Section: Paediatrics

Case Series

VESICULOBULLOUS DISORDERS IN PAEDIATRIC PATIENTS: A CASE SERIES OF UNCOMMON CONDITIONS

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ABSTRACT

Background: Vesiculobullous disorders are a heterogeneous group of disorders characterised by the presence of vesicles, bulla, erosions on skin or mucous membrane or both (1). Depending upon etiopathogenesis, these disorders may be inflammatory, infective, autoimmune, drug-induced, or genetic in origin (1,2). Infections are the most common cause of vesiculobullous diseases. **Materials and Methods:** Five children diagnosed with uncommon vesiculobullous disorders with variable clinical profile were selected for the study at BPS GMC (W) Khanpur, Kalan. In this case series, we describe these five uncommon vesiculobullous diseases of different pathogenesis in paediatric patients. **Result:** These five cases represent varied spectrum of vesiculobullous disorders with different clinicopathological features presenting at rural college of Haryana. **Conclusion:** Vesiculobullous disorders are common in children, with infection being the most common cause. Vesiculobullous disorders in children form a challenging task for treating clinicians due its varied presentation. Knowledge of clinical features and bedside procedures, along with skin biopsy and other procedures, helps establish the diagnosis and reduce morbidity and mortality. Our series gives insight into various causes of rare vesiculobullous conditions in paediatric patients.

INTRODUCTION

Vesiculobullous disorders are a heterogeneous group of disorders characterised by the presence of vesicles, bulla, erosions on skin or mucous membrane or both.[1] Vesicles is small fluid filled blister of size ≤ 5mm and bulla is fluid filled blister of size > 5mm. These are formed by acantholysis, hydropic degeneration, or basement membrane zone disruption leading to separation of skin layers and subsequent accumulation of fluid. Depending upon level of fluid accumulation vesicles can be flaccid or tense. Based upon etiopathogenesis, these disorders may be inflammatory, infective, autoimmune, drug-induced, or genetic in origin.[1,2] It can either be very benign or potentially fatal in some cases. Infections are the most common cause of vesiculobullous diseases. These diseases differ between children and adults mainly in underlying cause, presentation, and prognosis. In this case series, we describe five uncommon vesiculobullous diseases of different pathogenesis in paediatric patients.

Case 1- Toxic Epidermal Necrolysis:
Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare but life-threatening dermatological emergencies. Presently, SJS and TEN are regarded as two ends of the same spectrum of disease with varying degrees of severity categorised based on skin detachment relative to the body surface area (BSA): SJS is less severe with <10% BSA, SJS/TEN overlap (10–30% BSA), and TEN (>30% BSA).[3]
Case summary: We report a case of a 7 years old male child, a known case of epilepsy currently on carbamazepine, who presented with acute onset of extensive rash over face, chest, back, upper extremities, and oral mucosa for one day. On examination, diffuse dusky oedematous patches and erosions were present over the affected area. Mucosal erosions were present over the lips with bilateral conjunctival hyperaemia with purulent discharge [Figure 1a]. Nikolsky sign was positive. There was no significant family history or history of any drug allergy. Based on history, clinical findings and extent

of involvement, a diagnosis of SJS/ TEN overlap due to carbamazepine was made. The patient was admitted to the PICU & treated with supportive care and daily wound dressing. The offending drug was stopped, and the patient was started on I.V fluids, systemic steroids and antibiotics. Eyes were washed daily with normal saline, and carboxymethylcellulose eye drops were instilled Q6hrly. The dose of steroids was tapered over 10 days. Lesions gradually healed with hypopigmentation; no scarring was seen [Figure 1b].

Anticonvulsants, nonsteroidal anti-inflammatory drugs, sulphonamides, and antibiotics are among the drugs that may cause TEN.[4] Among anticonvulsants, carbamazepine is responsible for the majority of the instances.[4,5]

