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Clinical management of epidermolysis bullosa: case reports of five siblings

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Abstract

INTRODUCTION. Epidermolysis bullosa (EB) is a rare, inherited disorder characterized by mucocutaneous fragility, leading to blistering and ulceration following minimal trauma. Among its major subtypes, EB simplex (EBS) is the most common, typically presenting with milder clinical manifestations.

AIM. This case series aims to document the clinical and radiographic findings of five siblings diagnosed with EBS, emphasizing the role of dental professionals in diagnosis, management, and improving patient quality of life.

MATERIALS AND METHODS**.** Five siblings with no prior EB diagnosis underwent clinical, radiographic, and genetic assessments. Intraoral and extraoral examinations were conducted to evaluate mucosal involvement, dental anomalies, and associated systemic manifestations. Genetic analysis confirmed EBS Type 2 (KRT5 mutation) in all cases.

RESULTS. None of the cases exhibited significant oral mucosal bullae or erosions. However, hyperkeratotic lesions, nail dystrophy, and perioral bullous formations were observed in all patients. Two siblings presented with bilateral sensorineural hearing loss. Panoramic radiographs revealed missing teeth, likely due to caries-related extractions, highlighting challenges in oral hygiene maintenance. Dental management focused on atraumatic treatment approaches, preventive care, and dietary counseling.

CONCLUSIONS. Dentists play a crucial role in the early detection and multidisciplinary management of EB. Comprehensive oral assessments can aid in subtype identification and guide tailored treatment strategies. Patient education, preventive measures, and regular follow-ups are essential to improving long-term oral health outcomes and overall quality of life for individuals with EB.

Keywords: epidermolysis bullosa, EB simplex, oral health, dental management, case series

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Клиническое ведение пациентов с буллезным эпидермолизом: серия клинических наблюдений у пяти родных братьев и сестер

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Резюме

ВВЕДЕНИЕ. Буллезный эпидермолиз (БЭ) – редкое наследственное заболевание, характеризующееся повышенной хрупкостью кожи и слизистых оболочек, что приводит к образованию пузырей и язв при минимальной травматизации. Среди основных подтипов наиболее распространенным является простой буллезный эпидермолиз (ПБЭ), который, как правило, проявляется в более легкой клинической форме.

ЦЕЛЬ. Настоящая серия клинических наблюдений направлена на описание клинических и рентгенологических особенностей у пяти родных братьев и сестер с диагнозом ПБЭ, с акцентом на роль стоматолога в диагностике, комплексном ведении и улучшении качества жизни таких пациентов.
МАТЕРИАЛЫ И МЕТОДЫ**.** Пятеро родных братьев и сестер, ранее не обследованных на наличие БЭ, прошли клиническое, рентгенологическое и генетическое обследование. Внутриротовой и внелеротовой осмотры проводились с целью оценки поражения слизистой оболочки, наличия зубочелюстных аномалий и системных проявлений заболевания. Генетическое исследование выявило наличие ПБЭ II типа (мутация гена KRT5) у всех пациентов.
РЕЗУЛЬТАТЫ. Ни у одного из обследованных не было выявлено значимых буллезных или эрозивных поражений слизистой оболочки полости рта. Однако у всех пациентов наблюдались гиперкератотические поражения, дистрофия ногтей и буллезные образования в периоральной области. У двух пациентов диагностирована двусторонняя нейросенсорная тугоухость. Панорамные рентгенограммы выявили отсутствие отдельных зубов, предположительно вследствие удаления по причине кариеса, что подчеркивает сложности в поддержании гигиены полости рта. Стоматологическое лечение было направлено на атравматичный подход, профилактические меры и консультирование по питанию.
ВЫВОДЫ. Стоматологи играют ключевую роль в раннем выявлении и междисциплинарном ведении пациентов с буллезным эпидермолизом. Комплексная оценка состояния полости рта способствует уточнению подтипа заболевания и выбору индивидуализированной стратегии лечения. Образование пациентов, профилактические меры и регулярные осмотры необходимы для улучшения долгосрочных стоматологических и общих клинических исходов у пациентов с БЭ.

Ключевые слова: буллезный эпидермолиз, простой буллезный эпидермолиз, здоровье полости рта, стоматологическое ведение, серия клинических наблюдений

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INTRODUCTION

Epidermolysis bullosa (EB) is a rare group of mucocutaneous disorders characterized by localized or generalized blister formation on the skin and mucosa following minor trauma, often leading to scarring [1; 2]. The estimated prevalence of EB ranges from 8 to 19 cases per million individuals [3; 4]. Currently, there is no definitive cure for EB [1]. The condition arises due to mutations in genes encoding proteins in the epidermis or basement membrane, with genetic analysis playing a crucial role in diagnosis [5].

EB is classified into four major types, each with multiple subtypes: epidermolysis bullosa simplex (EBS), junctional epidermolysis bullosa (JEB), dystrophic epidermolysis bullosa (DEB), and Kindler syndrome [6; 7]. More than 30 subtypes have been identified, with epidemiological studies reporting that 92% of cases are EBS, 5% DEB, 1% JEB, and 2% non-classifiable [3].

