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Case Report of Vesiculobullous Impetigo

Ines Melania B. Ola[1], Novia Yudhitiara[2], Hari Darmawan[3]

[1]Medical Doctor Professional Program, Universitas Tarumanagara, Indonesia
[2]Department of Dermatology and Venereology, RSUD Ciawi, Indonesia
[3]Department of Dermatology and Venereology, Universitas Tarumanagara, Indonesia

ABSTRACT

Impetigo is a superficial bacterial skin infection commonly found in children, especially aged 2–5 years. This case report describes a 1 year 7 months old girl presenting with vesiculobullous lesions on the face and neck for one week, which ruptured and left yellowish crusts. The patient had previously received treatment from a primary health center without improvement. Clinical examination revealed multiple erythematous vesicles with crusting and well-defined borders on the face and neck. A diagnosis of vesiculobullous impetigo was established with differential diagnoses of contact dermatitis, staphylococcal scalded skin syndrome, and bullous pemphigoid. The patient was treated with gentamicin cream applied twice daily after bathing and oral amoxiclav syrup three times daily. Education on hygiene, infection prevention, and follow-up was also provided. Clinical improvement was evaluated after one week. Prognosis was good, both for life, function, and recovery. This case highlights the importance of accurate diagnosis and appropriate management in pediatric impetigo to prevent complications and transmission.

Keywords: Impetigo, vesiculobullous, pediatric, bacterial infection, case report

Correspondence:
Ines Melania B. Ola
Universitas Tarumanagara
Email address inesmelania31@gmail.com

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INTRODUCTION

Impetigo is a bacterial infection or inflammatory condition of the skin. The causative bacteria may include Staphylococcus aureus, group A β-hemolytic Streptococcus, or a combination of both, the latter also known as Streptococcus pyogenes. This infection affects the superficial layers of the skin with well-demarcated margins and most commonly occurs in children aged 2–5 years, although it may also occur in adults. Clinically, impetigo is classified into two forms, namely crusted impetigo and bullous impetigo (A, Djuanda M, Hamzah S, 2008).

Epidemiological evidence indicates that impetigo is a highly contagious skin infection that readily spreads within household settings, daycare centers, and schools. Transmission occurs through direct contact with lesions or infected skin. Poor hygienic conditions and crowded environments in tropical regions may contribute to the development of this disease (Guerra KC,

Toncar A, 2025). In the United States, approximately 9–10% of children presenting to clinics are diagnosed with impetigo, with a relatively equal incidence between males and females. The most common form is crusted impetigo, accounting for approximately 90% of cases, particularly in children under 2 years of age. Meanwhile, in the United Kingdom, the annual incidence of impetigo among children up to 4 years of age is 2.8%, and 1.6% among children aged 5–15 years, with approximately 70% of cases presenting as crusted impetigo (Guerra KC, Toncar A, 2025).

Several risk factors are known to contribute to the development of impetigo. First, childhood age, particularly between 2 and 5 years, during which the immune system is still developing and children frequently engage in close contact during play, thereby facilitating transmission. Second, poor skin hygiene, which increases the bacterial load on the skin surface and consequently elevates the likelihood of infection in the presence of cuts or abrasions. Third, densely populated living environments, which accelerate transmission either directly through physical contact or indirectly via contaminated objects (Bowen AC, Mahe A, Hay RJ, Andrews RM, Steer AC, 2015). Fourth, warm and humid climates that support the growth of Staphylococcus aureus and Streptococcus pyogenes, thereby increasing the risk of skin infection. Fifth, the presence of pre-existing skin injuries or conditions, such as insect bites or dermatitis, which facilitate bacterial entry into the skin (Koning S, van der Sande R, Verhagen AP, van Suijlekom-Smit LW, Morris AD, 2006).

Impetigo is classified into two types: primary and secondary. Primary impetigo occurs on normal skin due to direct bacterial invasion, while secondary impetigo occurs on areas of skin that have previously been injured or affected by other skin diseases (Stevens DL, 2016).

The pathophysiology of impetigo is characterized by the primary complaint of pruritus. The initial lesions typically present as erythematous macules measuring 1–2 mm, which subsequently develop into vesicles or bullae. These vesicles have thin walls and therefore rupture easily, releasing yellowish-brown seropurulent exudate that later dries and forms multilayered crusts. The crusts are easily removed, revealing an erosive area beneath that continues to exude fluid, leading to recurrent crust formation (A, Djuanda M, Hamzah S, 2008). In ruptured bullae, a collarette appearance is often observed at the lesion margins, with a varnish-like crust in the central area. When the crust is removed, the lesion base appears erythematous and moist. Intact bullae are rarely observed due to their fragile nature. Impetigo caused by Streptococcus typically occurs in body areas frequently exposed to the external environment, particularly the face and lower extremities. Lesions may be localized but are often multiple. Although regional lymphadenitis may occur, systemic symptoms are rarely present (Stevens DL, 2016).

