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Clinical Spectrum and Outcomes of Pediatric Mpox during the Sindh Outbreak: A Four-Case Series from Sukkur, Sindh, Pakistan

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Abstract

Background: *Mpox is a viral zoonotic disease caused by monkeypox virus (MPXV), with continuing international transmission and evolving epidemiology. Although pediatric cases are less frequently reported than adult cases, neonates and young infants may be at increased risk of severe disease. Published information regarding pediatric mpox from Pakistan remains limited.*

Case Presentation: *We describe four PCR-confirmed pediatric mpox cases managed at a tertiary pediatric referral hospital in Sukkur during the 2026 Sindh mpox outbreak. Case 1 was a 9-year-old boy with an atypical purpuric eruption predominantly involving the lower extremities, accompanied by abdominal pain and vomiting, clinically mimicking IgA vasculitis. Case 2 was a 21-day-old neonate with disseminated vesiculopustular lesions, oral involvement, seizures, poor feeding, and respiratory distress who died following progressive clinical deterioration. Case 3 was a 2-month-old infant with extensive disseminated and partly necrotic cutaneous lesions, oral involvement, feeding difficulty, and severe respiratory distress who also died. Case 4 was a 35-day-old infant with generalized vesiculopustular lesions and a reported household contact with confirmed mpox who recovered with supportive management. MPXV infection was confirmed by PCR from cutaneous lesion specimens in all four patients. Patient-level viral clade determination was not performed.*

Conclusion: *Pediatric mpox may present across a broad clinical spectrum, ranging from atypical localized purpuric eruptions to severe disseminated disease in neonates and young infants. The two fatal cases highlight the potential vulnerability of young infants, although the precise mechanisms of death could not be definitively established. Early recognition, laboratory confirmation, isolation, supportive care, and rigorous infection-prevention measures are essential during outbreaks.*

Keywords: *Mpox, Monkeypox Virus, Pediatric Infection, Neonatal Mpox, Purpuric Eruption, Sindh, Pakistan, Case Series*

Introduction

Mpox is an infectious zoonotic disease caused by monkeypox virus (MPXV), an enveloped double-stranded DNA virus belonging to the genus Orthopoxvirus of the family Poxviridae. [1,2] Human mpox was first recognized in the Democratic Republic of the Congo in 1970 and historically occurred predominantly in Central and West Africa. [2,3]

The first recognized outbreak outside Africa occurred in the United States in 2003. A large multinational outbreak subsequently emerged in 2022, with sustained person-to-person transmission and clinical presentations that frequently differed from historically recognized disease. [1,4]

The World Health Organization (WHO) declared the multinational outbreak a Public Health Emergency of International Concern in July 2022.[5]

Human-to-human transmission occurs predominantly through close contact with infectious skin or mucosal lesions, respiratory secretions during close contact, or contaminated materials. Maternal-fetal and perinatal transmission can also occur. [1,6] Classical disease may include fever, lymphadenopathy, systemic symptoms, and vesicular or pustular lesions progressing through stages before crusting and healing. [1,3] However, the clinical phenotype varies according to host characteristics, viral clade, route of exposure, and epidemiologic setting.

Children, particularly neonates and young infants, represent an important population at risk of complications. Severe manifestations may include

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extensive mucocutaneous disease, dehydration, secondary bacterial infection, respiratory compromise, neurological involvement, and systemic illness. [1,7] Maternal-fetal transmission has also been documented, including placental infection and congenital mpox.[8]

Pakistan reported its first laboratory-confirmed mpox case in April 2023. The epidemiologic situation changed substantially during 2026 when locally acquired cases and clusters were detected in Sindh Province.[9]

During the 2026 Sindh outbreak, a healthcare-associated cluster involving neonates, infants, and adults was identified in Khairpur district. According to the WHO External Situation Report of April 30, 2026, 249 suspected cases were reported across nine districts of Sindh between March 14 and April 20, 2026, of which 29 were laboratory confirmed. The outbreak in Khairpur disproportionately affected young infants and was associated with substantial mortality. Genomic sequencing undertaken during the broader outbreak investigation identified clade Ib MPXV.[9]

The four children described in this report were managed at the Sindh Institute of Child Health and Neonatology (SICHN), Children Hospital Sukkur, a tertiary pediatric referral center serving Sukkur and surrounding districts. Although they were identified during the period of increased regional mpox activity, the four patients were not demonstrated to constitute a single epidemiologically linked cluster. A household exposure was documented in Case 4, whereas no definite epidemiologic linkage among the remaining patients could be established from the available histories.