EBS is predominantly inherited in an autosomal dominant manner, although some subtypes exhibit autosomal recessive inheritance. The most frequently implicated genes include keratin 5 and keratin 14, which encode essential cytoskeletal proteins [1]. EBS is further divided into two major categories–suprabasal and basal–with at least 12 subtypes. The incidence of EBS is estimated to be between 6 and 30 cases per million births [4; 8].

Clinically, EBS lesions typically manifest at birth or during early childhood. These lesions predominantly present as blisters (bullae) and erosions, with milia formation observed in certain subtypes. Unlike other EB types, EBS lesions heal with hyperpigmentation rather than scarring, and nail dystrophy is commonly observed [8]. Histopathologically, intraepithelial separation is a hallmark feature unique to EBS, whereas other EB subtypes exhibit subepithelial separation. No granulation tissue formation is observed in EBS, and the prognosis is generally favorable. While oral mucosal lesions are uncommon in most EBS subtypes, one specific subtype has been associated with a higher prevalence of oral involvement. Additionally, no enamel hypoplasia has been reported, and the dental caries risk remains within the normal range [1].

JEB follows an autosomal recessive inheritance pattern and is characterized by extensive blistering of the skin and mucosa at birth. The condition frequently affects nails (dystrophic or absent) and teeth, with common dental anomalies including anodontia, enamel hypoplasia, neonatal teeth, and increased susceptibility to dental caries. Granulation tissue and oral erosions are often observed, particularly around the mouth, and thimble-like pitting may affect either all or select teeth [9].

DEB may be inherited in either an autosomal dominant or autosomal recessive manner. Across nearly all subtypes, blisters, milia, atrophic scars, and nail dystrophy or loss are characteristic findings. The prognosis varies, with some recessive DEB subtypes associated with severe morbidity and mortality due to squamous cell carcinoma [9]. Oral mucosal lesions and gastrointestinal involvement are frequently observed in DEB, leading to microstomia and ankyloglossia due to fibrosis resulting from repeated trauma [10].

Kindler syndrome follows an autosomal recessive inheritance pattern and presents distinct clinical and histological features that differentiate it from other EB types. These include multilevel skin cleavage, photo-

sensitivity, and progressive poikiloderma [9]. Many EB subtypes exhibit multisystem involvement, contributing to significant morbidity and mortality in some cases. Systemic complications associated with EB include malnutrition, growth retardation, delayed puberty, anemia, infections, osteopenia/osteoporosis, oral mucosal and ocular complications, nail dystrophy, gastrointestinal and genitourinary involvement, upper respiratory complications, musculoskeletal deformities, cardiomyopathy, squamous cell carcinoma, malignant melanoma, temporomandibular joint disorders, airway compromise, and dysphagia [4; 9].

AIM

This case series presents five genetically related siblings diagnosed with epidermolysis bullosa, emphasizing the diagnostic role of dental professionals and the clinical and radiographic findings observed during their assessments. Documenting these cases aims to increase awareness among dental and medical practitioners and contribute to the management strategies of similar cases in future practice.

CASE REPORT

All five patients are siblings and had not received a prior diagnosis of Epidermolysis Bullosa (EB) at any medical institution. Upon referral, genetic analysis was performed at the Dicle University Faculty of Medicine, Department of Genetics, which confirmed a diagnosis of Epidermolysis Bullosa Simplex Type 2 (KRT5 mutation). It was also noted that the parents are third-degree relatives.

Case 1. The first patient is a 1.5-year-old male, the youngest of five siblings. No pathological oral findings were observed upon clinical examination and anamnesis. Extraorally, the patient presented with hyperkeratotic lesions on the elbows, knees, and soles. Additionally, bullous lesions around the lips had developed within the last few months (Fig. 1).

Case 2. The 6-year-old girl, the fourth of the five siblings, presented with cutaneous, oral, and auditory manifestations. Clinical and radiological examinations were performed after applying dermal moisturizers for the lesions in the perioral region. No oral pathological findings were observed during the examination.

The frontal craniofacial examination showed perioral erosions and scarring (Fig. 2, *A*), while a lateral view revealed a cochlear implant, confirming bilateral sensory hearing loss (Fig. 2, *B*). The hand examination displayed scarring, nail dystrophy, and previous blistering sites (Fig. 2, *C*). The foot exhibited hyperkeratotic lesions, consistent with friction-induced blistering (Fig. 2, *D*). Bullous lesions around the lips were evident, correlating with recurrent perioral blistering (Fig. 2, *E*).

