Chapter 15

# Acute Fever with Rash

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## INTRODUCTION

Fever with rash is many times a challenge for pediatricians as there is an exhaustive list of causes that can be broadly classified as infective and noninfective. Infective causes are associated with acute onset of fever and majority of non-infective causes have insidious onset.

Rash in fever also adds concern and anxiety among parents and therefore many of these patients land up in emergency. Henceforth, it is very important to differentiate whether this rash is a feature of some underlying serious illness which may be associated with complications, or just a benign viral exanthem.

Most importantly as with any other case arriving in emergency, the first step is to differentiate whether the child is sick or not sick who has presented with rash along with fever. This implies, always looking at the vitals first, stabilizing the patient taking care of his airway, breathing, and circulation, and then proceed for the evaluation and management of the patient. There are some Red flag signs[1] in a patient with fever with rash which may indicate severe disease—young age, toxic appearance, hypotension or hemodynamic instability, mucosal involvement, purpuric lesions, desquamation, dyspnea, and decreased consciousness.

A thorough history, complete description and examination of rash and its clinical co-relation helps in the evaluation of the child and ordering the appropriate investigations. Before going to the list of causes of fever with rash, it is very important in an emergency to describe the morphology of rash (maculopapular, vesicular, erythematous, or purpuric), its onset and its temporal association with fever, generalized or localized, its distribution whether centrally or peripherally distributed, progression and transformation, any mucosal involvement and associated features. Also inquire in the history about any recent vaccination, exposure to animals, or any drug intake. It is important to undress the patient for examination and touch the patient with gloved hands only. Look for all areas including trunk, extremities, palms and soles, and mucus membranes. See, if lesion is raised or flat, and blanchable or not. Look for the nikolsky sign (the extension of peeling or blistering skin caused by separation of the layers of skin when firm, sliding pressure is applied to the skin) if erythroderma is present. In addition, look for the presence of lymphadenopathy, organomegaly, arthritis, any signs of meningeal irritation, or neurological dysfunction **(Flowchart 15.1)**.

It is important to isolate the patient if communicable disease is suspected and to ensure all necessary precautions.

## EPIDEMIOLOGY

The commonest cause for fever with rash in children is viral exanthems. In India, measles is a health concern with epidemics occurring in winters and early spring. There is 73% decrease in deaths due to measles between 2000 and 2018 worldwide due to improved vaccination.[2] Varicella zoster virus (VZV) has high infectivity rate of >85–90% and more cases are seen during month of January to May.[3] Unvaccinated rural population continues to witness outbreaks of chickenpox as its vaccine is not included in national immunization schedule.[4] Hand–foot–mouth disease (HFMD) another viral disease caused by coxackie virus A16 and enterovirus A71, has seasonal pattern more seen in temperate Asia in summer season and in spring and autumn season in subtropical Asia.[5] Dengue is another important cause of fever with a rash with an estimated 13,000,000 cases occurring among individuals aged 5–45 years in 2017.[6]

