# Approach to a Neonate with Vesiculo-Bullous Lesions

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## Introduction:

- Infections, congenital disorders, or other diseases can produce vesicles, bullae, and pustules in newborns. Erythema toxicum neonatorum, transitory neonatal pustular melanosis, and newborn acne are examples of benign and self-limited illnesses that do not require particular treatment.
- Certain infections and genetic abnormalities, on the other hand, must be distinguished from these self-limiting illnesses since treatment may be required.
- This article discusses benign pustular eruptions, vesiculopustular eruptions caused by infections, and congenital/inherited bullous disorders that manifest in newborns.

**Table. 1 Classification of Neonatal Blistering Disorders**

**Neonatal blistering disorders:**

***Infection* Bullous:**

Impetigo

Staphylococcal scalded Syndrome (SSSS)

***Inflammatory dermatosis***

Erythema Toxicum Neonatorum (ETN)

Transient pustular melanosis (TPM)

Miliaria

***Antibody mediated***

Pemphigus Vulgaris

Bullous Pemphigoid (herpes gestationis )

***Genodermatosis***

Bullous CIE

IncontinentiaPigmenti

Kindler Syndrome

EB - Junctional / Dystrophic

***Metabolic***

Acrodermatitis Enteropathica

## Inflammatory conditions :

### Erythema toxicumneonatorum :

- Most commonly seen usually between first day and 4$^{th}$ day of life and lasts about 3days . It is an evanescent rash,resemblingfleabite.
- Clincalpicture :erythematous macules with central vesicle or pustulewhich contains eosinophils .
- Diagnosis is done by clinical characteristic picture or scraping of pustule showing eosionpils . Treatment not required .

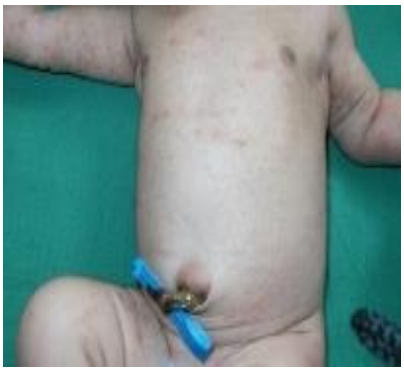


*Fig. 1 Erythema toxicum neonatorum1*

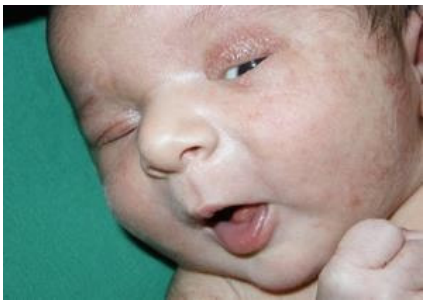


*Fig. 2. Erythema toxicum neonatorum2*



## Transient pustular melanosis

- This entity is characterised by pustules present at bitrth and evolve into araes of macular pigmentation. Pustules are neutrophilic Pigmentation lasts longer than usual.
- Clinical picture :pustules present at birth located mainly around chin neck and frictional sites. Clusters present at pressure areas. Typically the vesiculopustules coexist with pigmentation.There is no erythema .The vesicles rupture easily and form collarette scaling.
- Treatment not required .