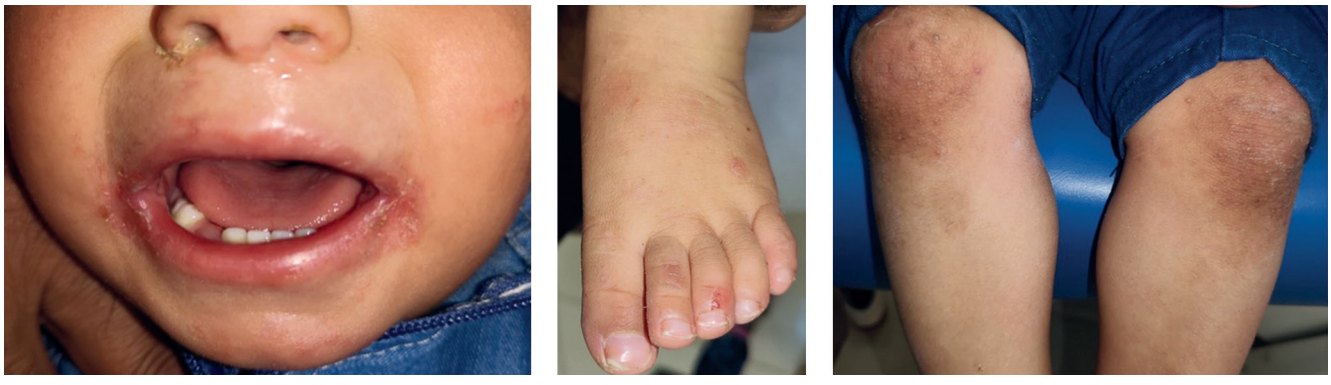
No visible oral mucosal abnormalities were noted, although mild enamel defects were observed (Fig. 2, *F*). Panoramic radiography revealed the congenital absence of tooth numbered 35 and tooth numbered 45 (Fig. 2, *G*).

In accordance with the principles of atraumatic treatment, extraction of tooth numbered 84 was planned, while restorative treatment was provided for tooth numbered 85.

The absence of teeth numbered 35 and 45 in Case 2 and Case 3 is a significant finding. It is likely that these teeth were extracted due to severe dental caries, which may have been exacerbated by a soft food diet and poor oral hygiene.

Case 3. The 10-year-old male patient, the third of five siblings, underwent clinical and radiological examinations following the application of dermal moisturizers for lesions in the perioral region. No oral pathological findings were observed during the examination.

The frontal craniofacial examination revealed perioral erosions and scarring (Fig. 3, *A*), while a lateral view showed a hearing aid, confirming bilateral sensory hearing loss (Fig. 3, *B*). Intraoral examination demonstrated dental plaque accumulation and enamel defects, though no active ulcerations were noted (Fig. 3, *C*). The hand examination revealed scarring and nail dystrophy, indicative of previous blistering episodes (Fig. 3, *D*). The foot exhibited hyperkeratotic lesions, consistent with recurrent friction-induced blistering (Fig. 3, *E*).



A B C

Fig. 1. Case 1: *A* – perioral region of showing bullous lesions; *B* – plantar surface with hyperkeratotic lesions; *C* – knee region with hyperkeratotic and bullous lesions

Рис. 1. Пациент 1: *A* – периоральная область с буллезными поражениями; *B* – подошвенная поверхность с гиперкератотическими поражениями; *C* – область колена с гиперкератотическими и буллезными поражениями

Panoramic radiography revealed the absence of teeth numbered 35 and 45, which is a notable finding (Fig. 3, *F*). The loss of these teeth was likely the result of extensive dental caries, potentially aggravated by a soft food diet and inadequate oral hygiene practices.

In accordance with the principles of atraumatic treatment, restorative treatment was planned for teeth numbered 54, 53, 63, and 64, while preventive fissure sealing and fluoride application were performed for teeth numbered 16, 26, 36, and 46.

Case 4. An 11-year-old male patient, the second of five siblings, was admitted to our clinic due to dental caries. He reported thermal sensitivity in his posterior teeth to hot and cold stimuli, but denied experiencing spontaneous or nocturnal pain.

Frontal craniofacial examination revealed perioral erosions and scarring, characteristic of epidermolysis bullosa-related lesions (Fig. 4, *A*). A lateral view demonstrated additional scarring along the cheek and jawline (Fig. 4, *B*). Examination of the feet showed multiple erosive lesions and hyperkeratotic plaques, consistent with recurrent friction-induced blistering (Fig. 4, *C*). The hand examination revealed scarring and dystrophic changes on the fingers, suggesting prior blistering episodes (Fig. 4, *D*).

Intraoral examination revealed dental plaque accumulation and the presence of Nasmyth's membrane, particularly on the posterior teeth (Fig. 4, *E*). Panoramic radiography, although of suboptimal quality, provided an overview of the patient's dentition and general oral health status (Fig. 4, *F*).

In accordance with atraumatic treatment principles, dermal moisturizers were applied to minimize lesion severity in the perioral region. Restorative treatment was subsequently performed to address the existing dental caries, improving the patient's oral health status.