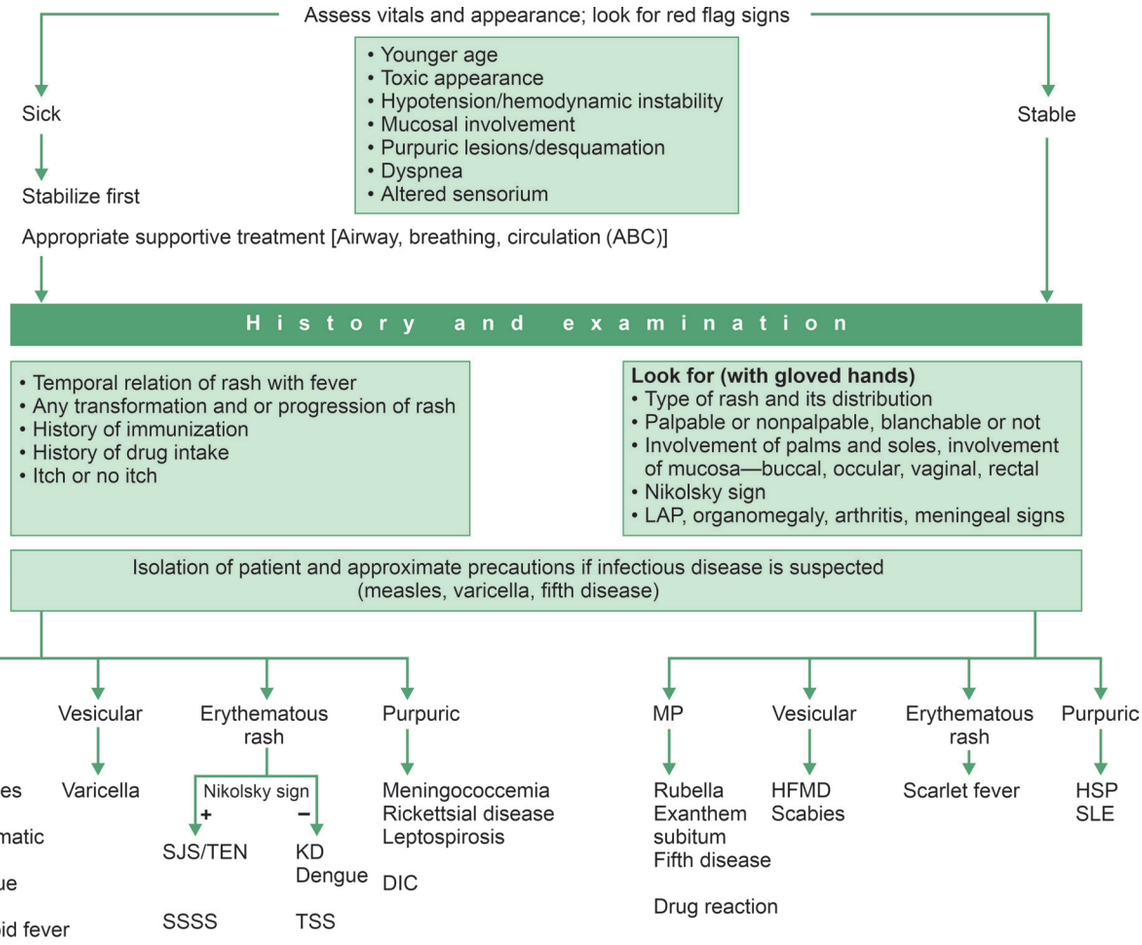
The reports of rickettsial diseases have increased in recent years from India. The cases of spotted fever group (SFG) are more seen in southern states Tamil Nadu, Karnataka and Maharashtra while scrub typhus is being reported from almost every part of India. Rickettsial diseases are more observed in May to February but can occur throughout the year.[7] Children with meningococcal infection can also present with fever and rash but are more likely to be ill, have purpura, and hemodynamic instability. The majority of outbreaks of this disease are reported from the large cities of northern and coastal areas of India.[8]

Rheumatic fever/Rheumatic heart disease (RHD) is diagnosed based on fever and rash along with a constellation of typical clinical features including carditis, polyarthritis, chorea, and subcutaneous nodules The estimated average prevalence of rheumatic fever/RHD has decreased in India to 0.5/1000 children in the age group of 5–15 years indicating economic progress of the country.[9]

## FEVER WITH MACULOPAPULAR RASH

Maculopapular rash is the most common form of rash seen with fever in children and viral exanthem is the leading cause. Most of viral exanthems are usually mild and self-limiting. Treatment is mainly symptomatic. Measles

**Flowchart 15.1:** Approach to child with fever and rash




(rubeola), erythema infectiosum (parvovirus), rubella, Infectious mononucleosis (EBV), roseola (herpesvirus 6) are some of the common viral illnesses presenting with rash. Important characteristics of these diseases are described in **Table 15.1**.

Maculopapular rash also occurs secondary to drug reaction which closely mimics viral exanthems. So, a detailed history is important to know the temporal relationship with prior drug exposure. It is also important to note that likelihood of a reaction occurring is increased in patients who have a co-existing viral infection.[16] Drugs which are commonly implicated are anticonvulsants like phenytoin, carbamazepine, benzodiazepines, phenobarbiturates, and antibiotics like penicillins, cephalosporins, sulfonamides. Onset of drug rash usually occurs after 1–2 weeks of drug administration and may persist even after the drug is discontinued because of long half-life of drug. Rash usually starts from the trunk to become generalized and is itchier. Palms and soles also get involved but not the mucus membranes. There may be desquamation and pigmentation but the patient looks nontoxic and healthy.

## Vesicular Rash with Fever

Varicella and hand foot and mouth disease (HFMD) are important causes of vesicular rash. Important characteristics of these diseases are described in **Table 15.2**.