Clinical manifestations of impetigo are superficial pyoderma limited to the epidermis and divided into three forms, namely crusted impetigo, bullous impetigo, and neonatal impetigo. Crusty impetigo is usually caused by β-haemolytic Streptococcus, with a predilection for the face, particularly around the nose and mouth. It is characterised by erythematous papules that develop into vesicles or pustules and rupture easily, leaving thick, honey-coloured crusts.

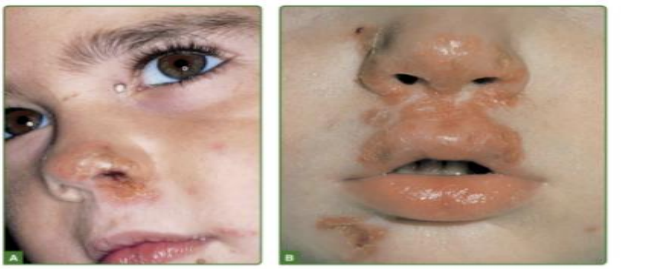

Figure 150-1 *Staphylococcus aureus:* impetigo. Erythema and honey-colored crusting on the nose and upper lip area (A), which can spread to involve the entire centrofacial region (B).

Figure 1. Crusted impetigo

Bullous impetigo is generally caused by Staphylococcus aureus and has a predilection for the axillae, chest, and back. It is characterized by erythema and thin-walled bullae containing clear fluid that subsequently becomes turbid; these bullae rupture, leaving erosions and collarettes, with a negative Nikolsky sign (A, Djuanda M, Hamzah S, 2008). This condition is caused by exfoliative toxins produced by the bacteria that cleave desmoglein-1 in the epidermis. When the toxin is locally distributed, bullous impetigo develops, whereas systemic dissemination may result in staphylococcal scalded skin syndrome. The neonatal variant of bullous impetigo is known as impetigo neonatorum (Kang S, Amagai M, Bruckner AL, Enk AH, Margolis DJ, 2019).

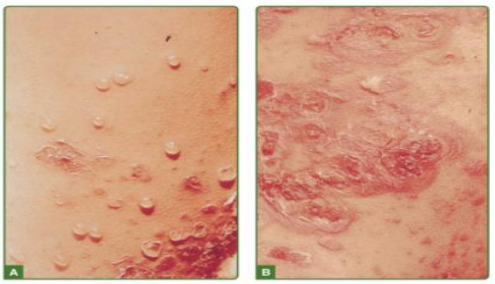

Figure 150-3 *Staphylococcus aureus*: bullous impetigo. Multiple vesicles with clear and turbid contents (**A**) that rapidly coalesce to form flaccid bullae (**B**).

Figure 2. Bullous impetigo

The diagnosis is generally established clinically through anamnesis, including a history of lesions presenting as papules or pustules that progress to easily ruptured vesicles or bullae with honey-colored crusts, accompanied by pruritus or pain, as well as a history of contact and the presence of risk factors. Physical examination reveals characteristic yellowish crusted lesions, vesicles, or bullae, particularly on the face, extremities, or areas of trauma. Additional investigations, such as bacterial culture, are indicated in recurrent, resistant, or nonresponsive cases. Most cases do not require further laboratory testing except in atypical conditions (Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJ, 2015).

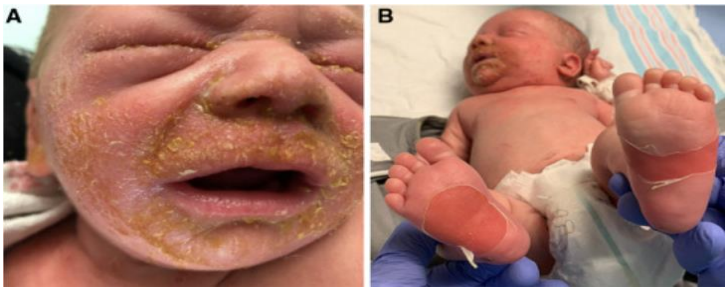

Figure 3. Neonatal impetigo

The differential diagnosis of nonbullous impetigo includes ecthyma, a deeper infection caused by Streptococcus pyogenes or Staphylococcus aureus characterized by thick brownish crusted ulcers that may result in scarring; atopic dermatitis, associated with a history of atopy, presenting with erythematous scaly plaques or lichenification, intense pruritus, age-specific distribution, and absence of the characteristic honey-colored crusts; seborrheic dermatitis, manifested as greasy erythematous scaly plaques in sebaceous-rich areas, commonly occurring in infants and known as cradle cap; allergic contact dermatitis resulting from exposure to specific allergens, characterized by erythema, edema, and vesicles confined to the area of contact; scabies caused by Sarcoptes scabiei, presenting with nocturnal pruritus and papules, vesicles,