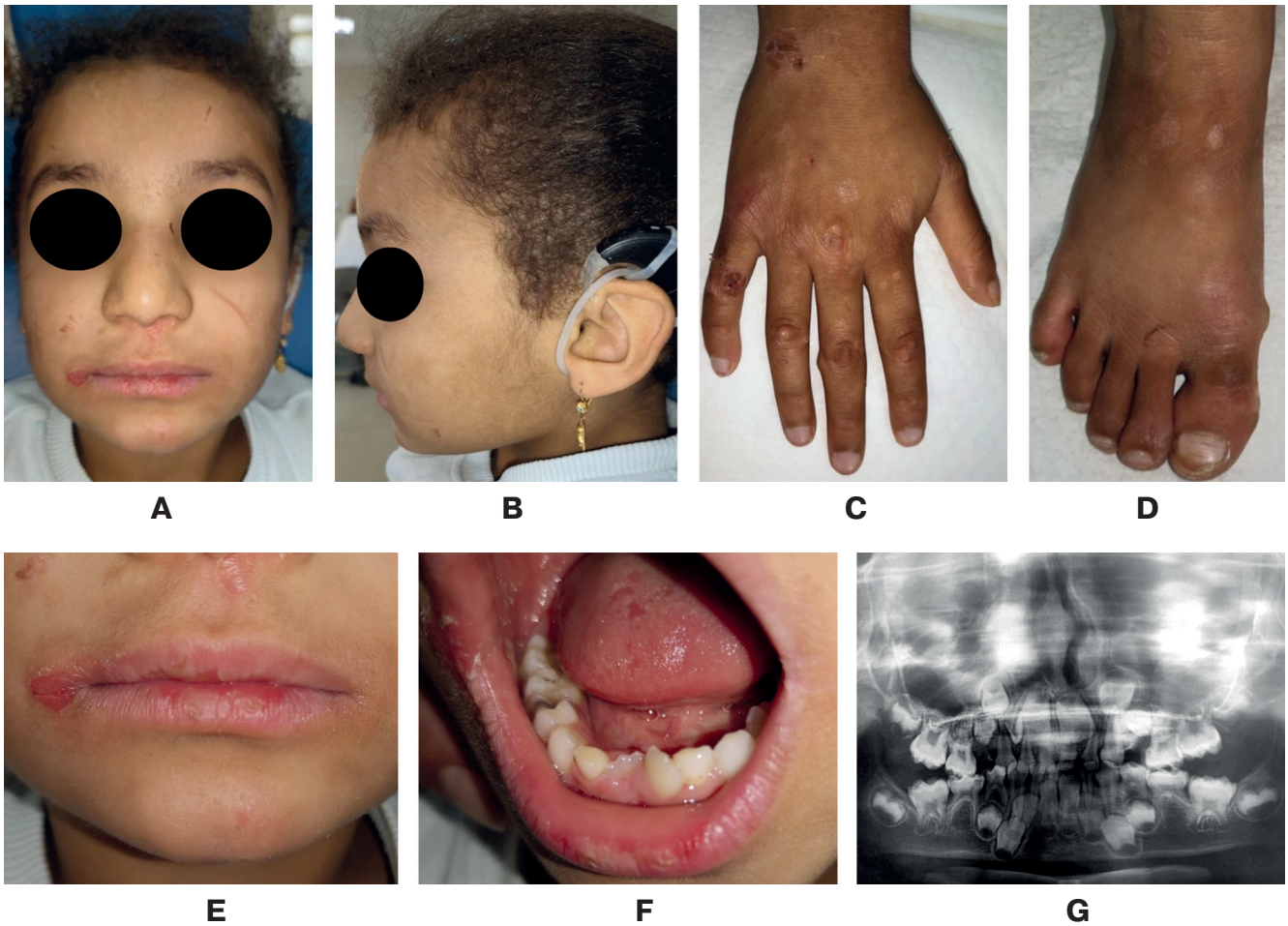


Fig. 2. Case 2: *A* – frontal craniofacial image showing perioral erosions and scarring; *B* – lateral craniofacial image with a cochlear implant; *C* – hand image displaying scarring and nail dystrophy; *D* – foot image showing hyperkeratotic lesions; *E* – perioral image illustrating bullous lesions; *F* – intraoral image showing mild enamel defects; *G* – panoramic radiograph demonstrating the absence of teeth numbered 35 and 45

Рис. 2. Пациент 2: *A* – фронтальное краниофациальное изображение с эрозиями и рубцеванием в периоральной области; *B* – боковое краниофациальное изображение с кохлеарным имплантом; *C* – изображение кисти с рубцовыми изменениями и дистрофией ногтей; *D* – изображение стопы с гиперкератотическими поражениями; *E* – изображение периоральной области с буллезными поражениями; *F* – внутриротовое изображение с незначительными дефектами эмали; *G* – панорамная рентгенограмма, демонстрирующая отсутствие зубов № 35 и № 45

Case 5. The patient is a 13-year-old female, the eldest of five siblings. Clinical and radiological examinations were conducted following the application of dermal moisturizers to the perioral lesions. No pathological findings were detected during the oral examination.

