Discoveries Journals
publishing the future

DISCOVERIES REPORTS 2021, 4: e23
DOI: 10.15190/drep.2021.8

REVIEW Article

Integrated approach for management of oro-dental manifestations in survivors of Stevens-Johnson syndrome and toxic epidermal necrolysis

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Submitted: May 19, 2021; *Revised*: June 21, 2021; *Accepted:* June 30, 2021; *Published:* June 30, 2021;
Citation: Miglani S, Ansari I, Boreak N, Vadav V. Integrated approach for management of oro-dental manifestations in survivors of Stevens-Johnson syndrome and toxic epidermal necrolysis. *Discoveries Reports*, 2021; 4: e23. DOI: 10.15190/drep.2021.8

ABSTRACT

Stevens-Johnson syndrome and toxic epidermal necrolysis are severe muco-cutaneous reactions seen in clinical practice, usually due to adverse reactions to certain drugs, such as sulfonamides, anticonvulsants, and non-steroidal anti-inflammatory drugs. They are characterized by the eruption of blisters and epithelial sloughing, which can turn into a life-threatening situation. Acute and chronic complications of Stevens-Johnson syndrome and toxic epidermal necrolysis are well documented in the literature, and it is recognized that survivors may develop oro-dental abnormalities as a long-term complication of Stevens-Johnson syndrome. To date and to our knowledge, there is no publication available emphasizing the importance of an integrated approach amongst dental specialties for the management of oro-dental complications of Stevens-Johnson syndrome and toxic epidermal necrolysis. This review aims to provide an update on a wide range of oro-dental sequelae as a long-term complication following an acute episode of Stevens-Johnson syndrome and toxic epidermal necrolysis, along with an emphasis on the need of an interdisciplinary collaboration of dental specialties for early diagnosis and management of such cases.

SUMMARY

Keywords
SJS, TEN, rhizomicry, short root anomaly, dental aberrations, Steven Johnson syndrome.

Abbreviations
Stevens-Johnson syndrome (SJS); toxic epidermal necrolysis (TEN); non-steroidal anti-inflammatory drugs (NSAID's); orthopantomogram (OPG).

Table 1. Long term complication of Steven Johnson Syndrome/Toxic Epidermal Necrolysis

Structure involved	Clinical presentation of lesions
Skin	Postinflammatory hyper- hypopigmentation
	Photosensitivity
	Chronic pruritus
	Eruptive naevi
	Hypertrophic scarring
	Anetoderma
	Verrucous hyperplasia
	Heterotopic calcification
	Ectopic sebaceous gland
Nails	Onychomadesis
	Cicatricial anonychia
	Dystrophy
	Pterygium formation
	Ridging
	Streaky pigmentation
	Longitudinal nail ridges
Hairs	Telogen effluvium
	Alopecia areata
Adnexals	Hyperhidrosis
Eyes	Lids - ectropion, entropion, trichiasis, distichiasis, lagophthalmos
	Conjunctiva - persistent hyperaemia, symblepharon, ankyloblepharon, forniceal shortening
	Cornea - superficial punctate keratopathy, loss of palisades of Vogt, epithelial defects, corneal scarring, neovascularization, keratinization, infectious keratitis, corneal thinning
	Others - Chronic photosensitivity, decreased visual acuity, lacrimal duct obstruction, dry eyes
Other (less common)	Pulmonary, renal/urogenital, hepatic/gastrointestinal, psychological sequelae

1. Introduction

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare, but life-threatening conditions characterized by the eruption of mucocutaneous blistering and epithelial sloughing[1]. The etiology for SJS/TEN can be multifactorial. However, severe adverse reactions to certain drugs is the most common cause. Some of the drugs responsible for these adverse reactions are allopurinol, antibacterial sulfonamides, lamotrigine, carbamazepine, nevirapine and piroxicam[2]. SJS and TEN are considered to be the two ends of a disease spectrum based on the amount of body surface area detachment, where SJS is the less (<10%) and TEN is the more extensive form (> 30%). Cases that fall within 10–30% epidermal detachment are referred to as SJS-TEN overlap[3]. The overall incidence of SJS/TEN is about 1–2 cases per million[4]. While the acute and chronic systemic complications of SJS/TEN are well described, it is increasingly recognized that survivors may also develop delayed oro-dental sequelae, which can be associated with significant morbidity[5].