and burrows in intertriginous areas, often affecting household members; and tinea capitis, characterized by alopecia with scaling, broken hairs, or kerion, confirmed by potassium hydroxide (KOH) examination or fungal culture (James WD, Elston DM, Treat JR, Rosenbach MA, 2020). Differential diagnosis of bullous impetigo includes contact dermatitis with a clear history of exposure and a characteristic distribution; staphylococcal scalded skin syndrome (SSSS), characterized by widespread desquamation, fragile bullae, systemic symptoms, sterile skin cultures, and a positive Nikolsky sign; bullous pemphigoid, an autoimmune disease in the elderly presenting with chronic tense subepidermal bullae that do not respond to antibiotic therapy; and pemphigus vulgaris, an autoimmune disorder characterized by flaccid intraepidermal bullae, painful erosions, frequent mucosal involvement, and a positive Nikolsky sign without honey-colored crusts (Stevens DL, 2020a).

Management is divided into non-pharmacological and pharmacological approaches. Non-pharmacological management includes bathing twice daily with soap; identification and treatment of predisposing factors or comorbidities such as parasitic infestations, atopic dermatitis, edema, obesity, or venous insufficiency; and incision and drainage of large, painful, or fluctuant abscesses. In principle, patients are managed on an outpatient basis, except in cases of severe erysipelas, cellulitis, or phlegmon that require hospitalization (Stevens DL, 2020b). Topical therapy includes open compresses with potassium permanganate 1:5000, salicylic acid 0.1%, rivanol 1‰, or povidone–iodine 1% for crusted lesions, applied three times daily for 30–60 minutes during the acute phase; whereas for lesions without pus or crusts, 2% fusidic acid ointment or cream or 2% mupirocin is applied two to three times daily for 7–10 days (Perhimpunan Dokter Spesialis Kulit dan Kelamin Indonesia, 2022).

Prevention can be achieved by maintaining personal hygiene through regular bathing, washing hands with soap, cutting nails, temporarily isolating patients until the wound has dried or at least 24 hours after antibiotic therapy, not sharing personal items, cleaning and covering small wounds with gauze, and treating underlying skin conditions such as dermatitis (World Health Organization, 2023).

Potential complications include cellulitis, lymphadenitis, bacteremia with a risk of sepsis, particularly in immunocompromised patients; post-streptococcal glomerulonephritis occurring 1–3 weeks after infection; staphylococcal scalded skin syndrome (SSSS) in severe cases due to exfoliative toxins of *Staphylococcus aureus*; and cicatricial scarring, especially in ecthyma or deeper infections (James WD, Elston DM, Treat JR, Rosenbach MA, 2022) The prognosis of impetigo is generally favorable, with spontaneous resolution possible within two weeks without sequelae; however, ecthyma may persist for several weeks with a risk of scarring, and in children the recurrence rate of abscesses or furuncles reaches 18–28% (Perhimpunan Dokter Spesialis Kulit dan Kelamin Indonesia, 2022).

METHOD

This study employed a case report design aimed at describing the clinical manifestations, diagnostic process, management, and outcomes of a pediatric patient with vesiculobullous impetigo. The study subject was a 1-year-7-month-old female child who received care at RSUD Ciawi (Ciawi Regional General Hospital) in August 2025. Data were collected through structured anamnesis, comprehensive physical examination, and review of the patient's medical records, including demographic characteristics, history of present illness and past medical history, family history, immunization status, growth and developmental history, nutritional status, daily habits, clinical findings, working and differential diagnoses, and therapeutic management.

The study was conducted at Ciawi Regional General Hospital, a secondary referral health care facility. Data analysis was performed using a qualitative descriptive approach, with clinical data presented in a systematic narrative manner and contextualized with relevant literature and

clinical guidelines. Ethical considerations were addressed by maintaining patient confidentiality through the use of initials and ensuring that all data were obtained from routine clinical examinations and medical records. Institutional permission was obtained, and written informed consent for scientific publication was secured from the patient's parents.