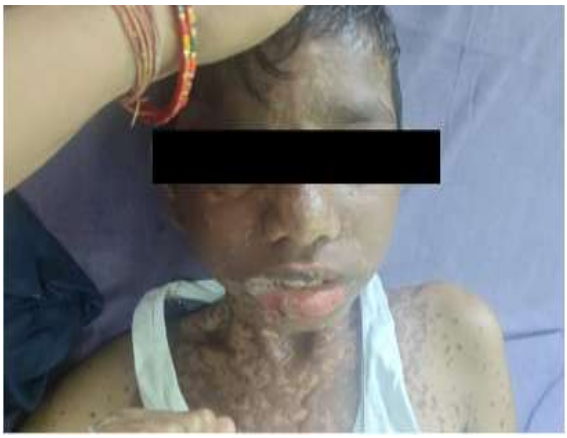

Figure 1a: showing involvement of skin and mucosa in patient with SJS/ TEN

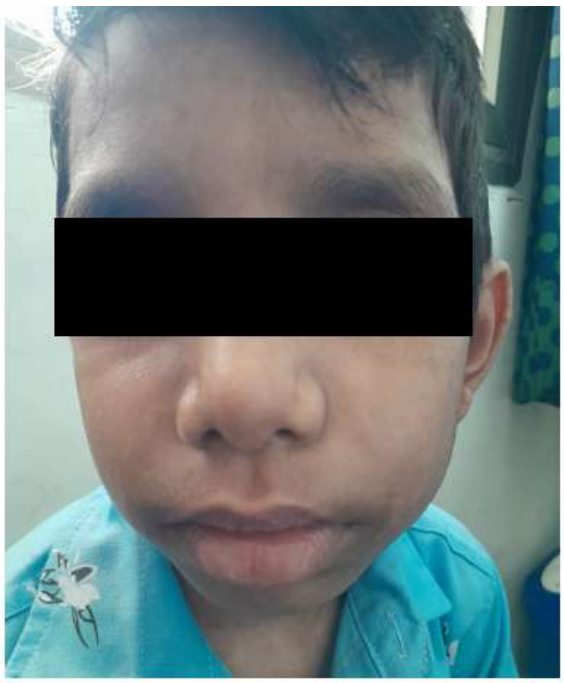

Figure 1b: complete healing with hypopigmentation on follow-up

Case 2- Epidermolysis bullosa

Epidermolysis bullosa is a heterogeneous group of inherited mechanobullous disorders characterised by skin and mucosal fragility, resulting in blister formation with minimal trauma.

Case summary: A 10 days old female child born from a non-consanguineous marriage to a primigravida mother by normal vaginal delivery presented with fluid-filled blisters and erosions since birth. Lesions developed with minor trauma over friction sites. On examination, blisters and erosions were present over the upper limbs, involving the elbow joint and extending to the dorsum of the hands; the lower limbs, below the knee, involving the soles; and the lips. Nails were spared (Figure 2). Systematic examination was normal. The patient was taking adequate feed. Routine investigations, including non-invasive prenatal testing, were normal. There was no family history of similar blistering disease. Parents declined biopsy and genetic testing due to financial constraints. Based on history and clinical examination, a diagnosis of epidermolysis bullosa was made. The patient was managed conservatively with IV antibiotics, adequate wound care with bland paraffin gauze, and proper nutritional support. The parents were counselled regarding the child's prognosis and further care.

There is presently no definitive cure for epidermolysis bullosa. Treatment aims to alleviate symptoms and provide supportive measures. Therapy is therefore focused on preventing and halting the progression of skin lesions and their complications. Nutritional support is important for adequate growth and development and to promote optimal wound healing. For families of affected children, prenatal diagnosis using molecular techniques offers genetic counselling. A definitive diagnosis of EB and its subtype is obtained through genetic mutation analysis or skin biopsy for IFM and EM.[6] It is imperative to establish an accurate diagnosis as there are extreme differences in the care and outcomes of patients along the spectrum of EB: from severe generalised disease with a poor prognosis to mild localised disease. However, a diagnosis of the type of EB does not always predict the prognosis. It is important to set expectations with the affected family and, in severe cases, help the family decide the extent of care to be provided.[7]

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Figure 2: epidermolysis bullosa

Case 3: Herpes Zoster

Herpes zoster (HZ) is viral infection presenting as a vesicular rash in the cranial nerve or dorsal root ganglia of latent varicella zoster virus (VZV).[8] The diagnosis of Herpes Z is typically based on clinical findings.[9] It is seen in single dermatome, usually in old age or patients with underlying diseases or immunosuppression, but it can also affect individuals of all ages, including those with a healthy immune system. We present a case of 12-year-old immunocompetent boy presenting with herpes zoster.