Extraoral examination revealed hyperkeratotic lesions on the elbows, knees, and soles, as well as bullous formations around the lips (Fig. 5). The patient's dental treatment followed the principles of atraumatic care. Additionally, oral hygiene was reinforced through comprehensive oral hygiene education and dietary counseling for caries prevention. The patient has been enrolled in a long-term follow-up program.

DISCUSSION

Epidermolysis bullosa simplex (EBS) has been reported as the most common subtype of EB, with no gender predilection [3]. In contrast, Junctional EB (JEB) and dystrophic EB (DEB) present with oral lesions larger than 1 cm, whereas EBS lesions are typically smaller than 1 cm [11]. Among the EB subtypes, dental anomalies are most frequently associated with JEB, while oral mucosal lesions are more commonly observed in DEB. However, enamel hypoplasia and oral mucosal lesions are rare in EBS [1], and no significant bullae or erosions were noted in the oral mucosa of our cases, which aligns with findings in the literature.

Two of the cases in this study presented bilateral sensorineural hearing loss, which is consistent with previously reported associations between EB and auditory impairment [12; 13]. Additionally, the onset of skin lesions in our cases was reported around 1.5 years of age, which corresponds with the typical clinical course of EBS [8; 11]. Given that consanguineous marriage was reported in the family history, genetic counseling should be strongly considered

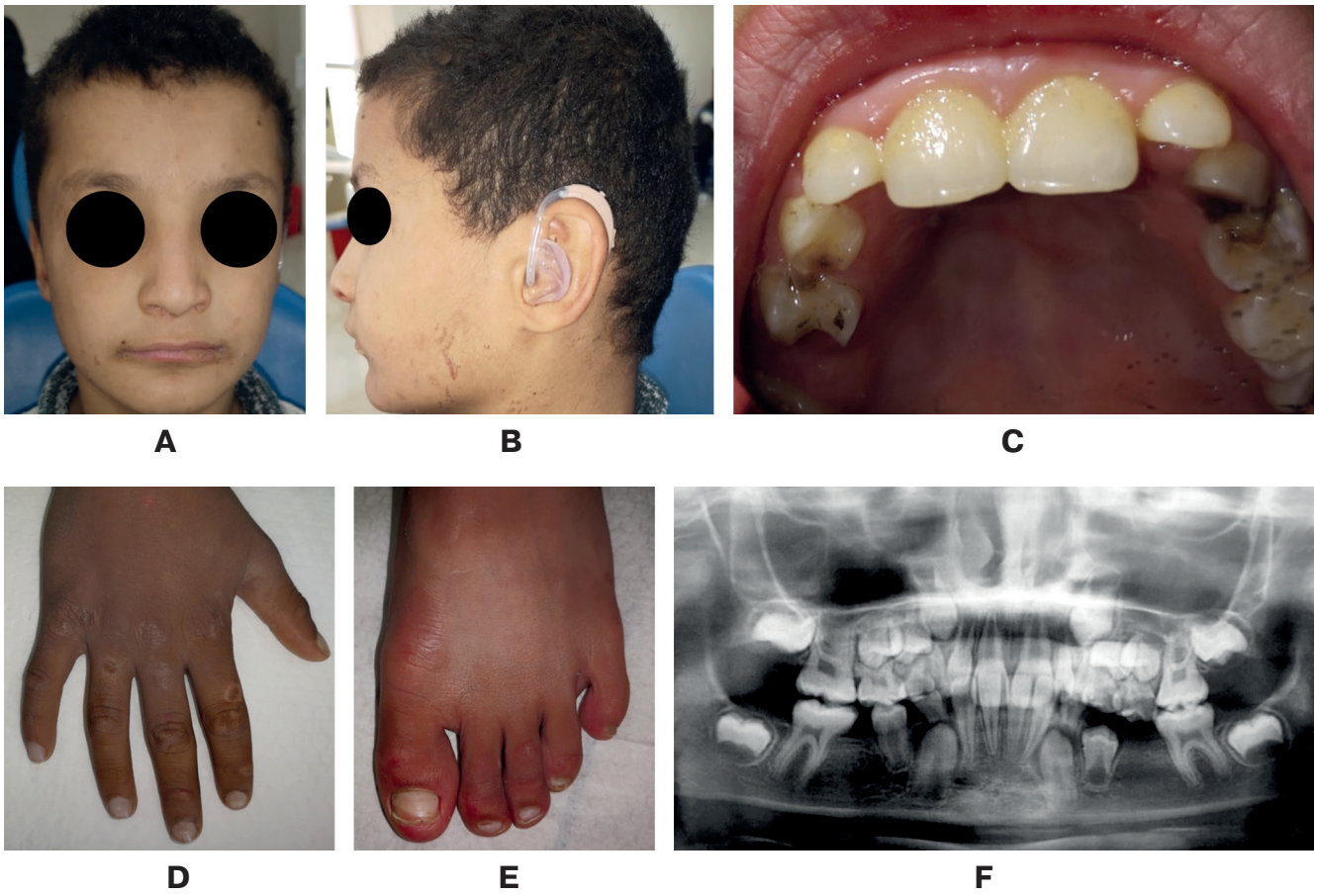


Fig. 3. Case 3: *A* – frontal craniofacial image showing perioral erosions and scarring; *B* – lateral craniofacial image displaying a hearing aid, indicative of bilateral sensory hearing loss; *C* – intraoral image showing enamel defects and dental plaque accumulation; *D* – hand image displaying scarring and nail dystrophy; *E* – foot image showing hyperkeratotic lesions suggestive of friction-induced blistering; *F* – panoramic radiograph demonstrating the absence of teeth numbered 35 and 45, likely due to severe dental caries