## Fever with Erythroderma

This group of patients with erythematous rash are usually very sick and may have fatal consequences if treatment is not started in time. Dengue fever, staphylococcal scalded skin syndrome (SSSS), Steven Johnson syndrome/toxic epidermolysis necrosis, toxic shock syndrome, Kawasaki disease, and scarlet fever are the diseases which come under this category.

***Dengue fever:*** Caused by Arbovirus; spread by Aedes mosquito; is characterized by erythematous flushing rash **(Fig. 15.1)** within 24–48 hours of the onset of high-grade fever along with body aches. After about 2–3 days of defervesence another rash may appear[17] that may be maculopapular **(Fig. 15.2)** or morbilliform or may become generalized confluent petechial rash which does not blanch upon pressure, with multiple small round islets of normal skin called "white islands in a sea of red". Palms and soles are usually not affected and some children may experience generalized pruritus. Rash slowly subsides in one week. Hemorrhagic manifestation also may be seen in form of petechiae, purpura, and ecchymosis in severe form of dengue.

This virus also involves mucosa in 15–30% of patients; commonly involved are sclera, conjunctiva and oral cavity.[18] Management includes supportive treatment, fluid resuscitation especially in patients with shock, and

**Table 15.1:** Differentials of fever with maculopapular rash in children and their characteristic features

| *Disease and virus* | *Route of transmission* | *Season* | *Rash* | *Remarks* | *Complications* |
|---|---|---|---|---|---|
| Measles<br><br>Paramyxovirus (ssRNA) | Respiratory droplets and aerosolized particles that remain in the air for up to 2 hours | Late winter and spring and even in the rainy season | Appear on day 4 of illness, starts behind the ear, and spreads in a cephalocaudal manner<br>The rash resolves with desquamation and pigmentation which fades in over 10 days | Koplik spots on buccal mucosa against lower molar teeth are pathognomic of disease | Pneumonia, encephalitis hearing loss, blindness, and even death in malnourished children |
| Erythema infectiosum (fifth disease)<br><br>Parvovirus B19 | Respiratory droplets and percutaneous exposure to blood and blood products | Spring | Slapped cheek appearance, sparing perioral region and nasal folds; whole body itchy, rash lasting for weeks | Arthropathy[10] usually involves knees and ankles develops in older children and rarely develops papular purpuric gloves and socks syndrome (PPGSS)[11] | Causes transient suppression of erythropoiesis that is mild and asymptomatic except in children with underlying hemoglobinopathy where it results in transient aplastic crisis[11] |
| Rubella<br><br>Togavirus (RNA) | Respiratory droplets, vertical transmission through the placenta to fetus also | Late winter and spring | Rash on the face within 24 hours of fever, spreads rapidly to trunk and extremities and disappear by 3rd day | Posterior cervical lymphadenopathy | Congenital rubella syndrome (CRS) can occur specifically, if acquired in the first trimester of pregnancy |
| Infectious mononucleosis Ebstein-Barr virus (DNA) | Saliva | Winter and spring | MP rash may take the form of erythema multiforme or urticaria | Pharyngeal inflammation, lymphadenopathy, and hepato-splenomegaly | Airway obstruction, encephalitis, and myocarditis. Splenic rupture is rare[12–14] but lethal complication |
| Roseola infantum (exanthem subitum)<br><br>Human herpesvirus 6 (DNA) | Saliva | Spring and autumn | The rash does not appear until approximately 12–24 hours after fever (high grade lasts for 3–5 days) resolves, start from the trunk, and spread outwards | Lack of upper respiratory tract infection. No meningeal signs or encephalopathy | Increased prevalence of febrile seizures in up to 15% of infected patients[15] |

**Table 15.2:** Differentials of fever with vesicular rash in children and their characteristic features

| *Disease* | *Route of transmission* | *Season* | *Rash* | *Remarks* | *Complications* |
|---|---|---|---|---|---|
| Varicella Varicella Zoster virus | Droplet infection<br>Face-to-face contact<br>Transplacental transfer | Winter and spring | Pleomorphic rash. Appears on the trunk first on the first day of fever then involves the whole body. Oral mucosa is commonly affected. Palms and soles are spared | Self-limiting disease. Infants and immuno-compromised children have severe disease that requires acyclovir 30 mg/kg every 8 hourly for 5 days | Secondary bacterial infection because of scratching<br>Varicella pneumonia and encephalitis, acute cerebellar ataxia is seen in immuno-compromised patients |
| Hand, foot and mouth disease<br>Coxsackievirus A16<br>Enterovirus A71 | Airborne respiratory droplets, by saliva and skin-to-skin contact | Summer | Blister-like lesions on hand and feet including palms and soles are tender to touch. Oral lesions; maybe in the posterior oropharynx herpangina | All patients may not have lesions at all three sites | Dehydration due to refusal to intake<br>Cardiac and CNS complications may develop |