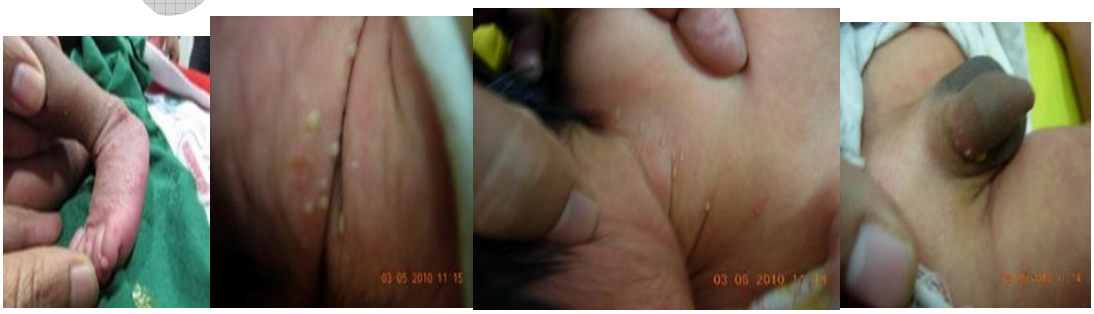


*Fig. 3. transient pustular melanosis*

## Miliaria

- Miliaria crystallina is commonin summer months and is alsonoted in infants housed in incubators.
- Clinical picture :It is characterised by superficial 1-2mm clear noninflammatory vesicles which are asymptomatic . Mostly the lesions are seen on forehead and on sixth or seventh day of life .transclucent vesicles are characteristic .
- Miliaria rubra usually occurs in later age .
- Treatment : Remove the baby from warm /humid nvironment. Cool bathing and air conditioning are best therapeutic measures .

*Fig. 4. miliaria*

## Infectious causes:

### Bullous impetigo :

- Usually caused by Staphylococcus aureus.
- Clinical picture :flaccid bullae with purulent fluid surrounded by erythema usually clinches the diagnosis .
- When The lesions are without erythema it is usually caused bystreptococci. Nursing staff carrying the organism usually spread the disease.

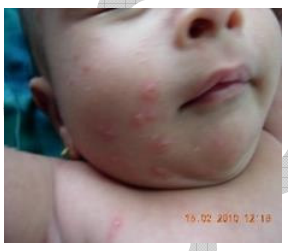
### SSSS: Staphylococcal scalded skin Syndrome:

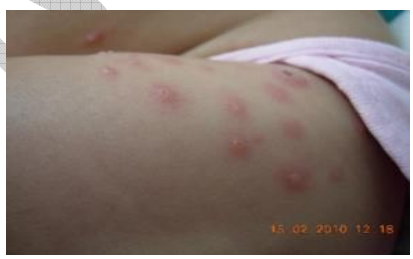
- As the name suggests it is caused by Staphylococcal infection .It is the exotoxin produced by organism which causes the skin lesions .

- Aetiology :ET A ET B and ET D are the toxins causing disruption at desmoglein 1 protein
- .This leads to separation of epidermal layers causing vesicles .Large area of involvement shows wrinkling of skin with eroded areas beneath because the split occurs in granular layer.
- The culture is negative as the lesions are caused by exotoxin. Usually the primary site of infection is umbilical stump or eyes .the culture from these sites may come positive .
- Treatment : as most of the time the organism is MRSA vancomycin is the drug of choice . ceftriaxone or penicillinase resistant penicillin is used .
- General measures to prevent dehydration local paraffin dressings are used .Healing occurs without scarring .

**Varicella:**

- Infection in neonate can occur because of maternal varicella 7days before the delivery or immediately after delivery.
- The neonate is born with vesicles or develops vesicular rashes in 1$^{st}$ week of life Treatment:
- Varicella zoster Immunoglobulin is given to new born in infection in motherjust before delivery.





- Local care with Calamine lotion to dry out the lesions .

*Fig. 5 Varicella*

**Neonatal herpes Infection:**

- It is severe and often fatal disease of neonates contracted through vertical transmission of HSV during vaginal delivery.It tends to manifest within first 4 weeks of life mostly first week. HSV 2 accounts for 75-80% of infections .Multiple grouped and disseminated vesicles occur along with erosions and ulcers especially at places of trauma .

- Treatment should be directed to prevention as no antiviral cures the disease.

## Congenital Bullous disorders :

- Immunobullous disorders are rare. They are acquired by passive transfer of maternal antibodies .
- Maternal history of blisters is relevant in infants born to women with pemphigus or herpes gestationis (bullous pemphigoid of preganancy ).
- Infants born to women with pemphigus are at the most risk whereas bullous pemphigoid is extremely rare in neonates

## Genodermatoses:

**Bullous Congenital Ichthyosiform Erythroderma** ( epidermolytic hyperkeratosis , BIE)

- This autosomal dominant condition is distinct from other congenital ichthyosis because of blister formation.
- Aetiology :In BIE there are mutations in epidermal keratin1 or keratin 10 genes resulting in abnormal keratin intermediate filament cytoskeleton . This results in abnormal clumping of suprabasal keratinocytes and impaired tonofilament network in differentiating epidermal cells.
- Clinical Picture :Genaralised flaccid bullae and extensive areas of denudation are present at birth.This is provoked by friction resulting from passage through birth canal.With time vesiculation decreases and hyperkeratotic skin lesions supervene. Episodes of focal vesiculation are common later replaced by dirty warty keratosis .