Long-term systemic complications of SJS/TEN include cutaneous, ocular, pulmonary, renal, gastrointestinal, hepatic, psychiatric, and psychosocial sequelae. Cutaneous pigmentation and

ocular changes are the most commonly evident in such patients. Cutaneous changes include scarring, dyspigmentation, along with malformed nails (pterygium formation and longitudinal ridging). Ocular changes, such as reduced tear production, partial loss of vision, can lead to corneal transplant surgery, bilateral keratitis of eyelids with symblepharon[5] (Table 1).

This review aims to provide an update on a wide range of oro-dental sequelae as long term complication following an acute episode of SJS/TEN, along with an emphasis on the need for interdisciplinary collaboration of dental departments for early diagnosis and management of such cases.

2. Acute and chronic oro-dental conditions

2.1 Acute oral lesions

In a study on 49 patients who underwent oto-laryngeal workup, it was observed that 48 patients (98%) had ear-nose-throat symptoms (i.e, odynophagia, dysphagia, breathing difficulties, dysphonia, nasal obstruction, earache) with odynophagia being the most common one[6]. Most of the lesions were seen in the oral cavity (94% of cases), involving mainly the lips, the buccal mucosa, and the gum. Lesions included erythematous maculae (n=45[92%]), superficial erosions (n=39 [80%]), oral ulcerations (n=7) [14%]), and bullous (n=5 [10%]). Most of the severe lesions were hemorrhagic bullous (n =5[10%]) on the palate as described by it was observed that complete healing took place in 75% of patients at 2 months, and > 95% of patients at 1 year[6]. However, an older series reported higher chronic oro-nasopharyngeal mucosal lesions (10–20%) in SJS/TEN survivors[7].

2.2. Chronic oral lesions

In a study on 45 patients with TEN, it was reported that 73% of the patients who had mucosal involvement in the acute phase of TEN developed long-term oral complications. The most common site was the oral mucosa (45%) and the esophageal mucosa (45%), followed by lung mucosa (18%), and genital mucosa (9%)[8]. The severity of acute oral mucosal involvement can be a risk factor for the development and continuation of chronic lesions.

2.2.1 Adhesions of oral mucosa, tongue and lip

Long-term oral complications present a wide spectrum of lesions, which include the formation of frenum like fibrotic bands connecting movable oral mucosa to attached oral mucosa (mainly labial mucosa), between the tongue and the floor of the mouth, and between gingival surfaces and adjacent structures. Such changes can affect mouth mobility sometimes[9]. It was also reported that 38% of survivors (9/16) had gingival adhesions[10].

2.2.2 Chronic or recurrent ulcers

Other oral manifestations include mouth discomfort due to the presence of chronic or recurrent ulcers in the oral cavity. In a follow-up study of 16 survivors with SJS/TEN, it was observed that all patient complained of mouth discomfort, 9 had altered tongue mucosa, 13 had gingival inflammation, and 14 had gingival recession. This can lead to difficulty in chewing and reduced taste sensation[11,12].

2.2.3 Sjogren sicca like syndrome

Long-term oral complications of SJS/TEN also include symptoms of dry eyes, dry mouth, and reduced salivary flow. A study reported that Sjogren-like sicca syndrome can be present in 55% of survivors of SJS/TEN (5/9), which is not associated with antinuclear antibody or Ro/La antibodies, since none of the survivors reported positive (0/9). Dry mouth was observed 4 of the 9 cases (44%), whereas 6 of the 9 cases had reduced salivary flow (66%)[12].

