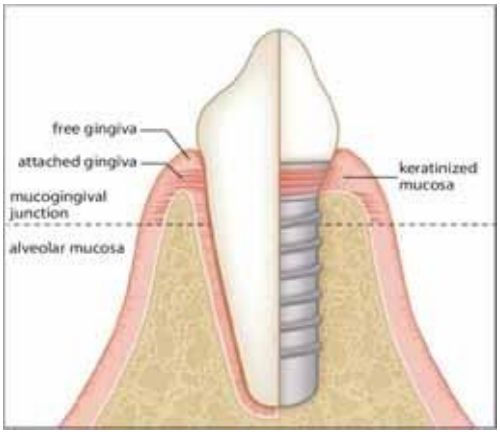

Fig. 8. keratinization of soft tissues

3.1 Socket types:

Several classification systems have been proposed to categorize extraction sockets [28]. Following years of investigation and study, it has been demonstrated evident that while multiple variables are associated with each extraction socket, the critical factor determining socket quality post-extraction is the condition of the buccal hard and soft tissues. Consequently, a straightforward classification system has been intended to facilitate easier

documentation and improve communication among clinicians, researchers, and authors [29].
This classification divides extraction sockets into three types (figure 9):

- Type I Socket: Both the facial soft tissue and buccal bone plate remain intact and at normal levels relative to the cementoenamel junction of the extracted tooth.
- Type II Socket: Facial soft tissue is preserved, but the buccal bone plate is partially lost following tooth extraction.
- Type III Socket: Both the facial soft tissue and the buccal bone plate are significantly reduced after extraction.

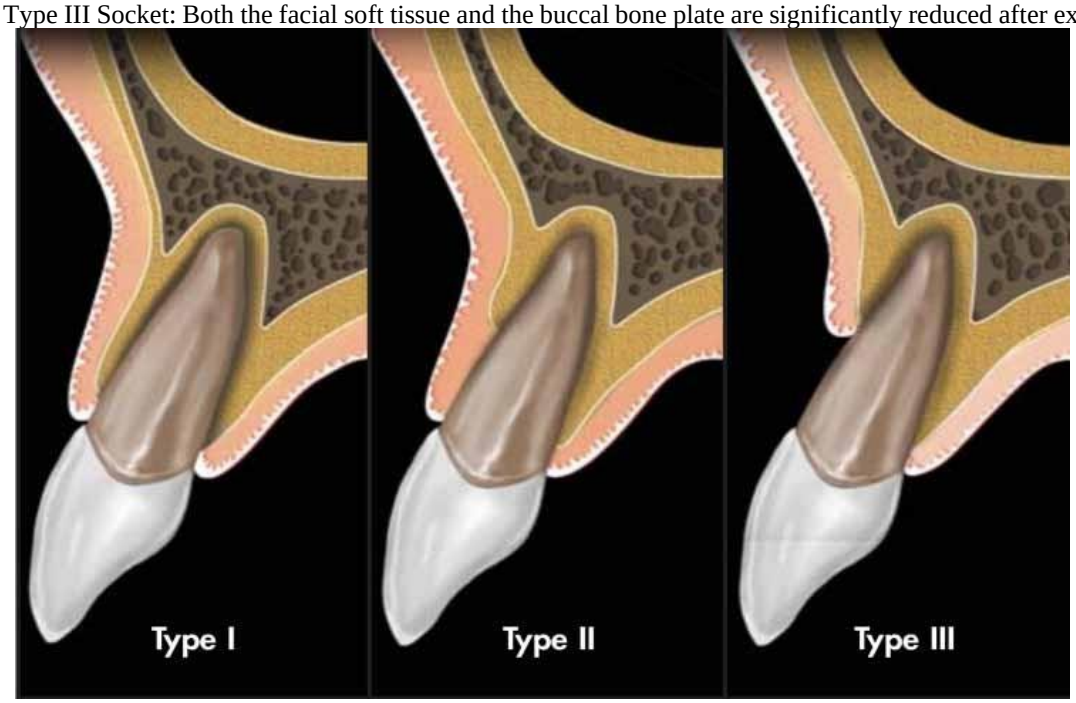

Fig. 9. Extraction sockets can be divided into three kinds according to the status of the facial soft tissue and buccal bone plate.

- Type I sockets are considered the simplest and most reliably manageable. Most Predominantly, cases displayed exceptional esthetic outcomes with dental implants belong to this category, especially when the soft tissue profile is thick and flat rather than thin and highly scalloped [19]
- Type III sockets are the most challenging to treat and typically need soft tissue augmentation, often combined with connective tissue or bone grafts in a staged approach to restore lost tissue. These sockets are usually coupled with prior soft tissue recession and buccal bone loss. Successful management of Type III sockets demands advanced clinical expertise, skill, and significant time investment.
- Type II sockets can be the most challenging to identify accurately. They are sometimes misdiagnosed as Type I sockets by less experienced clinicians, which often results in suboptimal esthetic outcomes. The majority of esthetic complications in implant therapy arise from improper management of Type II sockets, primarily due to post-treatment soft tissue recession. The phenomenon is particularly evident in cases of immediate implant placement. Various techniques have been recommended for the management of such extraction sockets [30]. Recently, treatment approaches have become more reliable with predictive and promising outcomes even with extraction sites with demanding needs for correction. A randomized clinical trial involving thirty extraction sites demonstrated that socket augmentation improved bone quality in both techniques. Additionally, the use of microsurgical instruments and techniques enhanced soft tissue quality, which is essential for successful implant placement and long-term survival [31].