Case summary: A 12 years old healthy male child presented with a painful rash on the left side of his body for 3 days. Three days back, he was apparently well when he started having burning pain over the left side of his trunk which was followed by the appearance of a fluid-filled lesion over the left side of the body. The associated pain was severe and was localised to the affected area. There was a history of chickenpox at 4 years of age. No history of any drug intake or insect bite was present. The child's general condition was good. On local examination, grouped vesicular lesions were present unilaterally along T8/T9 dermatome [Figure 3]. Systemic examination was within normal limits. There were no haematological abnormalities or any evidence of immunosuppression. The differential diagnosis included herpes zoster and insect bite reaction. Based on history and clinical examination, herpes zoster was diagnosed. He was immediately started on oral acyclovir and analgesics. The patient returned for follow-up on day 7 with scab formation. At the 2-week follow-up, the lesions had completely healed, with no complications.

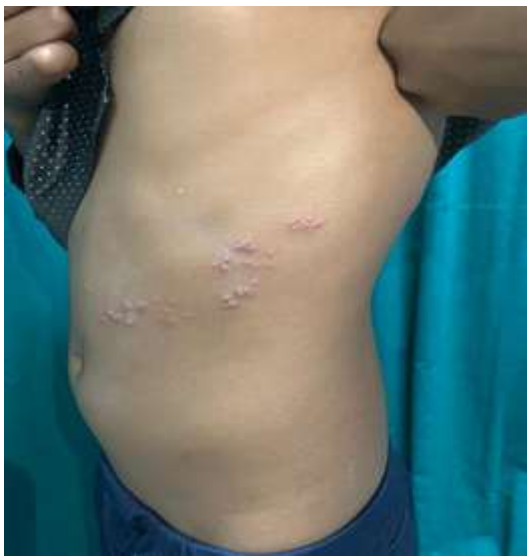

Figure 3: Herpes zoster involving left T8/T9 dermatome

Case 4- Primary herpetic gingivostomatitis (PHGS)

Primary acute herpetic gingivostomatitis is a common pattern of symptomatic primary herpetic infection caused by HSV-1.[10] In most cases, primary HSV-1 infection goes unnoticed and later presents as cold sores or herpes labialis. This condition may be preceded by prodromal symptoms such as fever, chills, headache, oral pain, enlarged lymph nodes, and decreased appetite (10). We present a case of a 10-year-old female suffering from acute herpetic gingivostomatitis with severe prodromal symptoms

Case summary: A 10-year-old female patient presented with chief complaints of fever for 5 days and painful ulcers in the mouth and lips for 3 days. She was apparently well five days ago when she developed fever which was high in grade and was not associated with chills and rigor. After two days she developed painful ulcers over lips. She also complained of difficulty in eating and drinking. There was no history of drug intake. On examination, multiple shallow oval ulcers were seen in the oral cavity, gingiva, and palate, with erosions and crusting over the lips [Figure 4]. Cervical lymph nodes were enlarged. Other areas were not involved. Differential diagnoses included primary herpetic stomatitis, aphthous ulcer, hand-foot-and-mouth disease, herpes zoster, and erythema multiforme. Based on clinical history and findings, a final diagnosis of primary herpetic gingivostomatitis was made. She was immediately started on acyclovir 400 mg, 1 tab TDS, and oral analgesics, after which her fever subsided and lesions healed over 7 days. She gradually started talking orally and was discharged.

PHGS is caused by infection with Herpes Simplex Virus (HSV1), but in some cases by HSV2.[11] In our case, we did not perform HSV serology due to financial constraints. It must be differentiated from hand, foot, and mouth disease, where we find papulovesicular lesions in the palms, soles, and buttocks as well. On the other hand, aphthous stomatitis runs a chronic course and is characterised

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by a few discrete oral ulcers usually involving movable mucosa like lips and cheeks.