Рис. 3. Пациент 3: *A* – фронтальное краниофациальное изображение с эрозиями и рубцеванием в периоральной области; *B* – боковое краниофациальное изображение с слуховым аппаратом, указывающим на двустороннюю сенсорную тугоухость; *C* – внутриротовое изображение с дефектами эмали и накоплением зубного налета; *D* – изображение кисти с рубцовыми изменениями и дистрофией ногтей; *E* – изображение стопы с гиперкератотическими поражениями, указывающими на пузыреобразование вследствие трения; *F* – панорамная рентгенограмма, демонстрирующая отсутствие зубов № 35 и № 45, предположительно вследствие выраженного кариозного поражения

in similar cases to assess hereditary risk and guide family planning.

Accurate subclassification of EB is crucial and is typically determined through family history, clinical findings, and laboratory investigations [1]. The role of the dental professional in EB diagnosis and management is essential, as intraoral examination can provide valuable diagnostic clues. Given the fragility of the oral mucosa in EB, dental treatment protocols should be adapted to minimize trauma and prevent blister formation.

Dental management in EB should prioritize preserving oral health while minimizing mechanical trauma. Preventive strategies should include topical fluoride application, chlorhexidine rinses, and fluoride mouthwashes, particularly in cases where soft tissue sensitivity hinders conventional oral hygiene practices. A soft, sugar-free diet should be encouraged to reduce mechanical irritation and caries risk. When dental treatment is required, lubricating agents such as petroleum-based gels or moisturizing creams should be applied to the lips and oral mucosa to reduce friction and prevent bullae formation [14].

To further mitigate complications, local anesthesia should be administered slowly and deeply to avoid unnecessary tissue trauma. In cases where bullae rupture occurs, oral antiseptics and topical antibiotics should be applied to reduce the risk of secondary infections. Comprehensive dental procedures may necessitate general anesthesia, particularly when extensive restorative or surgical interventions are required [14].