We therefore report these patients as a clinical case series illustrating the spectrum and outcomes of PCR-confirmed pediatric mpox encountered at a tertiary referral center during the 2026 Sindh outbreak rather than as a defined transmission cluster or representative epidemiologic sample of the entire provincial outbreak.

Laboratory Confirmation

For all four patients, MPXV infection was confirmed by polymerase chain reaction (PCR) performed on specimens obtained directly from active cutaneous lesions. Lesion specimens were collected using sterile swabs from representative vesicular, pustular, umbilicated, or crusted lesions and submitted for molecular testing according to the prevailing outbreak diagnostic protocol.

Detailed information regarding the specific PCR platform, molecular target, and assay manufacturer was not available in the clinical records available for this case series and is therefore not reported. All four results were reported as positive for MPXV.

Patient-level genomic sequencing and viral clade determination were not performed. Therefore, although genomic investigation of the broader 2026 Sindh outbreak identified clade Ib MPXV,[9] clade Ib cannot be definitively assigned to the individual patients described in this series.

Case Series

Case 1

A 9-year-old boy from Ghotki district presented with a 10-day history of abdominal pain and vomiting followed by development of a rash predominantly involving the ankles and lower extremities. There was no reported history of fever, pruritus, mucosal involvement, lymphadenopathy, travel, or known animal exposure.

Examination revealed multiple purpuric-to-violaceous papular and plaque-like lesions predominantly over the ankles and lower extremities (Figure 1). The lesions were painful but non-pruritic. The combination of lower-limb purpura and gastrointestinal symptoms initially raised clinical suspicion for IgA vasculitis (Henoch–Schönlein purpura). Other differential diagnoses included meningococcemia and hematologic causes of purpura.

Laboratory investigations demonstrated hemoglobin 11.3 g/dL, total leukocyte count 15.3 $\times 10^3/\mu L$, neutrophils 60%, lymphocytes 25%, and platelet count 599 $\times 10^3/\mu L$. Total bilirubin was 0.2 mg/dL, alanine aminotransferase 36 U/L, creatinine 0.5 mg/dL, and urea 25 mg/dL. Urinalysis was unremarkable. Malaria screening, HIV testing, and hepatitis screening were negative.

In view of ongoing regional mpox activity, a specimen obtained directly from a cutaneous lesion was submitted for MPXV PCR and tested positive.

Although IgA vasculitis was considered clinically, skin biopsy and histopathologic examination were not performed. Serum IgA, complement levels, and detailed coagulation studies were not available. Consequently, IgA vasculitis could not be definitively excluded, and histologically confirmed vasculitis cannot be attributed to mpox. We therefore describe the presentation as an atypical purpuric eruption mimicking IgA vasculitis rather than mpox-associated vasculitis.

The child remained clinically stable and received symptomatic supportive management under isolation precautions. His abdominal pain and vomiting resolved gradually and the skin lesions improved. The prolonged hospitalization of approximately three to four weeks occurred in the context of outbreak-related isolation and infection-control requirements while cutaneous lesions were resolving rather than because of progressive systemic illness. He was subsequently discharged in stable condition.

Case 2

A 21-day-old female neonate born at term by cesarean section presented with poor feeding, lethargy, respiratory distress, seizures, and generalized vesiculopustular lesions involving the skin and oral mucosa. She had initially been hospitalized at another healthcare facility for approximately four days before referral.

On examination, she was critically ill, with extensive disseminated umbilicated vesiculopustular lesions

involving the face, trunk, extremities, and oral cavity (Figure 2). Respiratory distress and neurological manifestations were evident.

Laboratory investigations demonstrated hemoglobin 16.1 g/dL, total leukocyte count 32.5 $\times10^3$/µL, platelet count 66 $\times10^3$/µL, neutrophils 4.6%, and lymphocytes 79.7%. Serum sodium was 135 mEq/L, potassium 6.4 mEq/L, chloride 95 mEq/L, creatinine 0.23 mg/dL, blood urea nitrogen 25.4 mg/dL, calcium 9.67 mg/dL, and magnesium 2.53 mg/dL.