Another study reported Sicca syndrome in all the patients who had a history of SJS/TEN. It was found that all of them have lower salivary volume and lower pH with lower buffering capacity. A few of them had abnormal viscosity of saliva. This can lead to gingival inflammation and periodontitis, by encouraging the growth of uncommon bacteria species, such as the Tannerella forsythia and Porphyromonas gingivalis[11].

In 33% of patients with SJS/TEN, raised serum amylase (hyperamylasemia) was observed, which could be due to salivary gland involvement. This increase could be predictive of post-SJS/TEN Sjogren-like sicca syndrome, since it was found to correlate with ocular sequelae[13].

2.3 Dental alterations

Dental alterations such as caries, missing tooth, severe dental growth abnormalities (agenesis, root dysmorphia, rhizomicry, and short root anomaly) were frequent among such patients[5].

2.3.1 Caries

Quantitative and qualitative changes to saliva occur due to Sicca-like syndrome in SJS/TEN survivors[11]. These changes are believed to promote the occurrence of caries. Depending on the progression of caries into dentin, pulpal or periradicular inflammation can take place, resulting in reversible or irreversible pulpitis, pulpal necrosis, symptomatic or asymptomatic apical periodontitis, and abscesses. If gross decay of tooth occurs it may lead to extraction of the involved tooth. De Man et al. reported a case where they have found mutilated dentition with multiple caries. Periapical radiolucent areas with multiple teeth were observed on intra oral periapical radiograph, which could be ascribed to necrotic pulps[14]. Brook et al. also reported gross caries of permanent dentition (incisors and molars) at the age of 14, who had past history of SJS at the age of 9 years[15,16]. In a retrospective dental checkup of patients with a history of SJS/TEN, it was found that 11/16 patients had a carious tooth, 8/16 at least had one missing tooth, and 3/16 had severe periodontal disease[11]. Bajaj et al. also obtained similar results, where multiple mandibular molars were carious and extraction of lower 3rd molars of both sides was performed due to gross caries[17]. Similar findings were reported by Sangwan et al.[18] and Song et al[19]. Additionally, hypomineralisation of teeth was reported by Song et al[19].

2.3,2 Dental growth abnormalities

Severe dental growth abnormalities, such as dental agenesis, root dysmorphia, short root, microdontia, and incomplete root apex closure can be seen in patients who have contracted SJS/TEN during early childhood[5.] Similar findings were reported in a few case reports. The summary of all the dental alterations is given in Table 2[11,14-19]. De Man et al. observed that all teeth except permanent first molars had short roots, where premolars had extremely short roots and they were loose in the socket[14]. Similar findings were reported by Ranalli et al.[15], Brook et al.[16], Bajaj et al.[17], and Sangwan et al[18]. One common element in all these findings was the age of occurrence of the SJS, within the range of 7-8 years. Gautlier et al. also reported similar findings, where root-building abortion with incomplete apex closure and root dysmorphia was observed in many cases[11]. Song et al. reported short roots in permanent first molars and incisors only[19]. The plausible explanation was attributed to the age of SJS attack around the development of roots of permanent dentition[14]. The disordered root development may cause limitation in food mastication and abnormal tooth mobility and predisposing to dental caries.

Research studies suggested that the abnormal root development after SJS/TEN can be attributed to secondary damage (apoptosis) to the keratinocytes of Hertwig's epithelial root sheath of the developing tooth[14]. This can also result in inhibition of differentiation of dental papilla cells into odontoblasts, with a limited number of odontoblasts, which can induce the malformation of the root dentin during root development[19].

The differential diagnosis of such short roots includes various conditions, such as external inflammatory root resorption, short root anomaly, and cessation of root development due to other systemic diseases (Table 3).

3. Treatment strategies for management of oro-dental sequelae in survivors of SJS/TEN

There is no specific protocol described in the literature for the management of severe dental aberrations in survivors of SJS/TEN. Thus, we propose an integrated approach of all dental specialties for more efficient diagnosis and treatment planning. This approach would involve a team consisting of oral medicine and radiologist, pedodontist, endodontist, orthodontist, oral surgeon, periodontist, and prosthodontist.