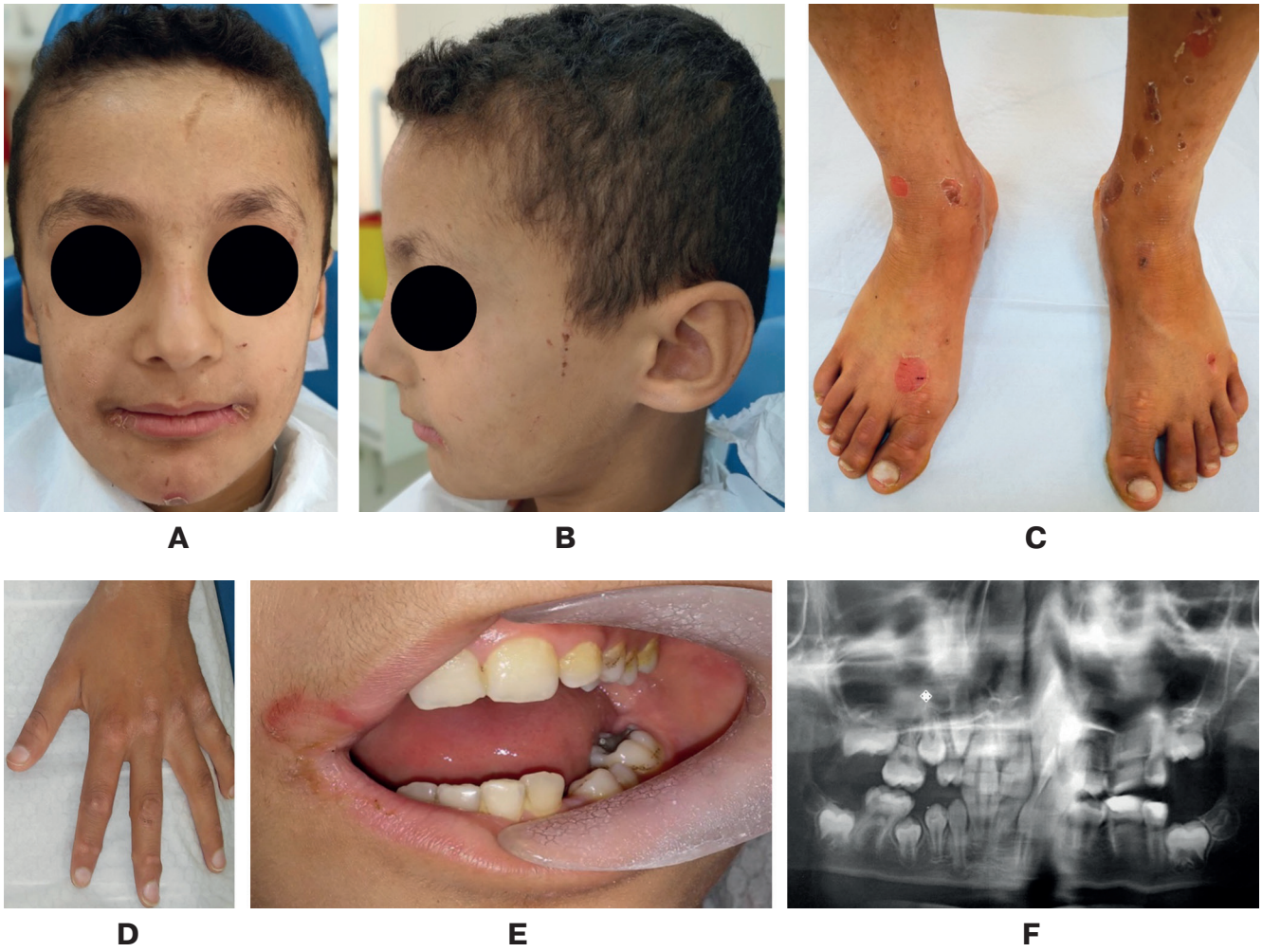
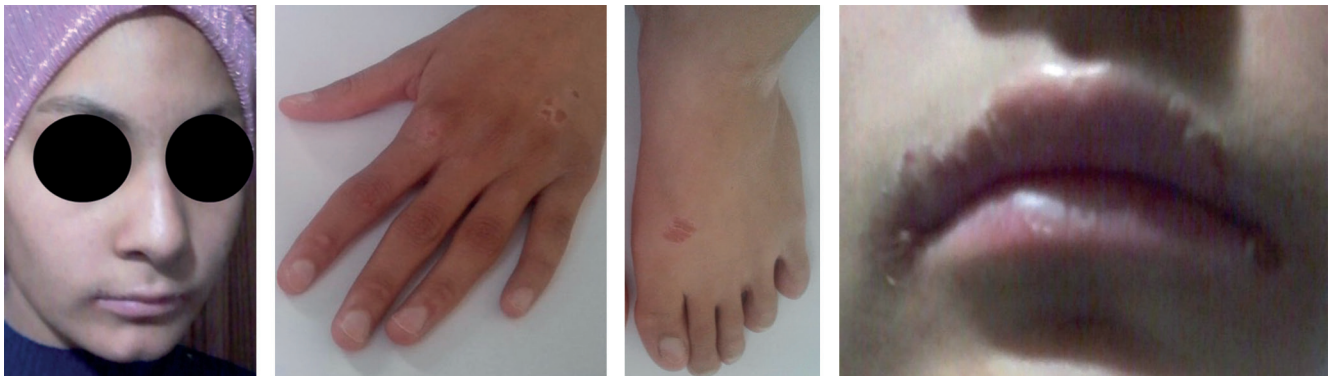


Fig. 4. Case 4: *A* – frontal craniofacial image, showing perioral erosions and scarring; *B* – lateral craniofacial image, displaying additional scarring along the cheek and jawline; *C* – foot image, highlighting multiple erosions and hyperkeratotic lesions; *D* – hand image, demonstrating scarring and dystrophic changes; *E* – intraoral image, showing dental plaque accumulation and Nasmyth's membrane, especially on the posterior teeth; *F* – panoramic radiograph, with suboptimal image quality

Рис. 4. Пациент 4: *A* – фронтальное краниофациальное изображение с эрозиями и рубцеванием в периоральной области; *B* – боковое краниофациальное изображение с дополнительными рубцовыми изменениями в области щеки и нижней челюсти; *C* – изображение стопы с множественными эрозиями и гиперкератотическими поражениями; *D* – изображение кисти с рубцовыми изменениями и дистрофическими нарушениями; *E* – внутриротовое изображение с выраженным накоплением зубного налета и наличием мембраны Нэсмита, особенно на жевательных зубах; *F* – панорамная рентгенограмма с недостаточным качеством изображения



A B C D

Fig. 4. Case 5: *A* – frontal craniofacial image; *B* – hand image, displaying previous blistering and mild nail abnormalities; *C* – foot image, illustrating hyperkeratotic lesions and a healing blister; *D* – perioral image, showing residual scarring and bullous formations

Рис. 4. Пациент 4: *A* – фронтальное краниофациальное изображение; *B* – изображение кисти с признаками ранее перенесенных буллезных поражений и незначительными аномалиями ногтей; *C* – изображение стопы с гиперкератотическими поражениями и заживающим пузырем; *D* – изображение периоральной области с остаточными рубцовыми изменениями и буллезными образованиями

Given the multisystem involvement of EB, a multidisciplinary approach is critical for optimal patient management. Collaboration between dentists (pediatric dentistry, orthodontics, oral surgery), dermatologists, geneticists, and other medical specialists is essential to ensure a comprehensive treatment plan. Additionally, family education and continuous patient monitoring are crucial components in the long-term management of EB.

