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CASE REPORT

PATHOLOGY/BIOLOGY

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Disseminated Neonatal Herpetic Infection Simulating Abusive Anal Trauma

ABSTRACT: Potential simulators of premortem trauma present problems of misinterpretation and possible false accusations of caregivers. A case of unsuspected neonatal herpes is reported with associated perianal ecchymosis that raises the possibility of sexual abuse. The decedent was an 8-day-old newborn infant who was born by Cesarean section and treated for 5 days postdelivery for sepsis. The newborn infant was discharged home but returned 2 days later with probable sepsis and new onset of perianal hemorrhage. She died 1 day later with autopsy, revealing neonatal disseminated herpetic infection with early anal involvement consisting of microscopic ulcerations with leukocytoclastic-like vasculitis and rare viral cytopathic changes. These histological changes produced grossly appearing anal ecchymosis with an absence of typical herpetic vesiculopapular lesions, which simulated abusive trauma. This case highlights the importance of considering occult neonatal herpes with associated perianal ecchymosis when presented with possible abusive anal trauma in a newborn infant.

KEYWORDS: forensic science, disseminated herpetic infection, simulating, abusive anal trauma, newborn infant

Heightened vigilance for child abuse is necessary to detect child abuse and to prevent future abusive injuries. However, awareness of potential simulators of premortem trauma is necessary to avoid unnecessary accusations of innocent caregivers. A case of unsuspected disseminated neonatal herpetic infection is presented in which early anal involvement simulated abusive anal trauma. The anal external findings consisted of ecchymosis with slight rectal prolapse and mucopurulent discharge. Internal examination at autopsy demonstrated herpetic hepatitis, pneumonitis, adrenalitis, and encephalitis. Histology of the anus ecchymosis revealed patchy foci of early microscopic herpetic ulcerations with underlying leukocytoclastic-like vasculitis and rare herpetic viral nuclear cytopathic changes. Given the clinically unsuspected nature of the disseminated herpes, the microscopic nature of the ulcerations, and the absence of typical gross herpetic vesiculopapular lesions, the perianal ecchymosis was misinterpreted as possible abusive injuries.

This case demonstrates how disseminated neonatal herpetic infection with early anal involvement can simulate abusive trauma. A review of PubMed reveals several other natural disease processes associated with anal changes that raise the possibility of abuse; however, there are no previous reports of disseminated neonatal herpes simulating anal sexual abuse. Therefore, clinicians and pathologists may be unaware of such an occurrence and misinterpret early anal infection in neonatal herpes as abusive injury. This misinterpretation is understandable given the potentially diverse presentation, risks, and manners of transmission of neonatal herpetic infections. In addition, occult genital infections of women and delayed manifestations of disseminated neonatal infections further complicate the correct diagnosis of herpes infections in the newborn (1,2). Consequently, the possibility of a neonatal herpetic infection must be considered in newborn infants who present soon after delivery with suspicious anal changes to prevent unfounded allegations of sexual abuse.

Case History

The decedent was an 8-day-old newborn female who was delivered by Cesarean section for fetal distress. Prior to delivery, the mother had a fever of 103°F with a vaginal swab positive for Group B Streptococcus growth. The prenatal maternal history was otherwise essentially unremarkable, except for a treated trichomonas infection. Of note, there was no reported history of maternal or paternal herpetic infections. After the delivery, the newborn infant developed a fever with blood bandemia and discoloration of her lower abdomen. Because the possibility of sepsis was raised, bacterial blood cultures were obtained and a prophylactic antibiotic regimen of ampicillin and gentamycin was initiated. The blood cultures were negative for growth, and the antibiotics were subsequently discontinued after 72 h. The newborn infant remained clinically stable with resolution of the bandemia. She was eventually discharged home 5 days following delivery.

While at home, the newborn infant developed progressive lethargy with decreased oral intake and new onset of blood tinged stools. The newborn infant was further evaluated and readmitted to the hospital 2 days following her initial discharge. At the time of her second admission, the newborn infant was experiencing tachycardia and hypothermia with a temperature of 95.7°F. Grunting respirations were documented with weak pulses and cool extremities. The physical examination revealed harsh breath sounds and a distended abdomen. Rectal prolapse was noted with perianal ecchymosis and a questionable superficial anal tear at the 1:00 o'clock

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position. The anal changes were of new onset with the mother noticing them on the day of this second hospital admission with previous rectal examinations reportedly normal in appearance. Given the new onset and the overall appearance, these anal findings were noted as suspicious for abusive injuries. Laboratory studies were significant for leukopenia (1.5 K/mm^3), anemia (Hgb 5.0 mg/dL with Hct 15%), thrombocytopenia (105 K/mm^3), hypoglycemia (39 mg/dL), and a metabolic acidosis. Chest and abdominal radiographs showed haziness of both lungs with a questionable right pulmonary infiltrate and distended loops of bowel with increased air. Based on these findings, the admission diagnosis was septic shock. Despite aggressive medical care including bacterial antibiotics, she was pronounced dead appropriately 24 h later.