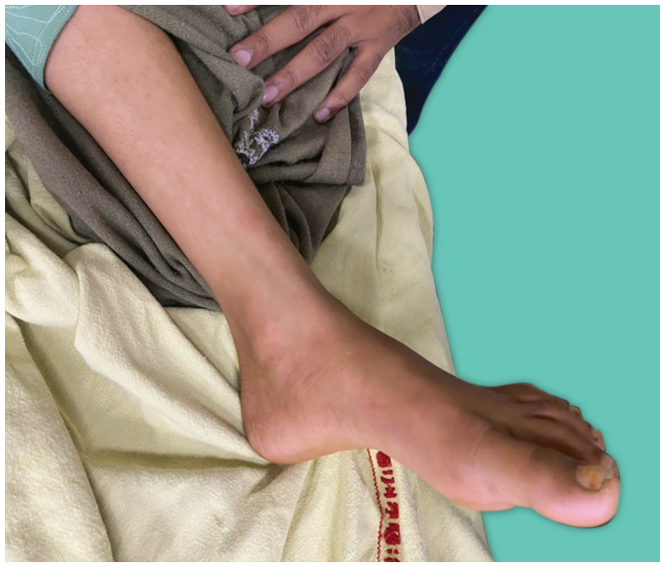


**Fig. 15.1:** Erythematous rash in dengue

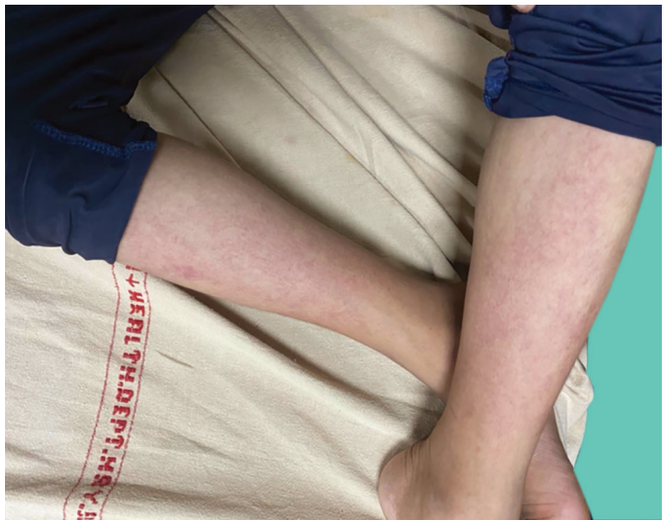


**Fig. 15.2:** Maculopapular rash in dengue

may require blood or its component therapy in case of hemorrhagic manifestation.

### Staphylococcal Scalded Skin Syndrome (SSSS)

SSSS is also called as Ritter's disease/Bullous impetigo. It is caused by the epidermolytic toxin (ET) of *Staphylococcus aureus*. This disease primarily affects neonates and young infants. In neonates, lesions are predominantly seen in the perineum and periumbilical areas while extremities are affected in older children. Large fluid filled bullae are formed along with fever and erythema which rupture with the slightest pressure (positive Nikolsky sign) to leave the extended area of denuded skin. Antistaph antibiotics (cloxacillin or vancomycin), temperature regulation, maintenance of fluid and electrolyte balance, nutritional and pain management, and skincare form the basis of management of this disease. Intact blisters are left as such to prevent further trauma to the skin. Topical eye antibiotic is also required to manage conjunctivitis.