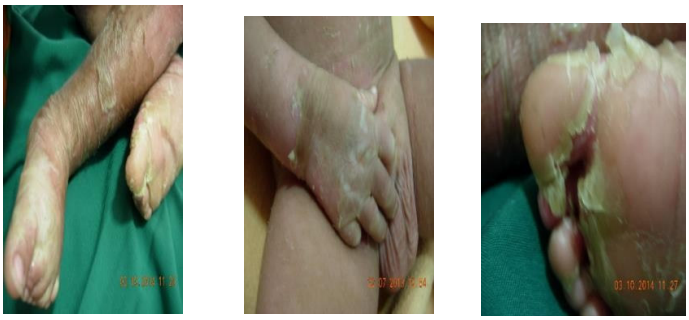


*Fig. 6. Epidermolysis hyperkeratosis*

- Management :Barrier nursing

Avoidance of mechanical trauma

Soft nonocclusive clothes

Erosions treated with paraffin soaked dressings

Protein rich diet and oral fluid supplementation

Use of ophthalmic lubricants

Monitor for sepsis

**Other genodermatosis which may present at birth with vesicles and erosions include**

**Incontinentiapigmenti (IP)**

- IP is a rare genodermatosis with x linked dominant inheritance
- It has three distinct morphologies :1 Vesiculobullous
  2. Verrucouspapules
  3. Whorledhyperpigmentation
- Aetiology :X linked dominant linkage showing mutation in gene for NEMO which is central to many immune ,inflammatoryand cell death pathways in the cell and required for the activation of the transcription factor NK-kB.
- This disorder is usually lethal in utero for males so affected infants are females .History of previous spontaneous abortions may be elicited in families with this disorder .

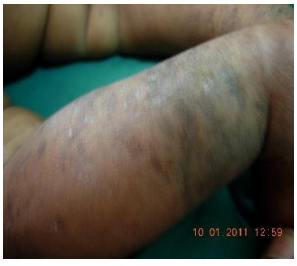


*Fig. 7. Pigmented IP*

**Kindler Syndrome** :

- It is another rare genodermatosis with congenital presentation of blisters at sites of trauma typically acral surfaces and persistent skin fragility throughout childhood . In addition to fragility patients develop poikilodermaand photosensitivity.
- Aetiology The defect in kindler syndrome results from unique abnormality in actin - cytoskeletonassociated protein - fermitin family homologue.

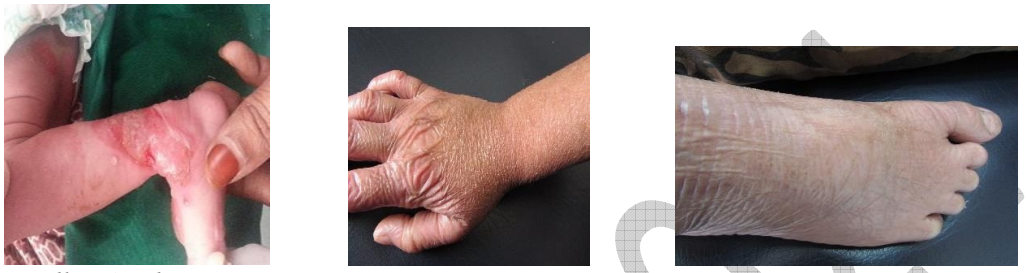


*Fig. 8. Kindler Syndrome*

**MechanobullousDisorders** :

- Epidermolysis bullosa Is the name given to a group of genetically detremined disorders characterised by excessive susceptibilty of the skin and mucosae to separate from underlying tissues following mechanical trauma .There are four broad categories depending on the level of split within the skin

They are mainly termed
Epidermolytic corresponds to Simplex
Lucidolytic corresponds to Junctional
Dermolytic corresponds toDystrophic