PCR testing of a cutaneous lesion specimen confirmed MPXV infection. HIV testing was negative.

The precise route of transmission could not be determined. Because disease developed during the neonatal period, congenital, perinatal, and early postnatal transmission were considered. However, detailed maternal MPXV testing, placental examination, and neonatal virologic testing immediately after birth were unavailable. Furthermore, the child had received inpatient care at another healthcare facility before referral; therefore, healthcare-associated exposure could not be excluded.

The neonate was managed in ICU isolation with supportive and respiratory care. Empirical meropenem was administered because of concern for concomitant severe bacterial infection. Intravenous acyclovir was administered initially while alternative viral etiologies were being considered; it was not used as mpox-specific therapy.

Despite intensive supportive management, the neonate progressively deteriorated and died. Although severe respiratory compromise and systemic deterioration were clinically evident, comprehensive microbiologic, physiologic, and postmortem investigations were unavailable. Therefore, the precise immediate and underlying causes of death could not be definitively established.

Case 3

A 2-month-old male infant from Sukkur presented with fever, severe respiratory distress, feeding difficulty, oral involvement, and progressive generalized skin lesions. He had initially received treatment at another healthcare facility before referral.

Physical examination demonstrated extensive disseminated umbilicated papulovesicular and pustular lesions involving the face, scalp, trunk, and extremities. Multiple lesions demonstrated crusting and necrotic transformation, with confluent facial involvement (Figure 3).

Laboratory investigations showed hemoglobin 18.0 g/dL, platelet count 73 $\times10^3$/µL, total leukocyte count 19.0 $\times10^3$/µL, neutrophils 21.4%, and lymphocytes 77.4%. Total bilirubin was 0.3 mg/dL, direct bilirubin 0.3 mg/dL, alanine aminotransferase 12 U/L, alkaline phosphatase 221 U/L, urea 22.6 mg/dL, and creatinine 0.24 mg/dL.

PCR from a cutaneous lesion specimen confirmed MPXV infection. HIV testing was negative.

No definite household or maternal epidemiologic exposure could be established from the available history. Because the infant had been hospitalized at another healthcare facility before referral, healthcare-associated acquisition was considered possible but could not be demonstrated.

The patient was managed under isolation precautions with oxygen supplementation, intravenous fluids, nutritional and other supportive care. Meropenem and vancomycin were administered empirically because of concern for concomitant or secondary bacterial infection. Mpox-specific antiviral therapy was not available.

The infant progressively deteriorated and died approximately five days after admission. The available clinical records did not permit definitive determination of the immediate and underlying causes of death. In particular, comprehensive microbiologic investigations and postmortem examination were unavailable; therefore, death cannot be attributed exclusively to MPXV infection.

Case 4

A 35-day-old male infant presented with a 15-day history of generalized skin lesions associated with fever, poor feeding, and lethargy. He had initially been hospitalized at another healthcare facility before referral.

A household epidemiologic exposure was identified: a sibling was reported to have laboratory-confirmed mpox diagnosed outside our institution.

On examination, the infant was clinically stable without respiratory distress or neurological manifestations. Dermatologic examination demonstrated multiple discrete, relatively monomorphic umbilicated vesiculopustular lesions involving the face, lower extremities, and plantar surfaces, with several lesions showing central crusting (Figure 4).

Laboratory investigations demonstrated hemoglobin 8.7 g/dL, total leukocyte count 12.0 $\times10^3$/µL, neutrophils 50%, lymphocytes 45%, and platelet count 162 $\times10^3$/µL. Blood urea nitrogen was 9.5 mg/dL, creatinine 0.25 mg/dL, sodium 133 mEq/L, potassium 5.6 mEq/L, chloride 95 mEq/L, and bicarbonate 19.0 mEq/L.

PCR testing of a cutaneous lesion specimen confirmed MPXV infection.

The patient was managed under isolation precautions with supportive care. He remained hemodynamically stable and demonstrated gradual clinical improvement. He was subsequently discharged in stable condition.

Discussion

This case series demonstrates the heterogeneous clinical spectrum of PCR-confirmed pediatric mpox encountered during the 2026 Sindh outbreak, ranging from an unusual purpuric eruption in an older child to extensive

disseminated disease associated with fatal outcomes in neonates and young infants.