## STEVEN JOHNSON SYNDROME (SJS)/ TOXIC EPIDERMOLYSIS NECROSIS

### Toxic Epiermolysis Necrosis

These are potentially life-threatening conditions involving skin and mucus membranes. SJS is characterized by epidermal detachment <10% of total body surface area whereas it is >30% in patients with toxic epidermolysis necrosis (TEN) **(Figs 15.3** and **15.4)**. Various drugs are identified as main culprits like penicillins, cephalosporins, sulfonamides, quinolones, allopurinol, phenytoin, carbamazepine, phenobarbitone, and nonsteroidal anti-inflammatory drugs (NSAIDs). This condition is also caused by infectious organisms like mycoplasma pneomoniae and herpes simplex.[19]

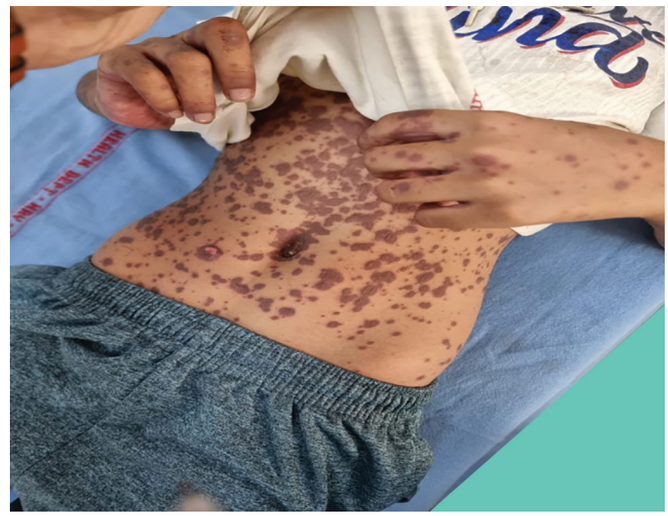


**Fig. 15.3:** Toxic epidermolysis necrosis (TEN)

**Fig. 15.4:** Toxic epidermolysis necrosis (TEN)

This disease is characterized by mucocutaneous tenderness, erythema, and severe epidermal detachment. Ocular manifestations are seen at the outset in the form of acute conjunctivitis, eyelid edema, corneal erosions to ulceration, and cicatrizing lesions. Chest and face are initially involved followed by a large area of epidermal detachment. Nikolsky's sign is also positive in this condition. Involvement of buccal, genital, and ocular mucosa occurs in >90% of cases. Sepsis is the leading cause of mortality in these patients. Management begins with immediately stopping the offending drug, meticulous skin and mucosal care, prevention of sepsis, early initiation of oral or parenteral corticosteroids (prednisolone 1–2 mg/kg/d or equivalent) for 7–10 days. Cyclosporine has also been used either alone or in combination with steroids (3–5 mg/kg/d) for 10–14 days.[20,21] Few Indian studies also found favorable results with the use of extremely low dose of intravenous immunoglobulin (<0.5 g/kg).[22–24]

### Kawasaki Disease

It is an important cause of acquired heart disease in children <5 years. KD is diagnosed based on clinical criteria of fever ≥5 days with the presence of four or more clinical features as described:

#### *Clinical Criteria for Kawasaki Disease*[25]

Fever ≥5 days (high grade and remittent type) plus any four of the following:

- The conjunctival injection is bilateral painless, non-exudative with perilimbal sparing.
- *Oral manifestations:* Bleeding, crusting, dryness, erythema, and fissuring of lips, strawberry tongue.
- Cervical lymphadenopathy (least common finding)-unilateral >1.5 cm in the anterior triangle of the neck.
- Maculopapular rash appear on day 5 of illness, starts from trunk and then spreads to extremities.
- *Extremities changes:* Erythema and edema of palms and soles followed by desquamation of fingersand toes occur after 10–20 days of onset which typically starts in the periungual region.

It is worth to note that in a given patient all findings may not be present at a single point of time, so it is important to take a detailed history of all the features[25] and exclude other diseases with similar findings. Laboratory tests provide supportive evidence but are nonspecific. Leucocytosis (neutrophilic) and thrombocytosis are usually seen in the acute phase of illness and thrombocytosis peaks by 3rd week of illness. Thrombocytopenia is a risk factor for development of coronary artery aneurysm and may be marker of macrophage activation syndrome. The role of the ECHO is to confirm or exclude cardiac involvement. Serial ECHOs are also done to evaluate the response to therapy and look for regression, persistence or progression of an aneurysm, along with any evidence of myocarditis, and valvular dysfunction.