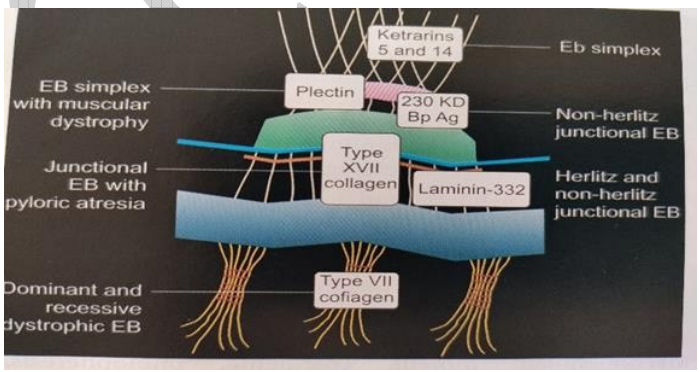



*Fig. 9. Diagramatic illustration*

Aetiology: It is true epidermolysis i.e.intracellular keratinocyte lysis .It is related to gene

mutation .The position of mutations within these genes is important in determining severity of the resulting disease.

- Red flag :Attempt to classify the type of Eb by clinical presentation can be deceptive .as Doweling Meara type of EB simplex variety may prove fatal as sepsis sets in earlier and oral lesions are more common may be mistaken for other form of EB .
- Particularly junctional and dystrophic Epidermolysis Bullosa are often present at birth or shortly after with bullae and erosions .The presence of scarring and milia indicates the intrauterine blistering has occurred .
- Infants with EB may have mucosal involvement as well as extensive cutaneous involvement and are at risk for sepsis fluid and electrolyte abnormalities ,difficulty in feedingand thermal regulation .

**Junctional EpidrmolysisBullosa** :

- Rare Autosomal recessive in all forms
- Plane of cleavage is lamina lucida of basement membrane zone .
- Clinical picture :generalised tense bullae abd widespread mucosal involvement . Sometimes with hoarseness of voice and Sometimes pyloric atresia.

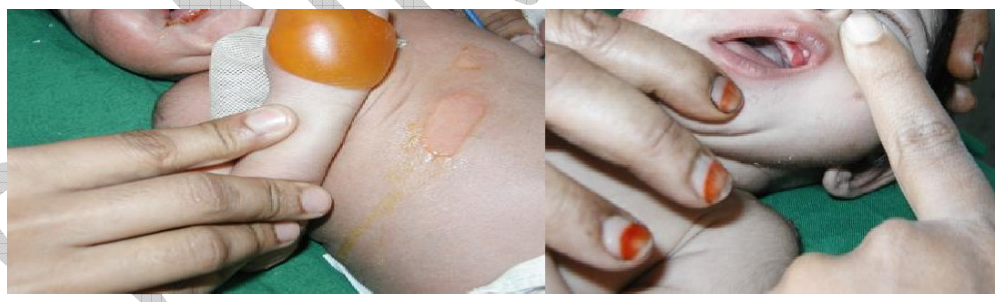


*Fig. 10. Junctional Epidermolysis Bullosa*

**Dystrophic Epidermolysis Bullosa** :

- This variant is characterised by marked scarring and milia formation and deformities. These follow AD or AR transmission.
- Aetiology: The plane of cleavge is sublaminadensa of basement membrane zone .Mutations in type the gene for type VII collagen (COL7A1)on 3p21.1
- Classical sites of involvement in dominant dystrophic are bony prominences on

extremities.

- Healing with atrophy and scarring and milia formation.
- Recessive type are severe scarring form of disease. Generalized vesicles and bullae present at birth. There is sever mucosal and cutaneous scarring and loss of nails.

*Fig. 11. Dystrophic Epidermolysis Bullosa*

**Diagnosis** :

- Simple treatable conditions should be excluded at birth.
- When clinical diagnosis is made it may be difficult to determine the type.
- Simple biopsy of a fresh vesicle within 24 hrs of eruption should be done .Electron microscopy helps in determining the site of clevage .
- Antigen mapping at the level of split by immunohistochemical study is helpful.