3.1 Role of the oral medicine and radiologist

They are the first specialists from the dental fraternity to examine a patient. Extensive knowledge of clinical presentation and dental developmental challenges are of great importance in managing patients' condition. Most of the patients with a history of SJS/TEN report complaints of difficulty in mastication. Thus, it is their role to note the extraoral features such as ocular damage, nail changes, and skin dyspigmentation, and correlate them with the past medical history. On further examination, they should look for chronic scars, ulcers, and oro-mucosal adhesions during the intraoral examination. A salivary flow test should be done to confirm reduced salivation. Palliative treatments should be provided in such cases, including supragingival cleaning and debridement. Use of palliative oral rinses and gels (magic mouthwash, chlorhexidine 0.12%, and lidocaine 2% gel) should be encouraged

Table 2. List of published cases of dental developmental abnormalities due to Stevens Johnson Syndrome

Title of publication	Age of SJS attack (years)	Findings	Radiographic evaluation	Drug intake
Severe and Unrecognized Dental Abnormalities After Drug-Induced Epidermal Necrolysis[11]	2.5-11	Carious tooth. Missing teeth. Severe periodontitis (growth of uncommon bacterial species (ie, *T forsythia* and *P gingivalis*)). Abnormal root development. Short root.	OPG	
Abnormal root development probably due to erythema multiforme (Stevens Johnson Syndrome)[14]	7 ½	a) Mutlilated dentition with much caries. b) Extraction of 22 and 43. c) Slight gingivitis in right and left lower tooth. d) Except permanent molars all the tooth present had short roots. e) Abnormally short premolars. .	IOPA	Aspirin Phenobarbital Codein phosahte
Stevens-Johnson syndrome: report of case with abnormal root development[15]	< 4	At age of 4 years – open apices of 2nd molar. At age of 8 years- abnormal root development.	OPG	
Stevens-Johnson syndrome and abnormal root development: a case report[16]	9	Gross caries of incisors and molars of permanent dentition. Social isolation and school phobia. Short rooted premolars.	OPG	Theophylline
Cessation in root development: ramification of Stevens- Johnson syndrome[17]	8	Teeth number 36, 37,46, 47 were carious and restorable. Teeth 38 and 48 were grossly decayed and indicated for extraction. Abnormal short root of premolars and 2nd molars. Except permanent 1st molars.	OPG	Acyclovir. Nemusulide, roxikid
Stunted root development: A rare dental complication of Stevens-Johnson syndrome[18]	7	Five of his teeth viz. 15, 25, 37, 47 and 45 were carious. Caries in 37 and 47 were deep, Generalised short roots of permanent teeth except 1st molar and incisors.	OPG	Amoxicylline
Stevens-Johnson Syndrome: A Case Report[19]	5	Caries on occlusal surface of 1st molar of the maxilla and mandible and the mesial proximal surface of 1st primary molar of the right axilla and central incisor of the right mandible hypomineralisation of few teeth. Abnormal root development of permanent first molars and mandibular incisors.	IOPA and OPG	Local medication for common cold

Intra oral periapical radiograph (IOPA); orthopantomogram (OPG).

along with chewing gums to increase the salivation[20]. If required, intra oral periapical radiograph and orthopantomogram (OPG) X-rays should be advised. It is necessary to reach to correct diagnosis for the cause of current dental disease[15-18].

Table 3. Differential diagnosis of short roots (rhizomicry) due to SJS/TEN

Condition	Findings	Etiology
Short root anomaly	Short, plump root in incisors and premolars	Genetic
	Dentin dysplasia and Dentinogenesis imperfecta	Hereditary autosomal dominant Dental developmental disturbance
	Hypoparathyroidism	Calcium Metabolic disorder (systemic disturbance)
	Thalassemia	Haematological disorder
	Long term phenytoin therapy in epilepsy	Calcium metabolic disorder
	Short roots due to radiotherapy and chemotherapy before age of 12	Environmental insult due to childhood malignancy
	Short root associated with short stature and syndrome	Down syndrome, Aarskog syndrome, Seckel (bird headed dwarfism), Rothmund – Thomson syndrome, skeletal dysplasia, "familial otodentodysplasia
	Short root associated with other syndromes.	Stevens– Johnson syndrome Scleroderma Laurence–Moon–Bardet–Biedl syndrome
Immature apex	Localized short root.	Trauma
External root resorption	Generalized/localized short root.	Orthodontic forces/ trauma, periapical lesion, cyst
Root hypoplasia in predecessor tooth	Short root of deciduous predecessor tooth.	Alveolodental trauma