Intravenous immunoglobulin (IVIg) (2 g/kg) is the mainstay of treatment that should be given within 10 days of illness, which reduces the risk of cardiac complications.[25] IVIg can be considered in patients with >10 days of illness if CRP and/or ESR is/are elevated or fever is persistent, or there is the presence of coronary artery aneurysm. CRP is a useful marker for treatment response. In addition, aspirin (30–50 mg/kg/d) is also given till the patient is afebrile for 48 hours in 3–4 divided doses, although its role in acute stages is controversial.[26] Aspirin dose is then tapered to 3–5 mg/kg/day and continued for 6–8 weeks and stopped. A long course of steroids (prednisolone) along with IVIg has also been suggested as per recently published Cochrane review in all children with KD.[27]

### Toxic Shock Syndrome (TSS)

TSS is toxin-mediated illness caused by *S. aureus* and streptococcal pyogenes; characterized by fever, rash, hypotension, multiorgan failure and desquamation. Its incidence is lower in children as compared to adults. This disease has rapid progressive course so early aggressive management is required. Management includes hemodynamic stabilization and appropriate antibiotics, aggressive fluid resuscitation, and inotropes remain the cornerstone. An adjuvant therapeutic strategy that includes an agent which can block superantigens like IVIg may be considered.

### Scarlet Fever (Scarlatina)

Scarlet fever (SF) caused by group A streptococci. Rash appears after 24–48 hours of upper respiratory infection along with characteristic strawberry tongue. The rash begins in skinfold (linear petechial line-Pastia lines) followed by diffuse spreading over trunk and extremities, erythematous, blanchable, and finely papular (like sandpaper), not involving the face. The rash desquamates after 3–4 days.[28,29] Penicillins or amoxicillin is the drug of choice.

## SARS–CoV2 (COVID-19 VIRUS)

Several cutaneous manifestations have been reported in children by this novel virus which includes chilblains-like lesions (or Covid toes) and viral exanthem variations (erythema multiforme-like, papulovesicular eruption, varicella-like eruption as well as prominent mucocutaneous involvement as in the more severe Kawasaki-like disease (KD) and multisystem inflammatory syndrome in children (MIS-C).[30,31] Pseudo-chilblains **(Fig. 15.5)** and Varicella-like eruptions tend to present in asymptomatic or mildly symptomatic patients within 1–2 weeks of respiratory symptoms or contact with infected persons and are self-limiting.

MIS-C is hyperinflammatory syndrome temporally associated with SARS-CoV2 virus that affects children and young adults. It is characterized by fever for >24 hours with multisystem involvement ≥2 (dermatological/renal/respiratory/hematologic/GI), with no alternative plausible diagnosis and positive for current SARS-CoV2 infection by RTPCR, or exposure to Covid-19 cases within 4 weeks of the onset of symptoms.

The close differentials of MIS-C are Kawasaki disease, TSS, and dengue with shock. MIS-C affects older children

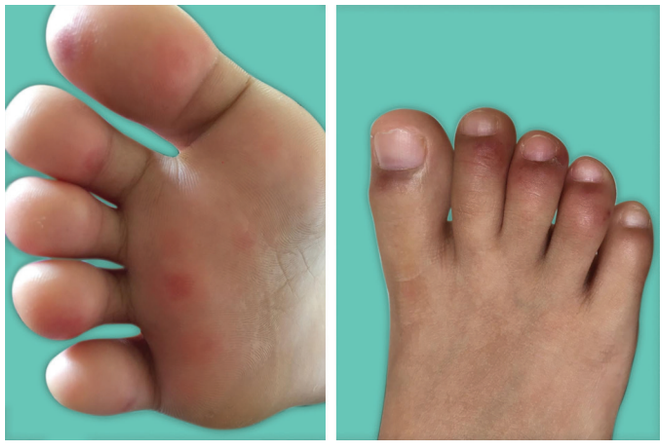


**Fig. 15.5:** Pseudo chil blains secondary to Covid infection

and they appear more sick as compared to children with KD[32,33] Feldstein, et al. observed 50% of MISC patients required vasopressor/inotropic support for cardiogenic shock as compared to 5% of children with KD in the US.[34] The underlying causes for the cardiac involvement are post-viral myocarditis; systemic hyper-inflammation, coronary involvement, endothelial dysfunction, and inflammatory vasculopathy.[32,33] Points of differentiation among mimickers of MIS-C are described in **Table 15.3**.