Given the sudden nature of the infant death and the suspicion of abusive anal trauma, the decedent was sent for autopsy. The autopsy revealed a female newborn infant with external dimensions that were normal for age. External examination was free of oral and nonanal cutaneous lesions other than iatrogenic needle puncture wounds of the wrists, antecubital fossae, left ankle, and heels. The anus was dilated and possessed a circumferential ecchymosis with fine vague erosions at the 1:00 o'clock position. The distal rectal mucosa was mildly prolapsed with light mucopurulent discharge (Fig. 1). The abdomen was moderately distended. Internal examination showed diffuse pallor of the muscles and organs with mild to moderate ascites and bilateral pleural effusions. The heart had a widely patent secundum atrial septal defect. There was patchy diffuse red-brown mottling of the lungs and liver with a moderate ileus of the small bowel. The intestinal mucosa including the rectum was essentially unremarkable. There was no evidence of internal traumatic injuries. Whole body skeletal radiograms were also free of boney trauma.

Hematoxylin and eosin (H&E) light microscopy demonstrated a patchy necrotizing pneumonitis and a diffuse necrotizing hemorrhagic hepatitis. The hepatocytes possessed herpetic cytopathic changes with occasional multinucleated cells, smudged nuclei, and fine granular light purplish intranuclear Cowdry A inclusions (Fig. 2). The adrenal glands had foci of hemorrhages with multinucleation and isolated Cowdry A viral nuclear inclusions. Rare encephalitis was present consisting of scant tiny clusters of cerebral mononuclear cells. The anus revealed areas of dermal hemorrhages with rare microscopic ulcerations and early leukocytoclastic-like vasculitis consisting of extravasated red blood cells with a mixed inflammatory infiltrate of neutrophils and lymphocytes with associated nuclear dust (Fig. 3). Within the ulcerations, a cluster of degenerating keratinocytes was identified with herpetic viral cytopathic changes consisting of steel-gray nuclei with nuclear margination and smudging (Fig. 4).

To confirm the initial H&E microscopic impression of disseminated herpetic infection, herpes types I and II immunohistochemical cocktail stains were performed of the liver and lungs, which showed strong nuclear and mild to moderate cytoplasmic positivity (Figs 5 and 6). A postmortem bacterial blood culture revealed *Escherichia coli* growth. The toxicology was noncontributory. Based on these autopsy findings, the cause of death was listed as systemic multiorgan failure caused by disseminated neonatal herpetic infection with *E. coli* sepsis as a contributory factor. The manner of death was natural. Of note, the autopsy revealed no evidence of nonaccidental traumatic injuries to document abuse. Instead, the grossly appearing perianal ecchymosis that had initially raised the possibility of sexual abuse was secondary to early anal involvement by the unsuspected neonatal herpetic infection.

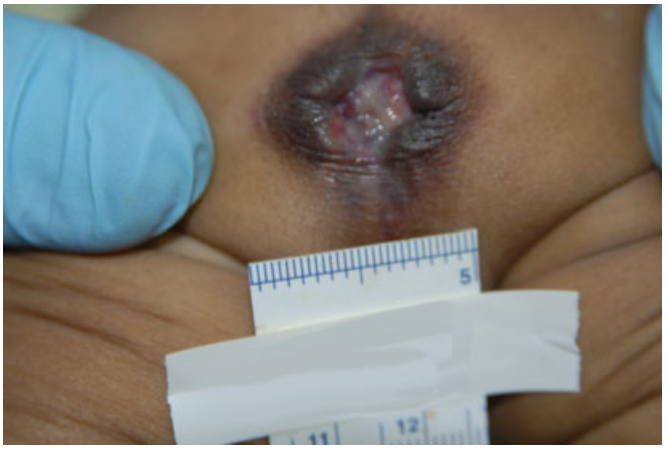

FIG. 1—*Postmortem picture demonstrating perianal hemorrhages and mucopurulent discharge.*