RESULT

A female pediatric patient aged 1 year and 7 months, identified as An. JA, residing in KP. Bojong, was admitted to Ciawi Regional General Hospital on August 5, 2025, at 12:30 PM, presenting with fluid-filled blisters on the face and neck that had ruptured and resulted in yellowish crust formation for one week prior to presentation. The condition initially manifested as small lesions on the chin, which progressively enlarged, contained yellowish turbid fluid, ruptured, and subsequently spread to the perioral area, cheeks, nose, and neck. The lesions were pruritic, leading to increased irritability and frequent scratching. There were no associated symptoms such as fever, vomiting, cough, or rhinorrhea.

Figure 4. Case Report

The past medical history revealed that the patient had never experienced similar complaints previously. A history of allergy to foods, medications, or dust was denied. The family medical history was unremarkable, with no family members reporting similar conditions. The patient was born at term via cesarean section in a hospital, with a birth weight of 2,600 g and a birth length of 49 cm. She is the second of two siblings and was delivered with a spontaneous cry immediately after birth, with no congenital abnormalities noted.

Patient had received complete basic immunizations according to the schedule at the primary health care center, including BCG, polio, DPT, hepatitis B, and DTP–HB–Hib vaccines. The patient had previously sought treatment at a primary health care facility and was prescribed powdered medication, gentamicin ointment, triamcinolone ointment, and a compounded topical preparation; however, no clinical improvement was observed. The child's growth history was appropriate for age, as indicated by continuous increases in body weight and height. Development was also age-appropriate, including gross motor skills such as running and pushing toys, fine motor skills such as holding a spoon and turning book pages, language development with the use of 10–20 words, and social skills demonstrated by imitation of adult activities.

Nutritional history indicated that the patient received exclusive breastfeeding since birth, which was later supplemented with UHT Greenfield formula milk. The dietary pattern followed the family menu, with three main meals per day accompanied by fruit and snacks. The patient also routinely consumed adequate amounts of fluids. The daily habit history included bathing twice daily using bar soap and Cussons shampoo, followed by the application of telon oil. Powder was rarely used due to the patient's sensitive skin.

On physical examination, the patient appeared moderately ill with compos mentis consciousness (GCS 15: E4M6V5). Pulse rate was 90 beats per minute, regular with adequate volume, respiratory rate was 20 breaths per minute, temperature was 36.7°C, weight was 8.5 kg, and height was 75 cm.

DISCUSSION

A female pediatric patient aged 1 year and 7 months presented with a one-week history of ruptured fluid-filled blisters on the face and neck, resulting in yellowish crust formation on the cheeks. The lesions initially appeared as small lesions on the chin and subsequently enlarged, contained yellowish turbid fluid, ruptured, and spread to the perioral region, cheeks, nose, and neck. The condition was accompanied by pruritus, leading to increased irritability and frequent scratching. A history of treatment at a primary health care center was noted; however, no clinical improvement was observed.

Cutaneous examination revealed regionally distributed lesions on the face and neck, with multiple lesions of irregular shape and nummular size, well-demarcated borders, and a moist appearance. The primary efflorescences consisted of erythematous vesicles, while the secondary efflorescences were crusts. Based on these findings, a working diagnosis of vesiculobullous impetigo was established, with differential diagnoses including contact dermatitis, staphylococcal scalded skin syndrome, and bullous pemphigoid.

Patient management included pharmacological therapy consisting of topical gentamicin cream applied twice daily to the affected areas after bathing and oral amoxicillin–clavulanate syrup at a dose of 4 mL administered three times daily until completion. Patient education included recommendations to maintain personal hygiene, avoid contact with others to prevent transmission, refrain from touching or scratching lesions in the infected areas, clean frequently touched surfaces such as door handles, tables, or toys, use medications as prescribed by the physician, and attend follow-up evaluation after one week. Evaluation was conducted to assess both clinical improvement and the patient's complaints. The patient's prognosis was considered favorable, including ad vitam bonam, ad functionam bonam, and ad sanationam bonam.

CONCLUSION

This case demonstrates that vesiculobullous impetigo in children can be diagnosed based on its characteristic clinical presentation, namely vesicles and bullae that easily rupture and leave yellowish crusts. Appropriate management with topical and systemic antibiotics, accompanied by education on personal hygiene and transmission prevention, results in a favorable prognosis. It is expected that health care providers will be able to recognize the clinical manifestations of impetigo at an early stage, provide guideline-based therapy, and deliver comprehensive education to parents in order to reduce the risk of complications and prevent the spread of infection.

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