4. IATROGENIC RISK FACTORS

4.1 Implant position:

A critical factor influencing the esthetic outcome of implant restorations is the availability of bone in all three dimensions. Sufficient bone volume is essential to position implant correctly and for maintaining stable soft tissue margins and papillae. Current evidence suggests that maintaining at least 2 mm thickness in buccal bone wall following implant insertion in a healed site is essential to provide Sufficient soft tissue support and to minimize the risk of buccal wall resorption after crown insertion [32][33]. In a study by Spray et al. (2000), the relationship between vertical bone loss and facial bone thickness in two stage implants inserted in healed sites was investigated. The results demonstrated that vestibular bone thinner than 1.4 mm was associated with

significantly greater bone loss, whereas sites with stable facial bone had an average vestibular thickness of 1.8 mm at the time of placement. The authors suggested that when bone thickness reached 1.8–2 mm, the extent of bone loss was markedly reduced, and in certain instances, evidence of bone recovery was also observed. Therefore, preserving a minimum facial bone thickness of 1.8 mm is essential to minimize vestibular bone resorption and the risk of subsequent soft tissue recession.

As well as both optimal positioning of the implant and the diameter of its platform seem to contribute to the magnitude of mucosal recession [34][35]. In a 3- to 5-year prospective study, Small et al. (2001) compared soft-tissue levels around wide- and standard-diameter implants. Their findings indicated that wide-diameter implants revealed a higher average recession and more recession sites at the time of crown placement compared with standard-diameter implants. Furthermore, soft-tissue recession around wide-diameter implants continued to progress at the 5year follow up. Similarly, Ross et al. (2014), in a retrospective analysis of 47 maxillary anterior single implants over a 5-year period, demonstrated a statistically significant correlation between mucosal recession and implant diameter at the lateral incisor site (4.3 mm vs. 3.5 mm). Recession increased proportionally with implant diameter. Moreover, in cases where narrow-diameter implants were utilized for immediate implant insertion and provisionalization, significantly less recession was reported compared with wider implants (0.080 mm vs. 0.812 mm). Collectively, these findings propose that implant diameter is a determinant factor for mucosal recession, with larger diameters posing a higher risk of soft-tissue dehiscence.

Following immediate implant placement, the orofacial position of the implant shoulder demonstrates a strong correlation with mucosal recession [36][37]. Evans & Chen (2008), in a retrospective study on esthetic outcomes, found that Implants placed in a buccal shoulder position showed threefold greater recession than those positioned lingually [38]. Similarly, Cosyn et al. (2012) observed that a buccal shoulder position markedly elevated the risk of mid-buccal recession (odds ratio = 17.2). Evidence consistently indicates that the more buccally the implant is positioned, the greater the apical displacement of the mid-buccal margin [36]. Additionally, Proclined implant axes and deeper implant platform placement considerably elevates the chance of buccal recession defects [18]. (As shown in the figure 10).

TABLE 1 BIOLOGICAL STANDARDS FOR HORIZONTAL PLACEMENT OF IMPLANTS

• A mesiodistal distance of 1.5–2 mm between the implant and adjacent teeth
• A mesiodistal distance of 3–4 mm between implants.
• A buccolingual distance of 2–3 mm from the cervical height of contour
• A coronoapical distance of 2.5–3 mm from the buccogingival margin

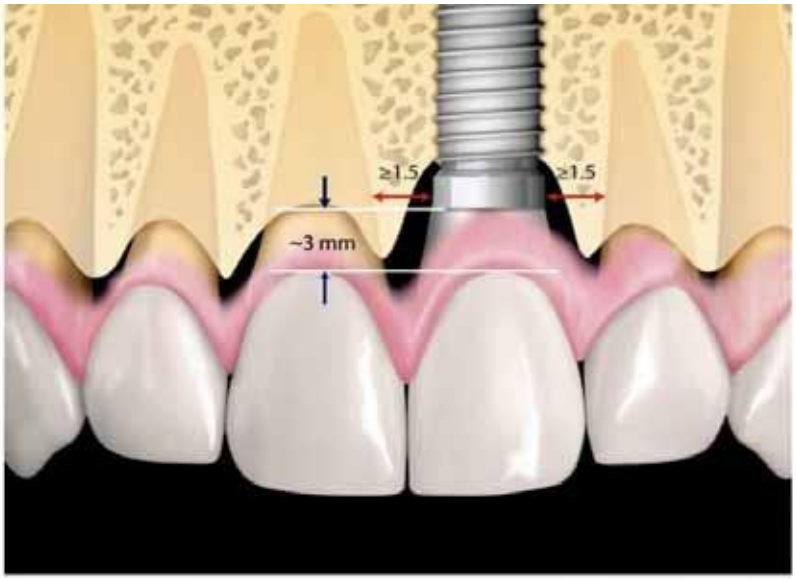

Fig. 10. Correct maxillary anterior implant position, mesiodistally and apicocoronally