Steven Johnson Syndrome (SJS); Toxic Epidermal Necrolysis (TEN).

An allergy screening test should be carried out to identify the causative drug.

3.2 Role of pedodontist

If the age of the patient is less than 14 years, a pedodontist will be the first dental specialist to perform any intervention. Their first role will be to provide psychological support to the patient, as such patients may have school phobias, social isolation and psychological fears. The next focus should be on the prevention of caries and space maintenance as it will make the job of other dental specialties easier as well. Various procedures, such as restoration of carious teeth, and vital pulp therapy procedures (pulp capping and pulpotomy) can be performed to treat the deeply carious teeth with short roots. Extraction of the grossly carious teeth should be performed. Also, parents can be asked for genetic consultation before they plan to have another child[15-19].

3.3 Role of endodontist

If the age of the patient is greater than 14 years, the role of the endodontist and conservative dentist comes into play. They should make sure to correctly diagnose and differentiate the condition of external inflammatory root resorption or immature apex due to trauma from developmental aberrations of short roots with immature apex due to SJS. Caries should be treated at the earliest and conservatively, since teeth in this syndrome might have dentinal dysplasia and are already compromised in morphology. If deep dentinal caries have resulted into pulpal inflammation, vital pulp therapy procedures should be performed with priority.[17-19].

3.4 Role of oral surgery

The role of an oral surgeon in such cases is very challenging, since they have to collaborate with the orthodontic department to decide whether to save or extract the impacted tooth with short roots as they

could have lost the eruption potential. Other than that, grossly decayed tooth or excessive mobile tooth should be extracted[19]. The extracted tooth with a short root or incomplete formed apex should be sent for histopathological examination. Oro-mucosal adhesions, such as lip adhesions should be treated by use of carbon dioxide lasers, under local anesthesia[10].

3.5 Role of oral pathology

Histopathological findings suggesting plausible cause of malformed tooth and short roots secondary to SJS/TEN has not been reported. Thus, it will be very useful to examine extracted teeth with short/malformed roots for histopathological, molecular and genetic findings. This will clear the confusion regarding the causation of a wide variety of dental aberration post SJS/TEN[15-19].

3.6 Role of orthodontist

These patients are usually referred to an orthodontist for impacted teeth and spacing in the dentition. The orthodontic force in teeth with short roots, predisposes the teeth to a higher risk of root resorption. At times, the orthodontic movement is contraindicated for teeth with severe forms of short root anomaly. In most cases, clinical and radiographic monitoring can be used to control root resorption during orthodontic tooth movement[21]. An accurate diagnosis is important to plan the treatment and biomechanics. The risk and benefits should be evaluated before treating a complex case, such as canine impaction[22]. Only light forces should be used with prolonged time in-between activation of appliances, along with sequential monitoring of root resorption clinically and radiographically. The tooth movements requiring heavy forces, like bodily retraction, intrusion, and torqueing of incisors, should be avoided. It is also recommended to bond only a few teeth and evaluate the progression of root resorption followed by complete arch bonding if there is no adverse effect. Extrusive mechanics to the teeth affected by short root anomaly is to be very carefully avoided. Anchorage planning is critical, as first molars show compromised root length and have poor anchorage value, thereby skeletal anchorage from tuned aperture device can be considered. Segmental mechanics with mini screw supported cantilever spring for canine eruption can help avoid forces on the already compromised dentition.