To establish the diagnosis, two-tier investigations have been suggested.[35] First-tier investigation include CBC, LFT including coagulation profile (D dimer and fibrinogen), KFT, CRP, ABG. If the first tier is positive then the second tier is also to be done and that includes trop T, BNP, procalcitonin, S. protein and albumin, amylase, C3, C4, peripheral smear. All blood culture, urine, and stool culture should also be done to rule out other differentials. Treatment includes IVIg (2 g/kg) in over 12 hours and methylprednisolone 30 mg/kg if the patient is in shock.[35] If the patient is not in shock, then methylprednisolone 1–2 mg/kg is to be started.

In case of D-dimer >5 times normal values and/or presence of other known prothrombotic factors, Enoxaparin 100 UI/kg BID should be administered. Aspirin is also to be started if coronary abnormalities are found on ECHO, as in Kawasaki disease.

## FEVER WITH PURPURIC RASH

Rickettsial infections, leptospirosis, meningococcal disease **(Fig. 15.6)**, HSP are the causes of palpable purpura while DIC causes nonpalpable purpura. Infective causes of purpuric rash are described in **Table 15.4**.

### Other Causes

Childhood vasculitis like HSP, SLE, and PAN can have varied manifestations; the initial presentation may be a rash along with nonspecific clinical features like fever (insidious onset), malaise, and weight loss. The most

**Fig. 15.6:** Purpura in meningococcemia

**Table 15.3:** MIS-C and its close differentials (fever with erythematous rash)

| | *TSS* | *KD* | *Severe dengue* | *MIS-C* |
|---|---|---|---|---|
| Etiology | Group A streptococci or staphylococcus | Vasculitis involving coronary vessels | Arbovirus, spread by *Aedes aegypti* | Hyperinflammatory reaction associated with SARS-CoV2 |
| Rash | Erythroderma | Maculopapular rash on day 5 of illness with erythema | Morbilliform rash involving extremities, sparing palms and soles | KD-like rash |
| Strawberry tongue | No | Yes | No | Yes |
| Desquamation | yes | Yes | No | Yes |
| Age group | Adolescents and adults | <5 yrs usually 1–2 yrs | 3 yrs and above | 7–11years |
| Shock | More common | Less common (5%) | Less common (5%) | More common |
| Cardiac involvement | + | + | + | ++ |
| GI manifestation | + | – | ++ | + |
| Coronary artery aneurysm | – | ++ | – | + |
| WBC count | Neutrophilia | Neutrophilia | Lymphopenia | Lymphopenia |
| Platelet count | Decreased | Increased or normal | Decreased | Decreased |

**Table 15.4:** Differentials of fever with purpuric rash in children and their characteristic features

| | *Route of transmission* | *season* | *Rash* | *Remarks and treatment* | *Complications* |
|---|---|---|---|---|---|
| Rickettsial disease | Bites (tick, mites,lice, or fleas) or inoculation of their infectious fluids | Spring and summer | The rash is macular or maculopapular, occasionally petechial. Typical painless eschar with an erythematous rim at the site of a vector bite. Palms and soles are affected last | Fever, headache, myalgia, hepatosplenomegaly, and lymphadenopathy Doxycycline (IV or oral @4mg/kg in two divided doses) is the drug of choice | DIC, ARDS, HLH, purpura fulminans, and myocarditis may occur |
| Leptospirosis | Direct or indirect contact with the urine of infected rats or dogs | Rainy season | Transient (<24 hours) rash in 10% of cases. The rash may be urticarial, petechial-purpurial or desquamating type | Biphasic pattern, high fever with chills, headache, conjunctival suffusion, hepatosplenomegaly (HSM), and generalized lymphadenopathy (LAD). Parenteral Penicillin G (6–8 MU/$m^2$/d divided every 4 hourly IV for 7 days. Doxycycline is alternative in children who are allergic to penicillin | Renal failure Respiratory failure The neurolepto-spirosis and disseminated intravascular coagulation (DIC) |
| Meningococcal | From colonized adult or patient with meningococcal disease | Winter | Palpable purpura involving trunk and extremities | Young child up to 3 yrs usually with features of meningitis. Third-generation cephalosporin-ceftriaxone or cefotaxime is recommended, with the addition of vancomycin in the regions with beta-lactam-resistant *S. pneumoniae* | Purpura fulminans associated with adrenal hemorrhage, hypotension, multiorgan failure (MOF) with or without meningitis |

common childhood vasculitis, Henoch schnolein purpura (HSP) affects children 2–8 year of age. It is characterized by palpable purpura along with arthritis/arthralgia, abdominal pain, and renal disease. The purpuric rash is typically seen on dependent areas like buttocks and lower extremities and uncommonly can be appreciated over arms, face, and ears **(Fig. 15.7)**. Subcutaneous edema of hands, feet, and scrotum are characteristic features. The diagnosis is mainly clinical but sometime biopsy of the skin or kidney demonstrating predominantly IgA deposition is required in case of incomplete or unusual presentation. Most children with HSP require only supportive management.[36]