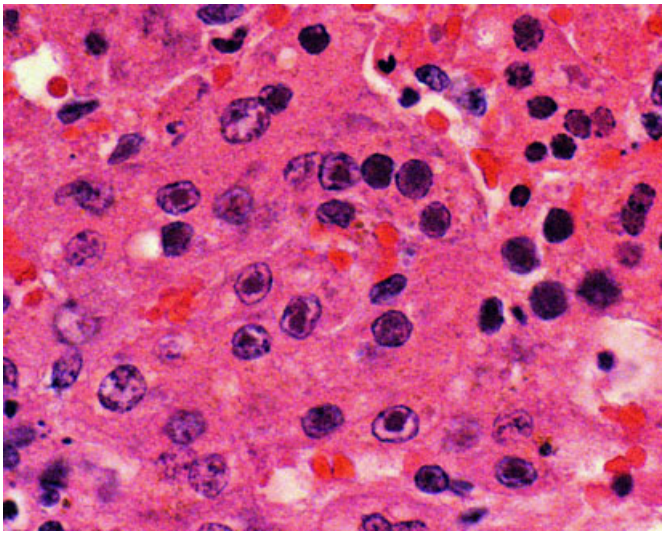

FIG. 2—*Herpetic Cowdry A nuclear cytopathic changes of the liver (H&E 40×).*

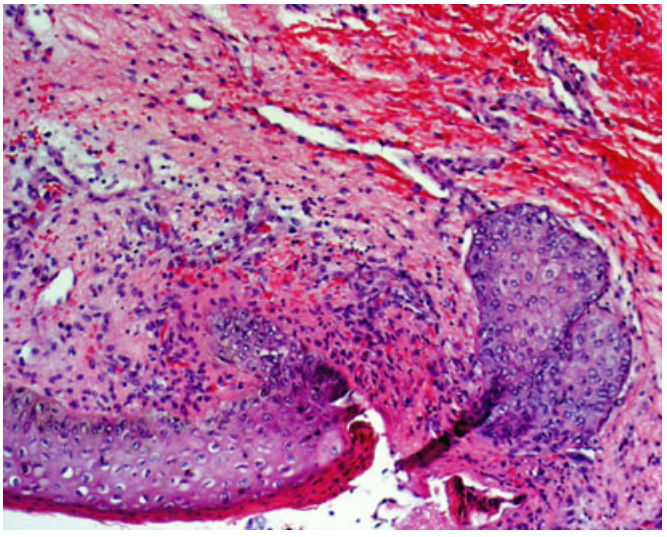

FIG. 3—*Perianal ulceration with early leukocytoclastic vasculitis consisting of extravasated red blood cells, mixed inflammatory infiltrates, and nuclear dust (H&E 20×).*

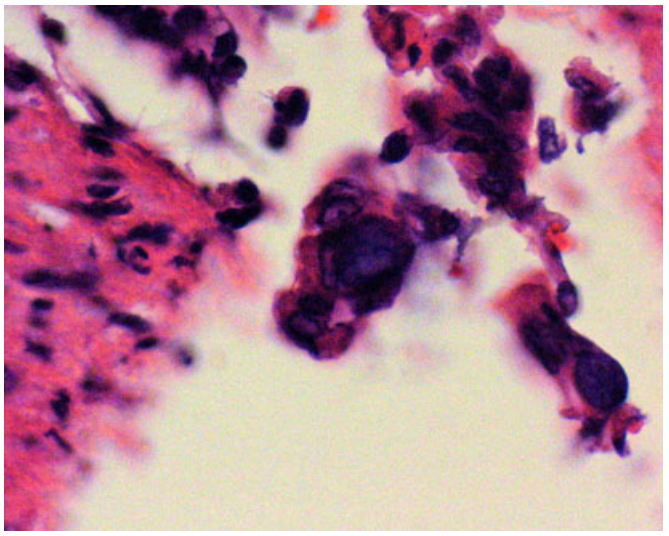

FIG. 4—*Perianal ulceration with cluster of degenerating keratinocytes with herpetic nuclear cytopathic changes (H&E 40×).*

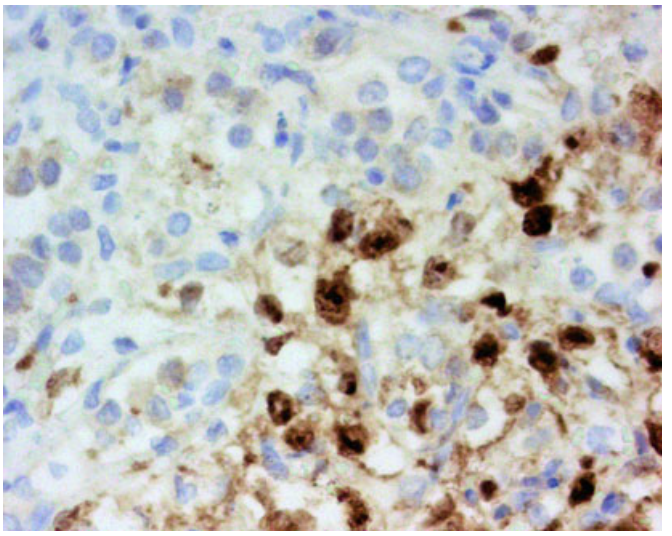

FIG. 6—*Herpes simplex virus I and II: immunohistochemical stain of lungs (40×).*