4.2 Immediate Versus Delayed Implant:

Immediate implant placement (IIP) has gained popularity among both patients and clinicians because it decreases the number of surgical procedures required and shortens the overall treatment duration. For many years, it was assumed that placing an implant immediately into a fresh extraction socket could prevent the three-dimensional resorption of the alveolar process typically following tooth extraction [39]. However, both preclinical and clinical evidence has failed to confirm this assumption, indicating that IIP may carry esthetic risks [40]. Significant post-extraction bone remodeling, particularly at the buccal aspect, is commonly observed and often leads to a compromised alveolar ridge. Consequently, immediate implant insertion into a fresh socket may result in insufficient buccal convexity and even midfacial recession. Supporting this, a recent systematic review and meta-analysis reported that IIP was associated with a higher risk of early implant failure and midfacial recession compared with delayed implant placement (DIP) [41].

Delayed implant placement (DIP) in healed bone is a straightforward procedure with well-documented long-term clinical and esthetic outcomes [42]. Consequently, DIP is considered the reference treatment modality in scientific research. Two systematic reviews have compared immediate implant placement (IIP) in fresh extraction sockets with DIP in healed sites [43][44] Both reviews, which included studies on single and multiple implants, demonstrated significantly lower survival rates for IIP. Nevertheless, the authors highlighted that these results should be interpreted cautiously because of a high risk of bias related to randomization, allocation, and blinding [44]. Moreover, the evaluated outcomes were limited to implant survival, marginal bone loss, and probing depth changes. Therefore, clinical decision-making should not rely solely on these parameters but should also incorporate surgical feasibility, esthetic considerations, and patient-reported outcomes.

5. DIAGNOSTIC METHODS:

In the direct method, gingival tissue thickness is assessed using a periodontal probe. A thickness greater than 1.5 mm is classified as a thick biotype, whereas values below 1.5 mm are regarded as thin. However, this method has inherent limitations, such as limited probe precision (0.5 mm), variability in angulation during probing, and tissue distortion upon insertion [45]. Using the probe transparency (TRAN) method, the gingival biotype is classified as thin if the probe outline is visible through the sulcus (figures. 11 and 12). This technique has demonstrated high reproducibility, with 85% intra-examiner repeatability (kappa = 0.7) in a clinical trial involving 100 periodontally healthy subjects [19][22]. Another approach involves usage of ultrasonic devices for non-invasive measurement of gingival thickness. Despite its advantages, this method is limited by the challenge of locating the precise measurement site for reproducibility, as well as the high cost and limited availability of the device. Eger's study on cadavers and human subjects found that 95% of repeated measurements deviated by ≤1 mm, with a repeatability coefficient of 1.20 mm [46]. Cone-beam computed tomography (CBCT) enables three-dimensional visualization of the cortical bone surrounding dental implants, providing detailed assessment of both the vestibular and lingual cortical plates. This imaging modality can therefore eliminate the need for re-entry procedures to evaluate peri-implant bone conditions, such as in cases of dehiscence [47]. In addition, CBCT has been applied for the evaluation and measurement of soft-tissue thickness. Fu et al. reported that CBCT provides accurate measurements of both bone and labial soft tissues, concluding that it may represent a more objective method for assessing the thickness of hard and soft tissues compared with conventional direct measurement techniques [48].

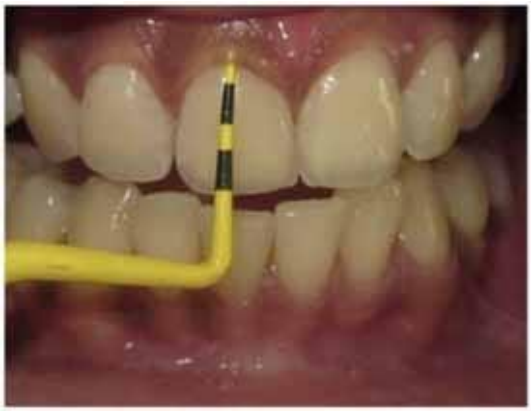

Fig.11. Probe not shown through the sulcus

Fig.12. Probe shown through the sulcus

6. CONCLUSION

When addressing esthetic challenges in dental implant therapy, particularly in the anterior region, multiple factors must be carefully evaluated before treatment. Among the primary concerns for both restorative dentists and surgeons is midfacial recession. Mucosal recession at implant sites is strongly associated with improper implant positioning, thin buccal soft tissue and bone, and reduced buccal bone height.

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Conflicts of Interest:

The authors declare no competing interests.

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