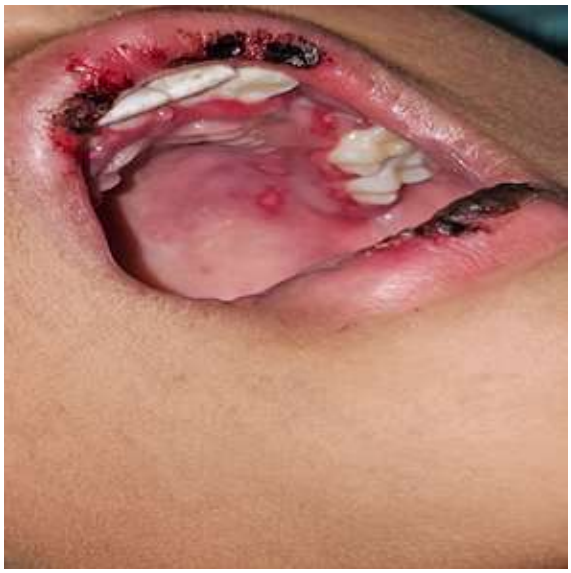

Figure 4: showing multiple shallow ulcers over palate gingiva and lips

Case 5- Linear IgA bullous dermatosis (LABD)
Linear IgA bullous dermatosis (LABD) is a rare autoimmune disorder that is characterized by the presence of itchy vesicular lesions arranged in a string-of-pearl or cluster-of-jewels appearance clinically and by linear deposits of immunoglobulins (IgA) at the basement membrane zone in Direct immunofluorescence.[12,13] It affects both adults and children, being more frequent in the latter, typically between 4 and 5 years of age.[12,13] We report a case of LABD in a girl child diagnosed clinically and confirmed by skin biopsy and direct immunofluorescence (DIF).

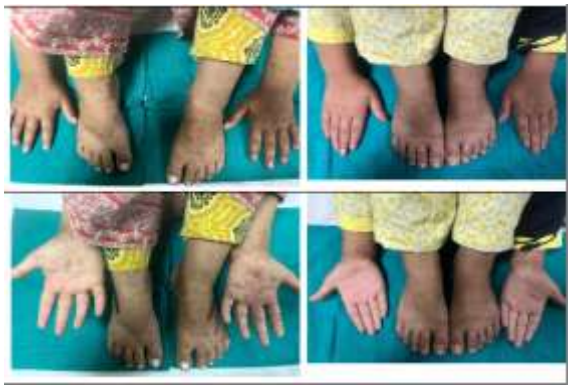

Figure 5a &b – Pre treatment. 5c & d- post treatment

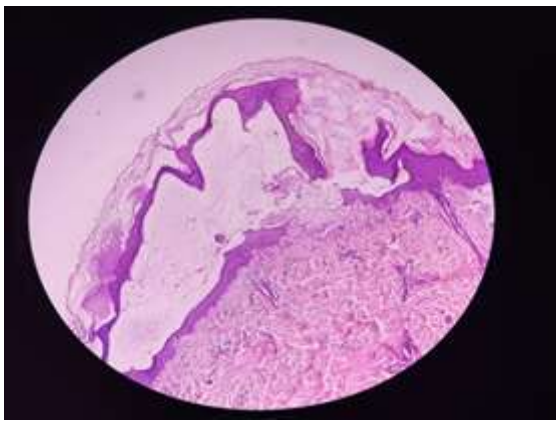

Figure 6: HPE showing spongiotic subepidermal bulla

Case summary: An eight years old girl child presented with multiple fluid-filled lesions over her trunk and extremities for one year. The lesions had an insidious onset and were itchy. There was no family history of such lesions. On examination, multiple tense vesiculobullous lesions were present over the extremities with a cluster of jewels (arranged in an annular or herpetiform pattern) appearance over the bilateral palms and dorsum of feet [Figure 5a, 5b]. The vesicles were tense on palpation. Excoriated lesions were also present over the trunk. Her systemic examination was with in normal limits. Routine investigations were normal. Acantholytic cells were absent in Tzanck smear. Differential diagnosis of LABD, Dermatitis herpetiformis and bullous pemphigoid were made. Histological examination showed a spongiotic subepidermal bulla containing neutrophils, a few eosinophils, lymphocytes & slight perivascular lymphocytic infiltrate, and mild oedema in the dermis [Figure 6], and DIF showed IgA linear deposits at the dermo-epidermal junction characteristic of LABD [Figure 7]. Patient was started on antihistaminic, topical steroids and oral dapsone after testing for G6PD deficiency. On follow-up, the patient showed significant improvement
[Figure 5c, 5d].
LABD is one of the rarer subepidermal autoimmune blistering diseases in pediatric age group. Its diagnosis requires expertise and a high degree of suspicion.