3.7 Role of periodontist

Their role is to reinforce good oral hygiene measures in such individuals. Regular follow-up is required to assess the gingival and periodontal status[11].

3.8 Role of prosthodontics

Finally, the prosthetic rehabilitation of missing teeth can be planned along with improvement of reduced vertical dimension.

4. Conclusion

In this review, we have presented the spectrum of delayed and chronic oro-dental sequelae that can occur in survivors of SJS/TEN, highlighting the importance of an integrated approach between different dental specialties for early diagnosis and treatment planning of such cases. Due to the small numbers of patients with SJS/TEN treated in any particular dental center, an understanding of long-term oro-dental abnormalities is based on small retrospective case series and individual case reports. Nonetheless, it is clear that whenever dental abnormalities are observed in permanent dentition, it necessitates careful scrutiny of the patient's past medical history. A correct diagnosis should be established by correlating the clinical (physical/dental) findings with past medical history. Referral to respective dental specialties should be guided by a coordinating dentist, usually an oral medicine and radiologist. The demand for clinical input from various specialists gives support to the development of dental centers which can draw upon all the expertise needed to manage the chronic, as well as the acute oro-dental sequelae of SJS/TEN. Thus, an inter-disciplinary integrated approach is crucial for effective management case with dental anomalies due to Steven's Johnson syndrome. Table 4 outlines a structured assessment protocol that can be used in outpatient follow-up during the post-acute period. The true extent of chronic oro-dental aberrations and complications in survivors of SJS/TEN needs to be mapped out in a large, collaborative, prospective study.

Conflict of Interest

The authors declare that there are no conflicts of interest.

Acknowledgements

The authors declare that no funding or support was received from 3rd parties for this work.

Table 4. Assessment protocol for in outpatient follow-up during the post-acute period of SJS/TEN for early diagnosis and referral to the specialty branch

Structure involved	Clinical presentation of lesions	Referral to the specialty branch
Skin	Dyspigmentation	Referral to Dermatology Department
	Pruritus	
	Abnormal photosensitivity	
	Abnormal sweating	
	Eruptive naevi	
	Scarring	
	Nail loss	
	Hair loss	
Eyes	Dryness	Ophthalmology Department
	Pain	
	Photophobia	
	Visual impairment	
	Eyelid dysfunction	
Mouth	Dryness of oral mucosa	Oral Medicine and Radiology
	Dental caries	
	Ulceration	
	Scarring	
	Missing tooth	Oral Surgery
	Loose mobile teeth	
	Lip adhesion	
	Short root anomaly or rhizomicry in OPG X-ray, spacing	Orthodontics/pedodontics Endodontics/prosthodontics
	Periodontal problems	Periodontics
Others	Fatigue	General Medicine and Department of Psychiatry
	Malaise	
	Sleep problems	
	Depression	
	Anxiety	
	Dysthymia	

Orthopantomogram (OPG).

References

1. Roujeau JC, Stern RS. Severe adverse cutaneous reactions to drugs. N Engl J Med. 1994 Nov 10;331(19):1272–85.
2. Roujeau JC, Guillaume JC, Fabre JP, Penso D, Fléchet ML, Girre JP. Toxic epidermal necrolysis (Lyell syndrome). Incidence and drug etiology in France, 1981-1985. Arch Dermatol. 1990 Jan;126(1):37–42.
3. Bastuji-Garin S, Rzany B, Stern RS, Shear NH, Naldi L, Roujeau JC. Clinical classification of cases of toxic epidermal necrolysis, Stevens-Johnson syndrome, and erythema multiforme. Arch Dermatol. 1993 Jan;129(1):92–6.
4. Yang M-S, Lee JY, Kim J, Kim G-W, Kim B-K, Kim J-Y, et al. Incidence of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis: A Nationwide Population-Based Study Using