CONCLUSION

Medical professionals and dentists play a crucial role in enhancing the quality of life for patients with epidermolysis bullosa (EB). Comprehensive intraoral examinations performed by dentists may contribute to identifying EB subtypes, aiding in more accurate diagnosis and management.

Patients and their caregivers should be informed about the functional, phonetic, and aesthetic challenges associated with tooth loss, as well as the importance of preventive strategies to maintain oral health. Additionally, individuals with EB who face difficulties in performing personal oral hygiene should receive adequate support to minimize the risk of dental caries, particularly given the increased susceptibility associated with enamel defects observed in certain EB subtypes.

In conclusion, dentists play a key role in improving the overall well-being of individuals with EB by facilitating early diagnosis, implementing appropriate treatment strategies, ensuring regular oral health maintenance, and promoting interdisciplinary collaboration. Furthermore, their involvement is essential in raising awareness among healthcare professionals to optimize the multidisciplinary management of EB patients.

REFERENCES / СПИСОК ЛИТЕРАТУРЫ

1. Uzun C., Zengin A. Z., Pancar Y**ü**ksel E. Dental ve mukozal oral bulgular ile birlikte epidermolizis b**ü**lloza simpleks: Olgu sunumu. *Ondokuz Mayıs Üniversitesi Diş Hekimliği Fakültesi Dergisi*. 2014;15(1):43–47.
2. Yangın Z., Erkalp K., Delatioğlu H., İlhan A.E., Kökten N., Anesteziyoloji I., Burun I.K. Epidermolizis büllozalı hastada mikrolaringeal cerrahinin anestezi yönetimi: olgu sunumu. *Gülhane Tıp Dergisi*. 2005;47:150–152.
3. Öncel M.Y., Sevim, Ü.N.A.L., Yılmaz A., Yıldız J., & Akbaş A.Y. Epidermolizis Bülloza tanısı alan yenidoğan olgu serisi. *Türkiye Çocuk Hastalıkları Dergisi.* 2011;5(1):5–10.
4. Karaduman A. Kalitsal Büllü Hastaliklar. Inherited Bullous Diseases. *Turkderm*. 2011;45(2):81–86.
5. Intong L.R., Murrell D.F. Inherited epidermolysis bullosa: new diagnostic criteria and classification. *Clin Dermatol*. 2012;30(1):70–77. https://doi.org/10.1016/j.clindermatol.2011.03.012
6. Fine J.D., Bruckner-Tuderman L., Eady R.A., Bauer E.A., Bauer J.W., Has C. et al. Inherited epidermolysis bullosa: updated recommendations on diagnosis and classification. *J Am Acad Dermatol*. 2014;70(6):1103–1126. https://doi.org/10.1016/j.jaad.2014.01.903
7. Wright J.T. Oral manifestations in the epidermolysis bullosa spectrum. *Dermatol Clin.* 2010;28(1):159–164. https://doi.org/10.1016/j.det.2009.10.022
8. Sprecher E. Epidermolysis bullosa simplex. *Dermatol Clin*. 2010;28(1):23–32. https://doi.org/10.1016/j.det.2009.10.003
9. Fine J.D. Inherited epidermolysis bullosa. *Orphanet J Rare Dis*. 2010;5:12. https://doi.org/10.1186/1750-1172-5-12
10. Serrano-Martínez M.C., Bagán J.V., Silvestre F.J., Viguer M.T. Oral lesions in recessive dystrophic epidermolysis bullosa. *Oral Dis*. 2003;9(5):264–268. https://doi.org/10.1034/j.1601-0825.2003.03971.x

11. Wright J.T., Fine J.D., Johnson L. Hereditary epidermolysis bullosa: oral manifestations and dental management. *Pediatr Dent*. 1993;15(4):242–248.
12. Hore I., Bajaj Y., Denyer J., Martinez A.E., Mellerio J.E., Bibas T., Albert D. The management of general and disease specific ENT problems in children with Epidermolysis Bullosa – a retrospective case note review. *Int J Pediatr Otorhinolaryngol*. 2007;71(3):385–391. https://doi.org/10.1016/j.ijporl.2006.11.005
13. Fine J.D., Johnson L.B., Weiner M., Suchindran C. Tracheolaryngeal complications of inherited epidermolysis bullosa: cumulative experience of the national epidermolysis bullosa registry. *Laryngoscope*. 2007;117(9):1652–1660. https://doi.org/10.1097/MLG.0b013e318093ed8e
14. Wright J.T. Comprehensive dental care and general anesthetic management of hereditary epidermolysis bullosa. A review of fourteen cases. *Oral Surg Oral Med Oral Pathol*. 1990;70(5):573–578. https://doi.org/10.1016/0030-4220(90)90401-d

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