Polyarteritis nodosa (PAN) is a systemic vasculitis affecting medium sized arterial vessels but may affect small vessels also. Skin involvement in the form of livedo reticularis, nodules, and superficial or deep skin infarctions can occur. Cutaneous PAN is characterized by fever and skin involvement without any systemic features (except for myalgia, arthralgia, and nonerosive arthritis). The condition usually remains localized to the skin although few of them may progress into systemic PAN.[37]

Children with SLE can present with various type of rash often causing a diagnostic dilemma. The hallmark though is the malar rash also called as butterfly rash involving the bridge of nose and cheeks sparing nasolabial folds. It is erythematous rash which is non pruritic.[38] Antimalarials, hydroxychloroquine, and chloroquine are used for the treatment.

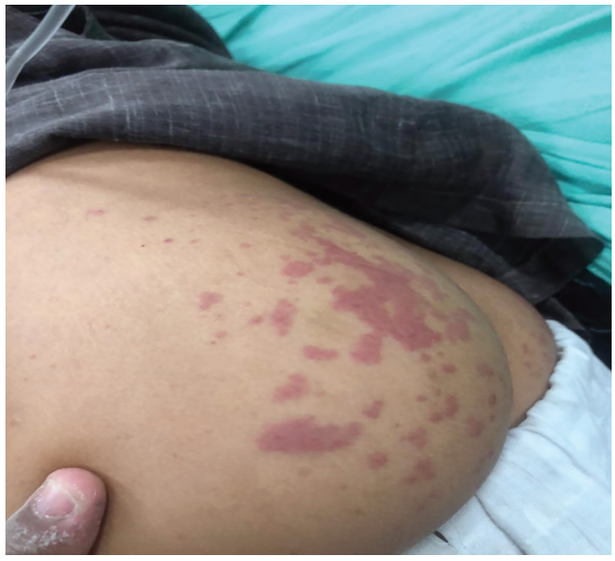


**Fig. 15.7:** Palpable purpura in HSP

Impetigo is another common cause of fever with rash in children. The rash is characterized by bullous or nonbullous lesions. Bullous lesions are commonly seen in neonates and nonbullous in older children. *S. aureus* and streptococcal pyogenes are commonly implicated organisms.[39] Uncomplicated impetigo can be managed with either topical mupirocin or fusidic acid however, systemic antibiotics are warranted for extensive or complicated impetigo.

Rose spots may be seen in about 25% of cases of enteric fever which may be difficult to see in Indian population due to their dark skin color. These are macular or maculopapular regions which appear in crops over chest and abdomen and disappear in 2–3 days.[40]

Erythema marginatum is seen in acute rheumatic fever which is flat or slightly raised evanescent rash seen on trunk and proximal extremities. It is one of the major diagnostic criteria of acute rheumatic fever and it resolves with mild hypopigmentation without any atrophy or scaling.[41]

## CONCLUSIONS

Exanthematous fever is common in children, and many of them are benign. Symptomatic management is only required in many of them especially in patients with maculopapular rash. The presence of red flag signs must be sought for, which indicate severe disease especially associated with erythematous or purpuric rash and these conditions need close monitoring and aggressive management. Thorough history and examination along with epidemiological knowledge help in making diagnosis and management.

### *Key Learning Points*

- Fever with rash is many a times challenge for pediatricians because there is an exhaustive list of causes.
- Red flag signs must be sought for in all children.
- A stepwise approach consisting of proper history and detailed physical examination to identify the systemic manifestation of the disease.
- It is important to identify conditions that require isolation to prevent the spread of contagious diseases.
- In children, viral exanthems are the most prevalent cause of fever with maculopapular rash.
- Presence of erythematous rash or purpuric rash usually indicates severe disease.
- Novel virus SARS-CoV2 is also implicated in various cutaneous manifestations in children.
- MIS-C is hyperinflammatory syndrome temporally associated with SARS-CoV2 which closely mimics Kawasaki disease, severe dengue, and TSS.

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