National Health Insurance Database in Korea. PLoS One. 2016; 11(11): e0165933.
5. Lee HY, Walsh SA, Creamer D. Long-term complications of Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN): the spectrum of chronic problems in patients who survive an episode of SJS/TEN necessitates multidisciplinary follow-up. Br J Dermatol. 2017;177(4):924–35.
6. Bequignon E, Duong TA, Sbidian E, Valeyrie-Allanore L, Ingen-Housz-Oro S, Chatelin V, et al. Stevens-Johnson syndrome and toxic epidermal necrolysis: ear, nose, and throat description at acute stage and after remission. JAMA Dermatol. 2015 Mar;151(3):302–7.
7. Revuz J, Penso D, Roujeau JC, Guillaume JC, Payne CR, Wechsler J, et al. Toxic epidermal necrolysis. Clinical findings and prognosis factors in 87 patients. Arch Dermatol. 1987 Sep;123(9):1160–5.
8. Oplatek A, Brown K, Sen S, Halerz M, Supple K, Gamelli RL. Long-term follow-up of patients treated for toxic epidermal necrolysis. J Burn Care Res Off Publ Am Burn Assoc. 2006 Feb;27(1):26–33.
9. Sedghizadeh PP, Kumar SKS, Gorur A, Mastin C, Boros AL. Toxic epidermal necrolysis with a rare long-term oral complication requiring surgical intervention. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008 Apr;105(4):e29-33.
10. Royan SJ. Lip adhesion: unusual complication of Stevens-Johnson syndrome. J Oral Maxillofac Surg Off J Am Assoc Oral Maxillofac Surg. 2010 Apr;68(4):901–3.
11. Gaultier F, Rochefort J, Landru M-M, Allanore L, Naveau A, Roujeau J-C, et al. Severe and unrecognized dental abnormalities after drug-induced epidermal necrolysis. Arch Dermatol. 2009 Nov;145(11):1332–3.
12. Roujeau JC, Phlippoteau C, Koso M, Wechsler J, Andre C, Binaghi M, et al. Sjögren-like syndrome after drug-induced toxic epidermal necrolysis. Lancet Lond Engl. 1985 Mar 16;1(8429):609–11.
13. Chosidow O, el Wady Z, Devanlay M, Jaffray P, Revuz J, Roujeau JC. Hyperamylasemia in toxic epidermal necrolysis. Arch Dermatol. 1993 Jun;129(6):792–3.
14. De Man K. Abnormal root development, probably due to erythema multiforme (Stevens-Johnson syndrome). Int J Oral Surg. 1979 Oct;8(5):381–5.
15. Ranalli DN, Elliott MA, Whaley TM, Campagna ED Jr. Stevens-Johnson syndrome: report of case with abnormal root development. ASDC J Dent Child. 1984 Jul-Aug;51(4):298-301.
16. Brook U. Stevens-Johnson syndrome and abnormal root development: a case report. Int J Paediatr Dent. 1994;4(2):101–3.
17. Bajaj N, Madan N, Rathnam A. Cessation in root development: Ramifications of 'Stevens-Johnson' syndrome. J Indian Soc Pedod Prev Dent. 2012 Jul 1;30(3):267.
18. Sangwan A, Saini H-R, Sangwan P, Dahiya P. Stunted root development: A rare dental complication of Stevens-Johnson syndrome. J Clin Exp Dent. 2016;8(4):e462–4.
19. Stevens-Johnson Syndrome: A Case Report. J Korean Acad Pediatr Dent. Accessed in 2021. Available from: http://journal.kapd.org/journal/view.php?number=1549
20. Barea-Jiménez N, Calero J, Molina-Negrón D, López Del-Valle LM. Treatment for oral lesions in pediatric patients with Stevens-Johnson's syndrome: A case report and literature review. Int J Paediatr Dent. 2020 Jul;30(4):489–96.
21. Valladares Neto J, Rino Neto J, de Paiva JB. Orthodontic movement of teeth with short root anomaly: Should it be avoided, faced or ignored? Dent Press J Orthod. 2013 Dec;18(6):72–85.
22. Vishwanath M, Chen P-J, Upadhyay M, Yadav S. Orthodontic management of a patient with short root anomaly and impacted teeth. Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod. 2019 Mar;155(3):421–31.