The epidemiologic context is important. The 2026 Sindh outbreak included a healthcare-associated cluster in Khairpur that disproportionately affected neonates and young infants. WHO reported 249 suspected cases across nine districts between March 14 and April 20, 2026, including 29 laboratory-confirmed infections. Genomic sequencing performed during the broader outbreak investigation identified clade Ib MPXV.[9]

The four cases in our series should not, however, be interpreted as a single epidemiologically linked cluster. They were managed at our tertiary referral center following presentation or referral from different locations. A documented household exposure was identified only in Case 4. In the absence of comprehensive contact tracing and patient-level genomic sequencing, epidemiologic relationships among the remaining cases cannot be established.

Case 1 was particularly noteworthy because the cutaneous eruption was predominantly purpuric, involving the ankles and lower extremities and accompanied by abdominal pain and vomiting. This combination clinically mimicked IgA vasculitis. However, the positive MPXV PCR obtained from a cutaneous lesion establishes MPXV infection but does not demonstrate a vasculitic mechanism.

Because histopathology, serum IgA, complement testing, and other investigations required to characterize vasculitis were unavailable, we deliberately avoid describing the case as mpox-associated vasculitis. Instead, we consider it an atypical purpuric eruption mimicking IgA vasculitis in a child with PCR-confirmed mpox. This distinction is important because mpox can demonstrate variable dermatologic manifestations and can mimic other infectious and inflammatory dermatoses. [4,10]

Cases 2 and 3 demonstrate the potential severity of disease among neonates and young infants. Both had extensive disseminated cutaneous disease, mucosal involvement, feeding difficulty, respiratory compromise, thrombocytopenia, and progressive deterioration. Historically, younger children have been recognized among populations at risk for severe mpox. [3,7]

Oral and extensive mucocutaneous involvement may further complicate illness in young infants through impaired feeding, dehydration, nutritional deterioration, disruption of the cutaneous barrier, and potential secondary bacterial infection.

Both fatal cases demonstrated thrombocytopenia, whereas the clinically stable older child had thrombocytosis. Case 2 also had marked leukocytosis. However, the very small number of patients prevents interpretation of these abnormalities as predictors of severity. They are therefore presented descriptively.

The causes of death in Cases 2 and 3 warrant particular caution. Both infants had PCR-confirmed mpox with extensive disease and clinical deterioration, but comprehensive microbiologic, physiologic, and postmortem investigations were unavailable. Consequently, it is not possible to distinguish with certainty among direct severe viral disease, respiratory complications, secondary bacterial infection, circulatory failure, or a combination of these processes. We therefore avoid attributing either death exclusively to mpox.

Case 4 provides an important contrast. Despite his young age, the infant remained clinically stable and recovered with supportive management. The documented infection in a sibling provides the strongest epidemiologic link among the four cases and is compatible with household transmission, a recognized route of MPXV spread.[1]

The transmission routes in Cases 2 and 3 remain uncertain. Congenital and perinatal transmission are biologically and clinically relevant considerations in neonatal disease. MPXV has been demonstrated in placental and fetal tissues, establishing that transplacental transmission can occur.[8] However, maternal testing, placental examination, and virologic testing at birth were unavailable in Case 2. Consequently, congenital, perinatal, and early postnatal transmission cannot be distinguished.

Healthcare-associated acquisition must also be considered because Cases 2–4 received care at other healthcare facilities before referral. This possibility is particularly relevant in light of the documented healthcare-associated component of the 2026 Sindh outbreak.[9] Nevertheless, no direct transmission link was established for our patients, and healthcare-associated acquisition therefore remains a possibility rather than a confirmed route.

Patient-level viral clade determination was not performed. Although genomic sequencing from the broader Sindh outbreak identified clade Ib MPXV,[9] these outbreak-level findings cannot be directly extrapolated to the individual patients in this series.

All four patients had PCR confirmation from cutaneous lesion specimens, strengthening diagnostic certainty. HIV testing was negative in Cases 1–3. Severe disease in the two young infants therefore occurred without documented HIV infection. Pediatric HIV data from the same regional referral setting have previously demonstrated that HIV infection is concentrated among selected high-risk pediatric populations rather than representing a universal explanation for severe infectious presentations [11]. Furthermore, our institution has previously documented a substantial burden and high mortality associated with other vaccine-preventable and re-emerging pediatric infections, including diphtheria.[12] Together, these observations highlight the continuing challenges posed by emerging and re-emerging infectious diseases among children in this resource-limited region of Pakistan.