**Treatment :**

- All forms of EB are mainly treated symptomatically and the aim is to prevent appearance of new lesions, infections ,contractures and deformity .

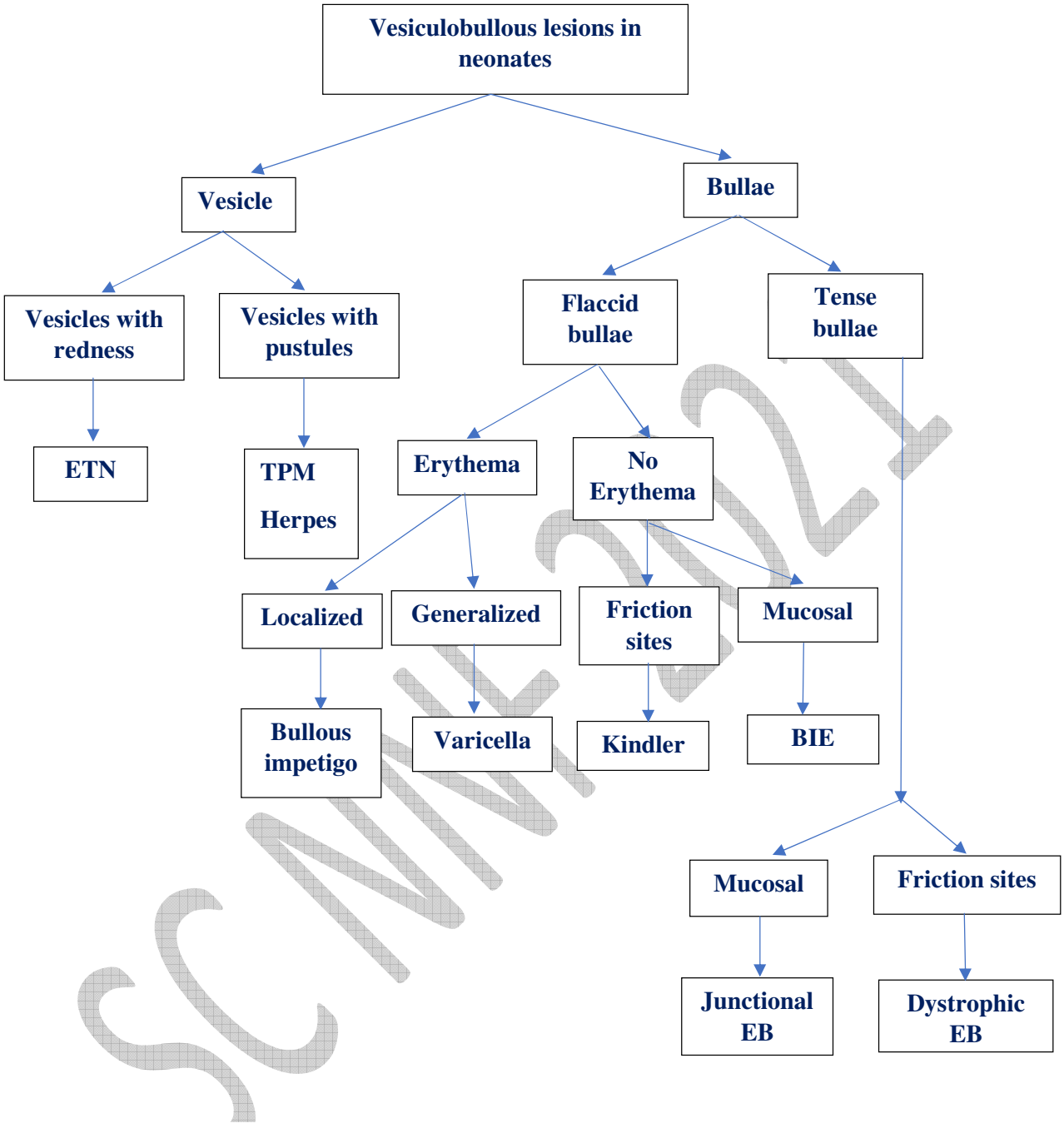



**Fig. 12. Approach to a neonate with vesiculobullous lesions**