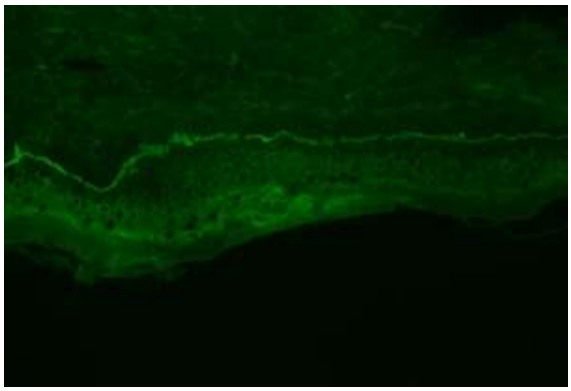

Figure 7: DIF showing IgA linear deposits at dermo-epidermal junction

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DISCUSSION

Vesiculobullous diseases cause significant morbidity and mortality if the correct diagnosis is not made at the proper time. Recognition of morphology, distribution of vesicles/bullae, onset help in distinguishing infectious, allergic, drug induced, genetic and autoimmune conditions.[1,2] SJS/TEN is one such condition where prompt medical intervention helps in reducing significant mortality as it causes extensive skin loss, fluid and electrolyte imbalance, infection and death. So offending medication should be immediately stopped. Epidermolysis bullosa and SJS/TEN both cause blistering and erosions, but they differ in aetiology, presentation, and prognosis. SJS/TEN is immune-mediated, mostly drug-induced or infectious in origin, acute in presentation, and rapidly progressive; epidermolysis bullosa is a mechanobullous disorder, genetic in origin, present at birth or infancy, involving sites of friction, and running a chronic course. It is caused by mutations in genes encoding proteins responsible for epidermal dermal adhesion. Defects in keratin 5 /14 cause epidermolysis bullosa simplex, defects in laminin-332 or collagen XVII cause junctional EB and defects in collagen type VII cause dystrophic EB.[6] So genetic testing is indicated . Herpes zoster caused by VZV is a painful condition, dermatomal in distribution, and have flaccid vesicles and multinucleate giant cells on Tzanck smear. It is caused due to reactivation of VZV in the dorsal root or cranial nerve sensory ganglia, usually when cell mediated immunity decreases, however it may uncommonly involve children with early age varicella infection. Thoracic dermatome is most common involved because of extensive distribution of thoracic sensory ganglion. PHGS has to be differentiated from herpes zoster of oral cavity. Herpes zoster of oral cavity present with severe burning or neuralgic pain followed by unilateral vesicles and ulcers confined to a trigeminal dermatome, usually not crossing the midline. Gingival involvement is localized to the affected side. In contrast, primary herpetic gingivostomatitis presents mainly in children with fever, malaise, lymphadenopathy, diffuse painful gingivitis, and widespread oral vesicles and ulcers involving both sides of the mouth. PHGS is also to be differentiated from herpangina where small vesicles or ulcer are seen mainly over soft palate, tonsillar pillars uvula with no gingivitis. In contrast to painful grouped vesicular lesions of herpes zoster, LABD is characterised by itchy, tense grouped vesicles often annular or string of pearl appearance, primarily over the trunk and extremities. Itchy vesicular tense lesions are also seen in dermatitis herpetiformis. Skin biopsy in such autoimmune disease may show intraepidermal or subepidermal blister, however it cannot identify the specific disease. In both LABD and dermatitis herpetiformis (DH) subepidermal blister is seen in histopathology. In such cases DIF is contributory as it demonstrates characteristic immune deposition. In LABD linear IgA deposition is seen along basement membrane zone in contrast granular IgA deposition is seen in dermal papillae in DH.[14] Thus, DIF is highly valuable diagnostic gold standard test for autoimmune vesiculobullous diseases. Thus, even with similar morphology, we may have different diagnoses.

CONCLUSION

Vesiculobullous disorders are common in children, with infection being the most common cause. Other causes include metabolic, drug-induced, and autoimmune. Vesiculobullous disorders in children form a challenging task for treating clinicians due its varied presentation. Knowledge of clinical features and bedside procedures, along with skin biopsy and other procedures like DIF, helps establish the diagnosis and reduce morbidity and mortality. Our series gives insight into various causes of rare vesiculobullous conditions for paediatricians and dermatologists.

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