Management was primarily supportive because mpox-specific antiviral therapy was unavailable. Empirical antibacterial agents were used in critically ill infants when concomitant or secondary bacterial infection was clinically suspected. Acyclovir given in Case 2 was empirical treatment while other viral etiologies were being considered and should not be interpreted as MPXV-directed therapy.

The healthcare-associated component of the Sindh outbreak emphasizes the importance of infection-prevention and control practices in neonatal and pediatric settings. Prompt identification and isolation of suspected cases, appropriate personal protective equipment, hand hygiene, environmental decontamination, safe handling of linen and clinical equipment, and systematic contact tracing are important measures for limiting transmission. [1,9]

Limitations

This study has several limitations.

- First, the small number of patients inherent to the case-series design limits generalizability, and these four cases should not be considered representative of the complete clinical or epidemiologic spectrum of pediatric mpox in Sindh.
- Second, comprehensive contact-tracing data were unavailable for all patients. Except for the reported household exposure in Case 4, epidemiologic linkage among the four cases could not be established.
- Third, detailed maternal, perinatal, household, and healthcare exposure histories were incomplete, particularly for the neonatal and infantile cases. Consequently, congenital, perinatal, household, community, and healthcare-associated transmission could not always be distinguished.
- Fourth, patient-level genomic sequencing and viral clade determination were not performed. Although clade Ib MPXV was identified during genomic investigation of the broader Sindh outbreak, the viral clade of the individual cases in this series cannot be definitively assigned, nor can genomic relatedness among these cases be assessed.[9]
- Fifth, detailed information regarding the PCR platform, assay manufacturer, and molecular target was unavailable from the clinical records available for this report. Although all four lesion specimens were reported as PCR-positive for MPXV, this limits the completeness of our description of the molecular methodology.
- Sixth, skin biopsy and histopathologic examination were not performed in Case 1, and serum IgA, complement levels, and detailed coagulation studies were unavailable. Consequently, the purpuric eruption cannot be classified as histologically confirmed vasculitis, and IgA vasculitis cannot be definitively excluded.
- Seventh, complete serial physiologic data, inflammatory markers, imaging, microbiologic investigations, and detailed ICU parameters were not consistently available for the two fatal cases. Postmortem examinations were also not performed. The immediate and underlying causes of death therefore could not be definitively established.
- Finally, systematic long-term follow-up was unavailable for the surviving patients.
- Despite these limitations, this case series provides clinically relevant information regarding PCR-confirmed pediatric mpox during the 2026 Sindh outbreak and highlights both atypical presentations and severe disseminated disease among neonates and young infants.

Conclusion

- Pediatric mpox may present with a broad spectrum of manifestations ranging from relatively mild or atypical localized disease to severe disseminated infection in neonates and young infants.
- An atypical purpuric eruption accompanied by abdominal symptoms may clinically mimic IgA vasculitis. However, in the absence of histopathologic evidence, such lesions should not be characterized as mpox-associated vasculitis solely on the basis of positive lesion PCR.
- The fatal outcomes observed in two young infants demonstrate the potential severity of pediatric mpox, although the precise mechanisms of death could not be definitively established. The healthcare-associated component of the broader 2026 Sindh outbreak further emphasizes the importance of rigorous infection-prevention practices in neonatal and pediatric healthcare facilities.
- Early recognition, molecular confirmation, prompt isolation, supportive management, evaluation for complications and secondary infections, contact tracing, and strengthened infection-prevention measures are essential during outbreaks. Further studies incorporating detailed epidemiologic investigation, patient-level genomic sequencing, comprehensive severity assessment, and longitudinal follow-up are needed to better define transmission patterns and determinants of severe pediatric disease.
- Figure Legends

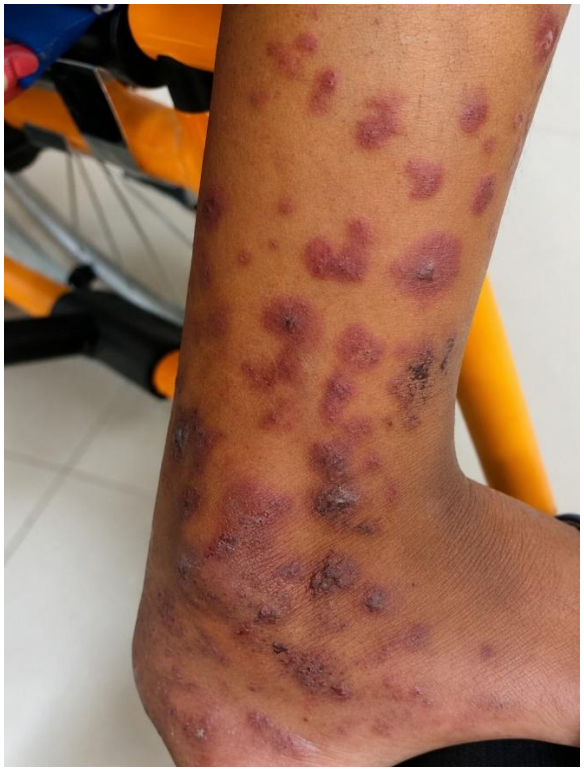

Figure 1 Atypical purpuric presentation in a 9-year-old boy with PCR-confirmed mpox, showing purpuric-to-violaceous papular and plaque-like lesions predominantly involving the lower extremity and clinically mimicking IgA vasculitis

Figure 2 Disseminated mpox in a 21-day-old neonate showing extensive vesiculopustular and umbilicated lesions involving the face, trunk, extremities, and oral mucosa

Figure 3 Severe disseminated mpox in a 2-month-old infant showing extensive papulovesicular and pustular lesions with crusting, necrotic transformation, and confluent facial involvement

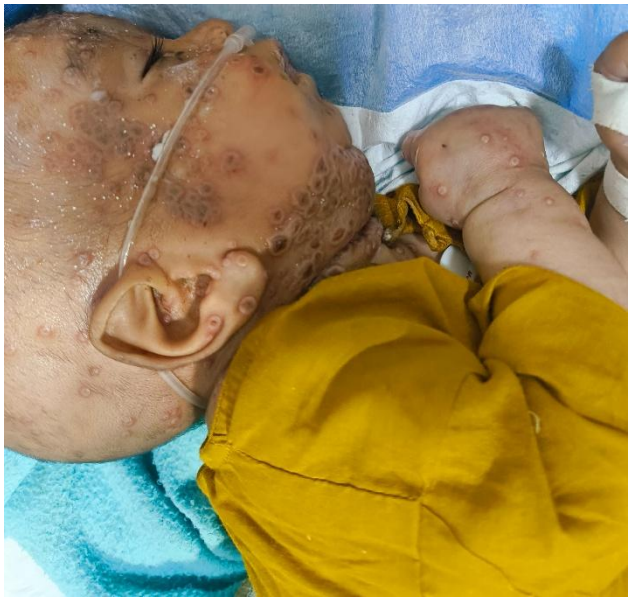

Figure 4 Mpox in a 35-day-old infant showing discrete umbilicated vesiculopustular lesions with central crusting involving the face, lower extremities, and plantar surfaces

Table 1: Clinical Characteristics of Pediatric Mpox Cases

Variable	Case 1	Case 2	Case 3	Case 4
Age	9 years	21 days	2 months	35
Gender	Male	Female	Male	Male
Residence	Ghotki	Shikarpur	Sukkur	Sukkur
Fever	Absent	Present	Present	Present
Rash Pattern	Purpuric vasculitic-like	Disseminated vesiculopustular	Disseminated necrotic papulovesicular	Umbilicated vesiculopustular
Oral Involvement	No	Yes	Yes	No
Respiratory Distress	No	Yes	Yes	No
Neurological Manifestations	No	Seizures	No	No
HIV Status	Negative	Negative	Negative	Negative
PCR for Mpox	Positive	Positive	Positive	Positive
ICU Admission	No	Yes	Yes	No
Outcome	Recovered	Expired	Expired	Recovered

Funding Declaration

No Funding

Ethics approval and consent to participate

Ethical approval was waived by the Ethics Review Committee of Sindh Institute of Child Health and Neonatology as this study was a retrospective case series using anonymized clinical data. The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from the parents/legal guardians of all pediatric patients included in the study

Consent for publication

Written informed consent for publication of anonymized clinical details and images was obtained from the parents/legal guardians of all patients included in this case series.

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