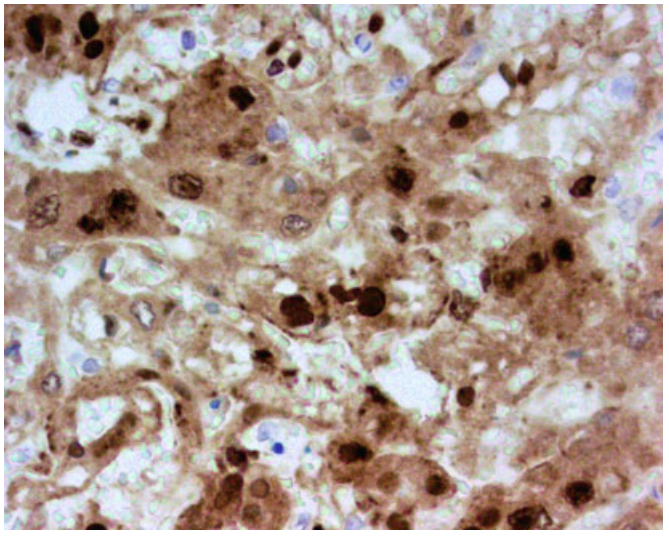

FIG. 5—*Herpes simplex virus I and II: immunohistochemical stain of liver (40×).*

Comment

This case demonstrates an unusual presentation of disseminated neonatal herpes that was clinically unsuspected and produced anal changes that were suspicious for sexual abuse. A review of PubMed was performed and revealed no previous reports of such an occurrence. This case report will promote awareness of possible unsuspected neonatal herpes in newborn infants with anal abnormalities, thereby preventing misinterpretation of the anal findings. Unfortunately, such interpretation is complicated by the diverse presentation, risks, and manners of transmission in neonatal herpes. Neonatal herpetic infection can present in one of three ways, including disseminated visceral infections, isolated meningoencephalitis, or limited infections of skin, eyes, and/or mucous membranes (3). The risk of neonatal herpes depends on the type of maternal herpetic infection during pregnancy with risks of 30–50% for primary genital herpes, 4–8% for active recurrent herpes at parturition, and 0.3–3% for asymptomatic viral shedding (1).

The manner of transmission varies in three ways. First, direct inoculation is probably the most widely recognized manner with transmission occurring during vaginal delivery with exposure to the virus from either active lesions or asymptomatic viral shedding. Direct inoculation is also possible by the use of fetal scalp electrodes with a documented case of herpetic skin lesions developing at the site of electrode implantation with subsequent fatal disseminated infection following delivery (4). Interestingly, this case of electrode-induced disseminated herpes was associated with no maternal history of herpes. Second, vertical ascension is another possible manner of transmission with intrauterine infection occurring following ruptured fetal membranes (5). Finally, the manner of transmission may remain undetermined with no obvious route as demonstrated by a case of neonatal keratoconjunctivitis following an elective Cesarean section with intact membranes and a negative maternal herpetic history (3).

Besides the above-described variations in presentation, risk, and manners of transmission, the diagnosis of neonatal infection is further complicated by the fact that maternal genital herpes may be occult in 60–80% of women (6). This occult genital infection leads to potentially untreated asymptomatic shedding, which may be a source of neonatal infection. A final problem in diagnosis centers on neonatal herpetic infections that occur in the background of no maternal history of herpes. This absence of a maternal history promotes a false impression with failure to consider the possibility of neonatal herpes, despite the fact that such infections arise with a negative maternal history as documented in the above electrode implantation and keratoconjunctivitis cases (3–5). Of note, the decedent of this case report also presented with no maternal or paternal herpetic histories.

Given these problems in diagnosing neonatal herpetic infection, pathologists must possess a heightened suspicion at autopsy in detecting occult herpes. The importance of correctly diagnosing occult neonatal herpes infection is highlighted by this case, which was associated with mucocutaneous involvement of the anus simulating abusive injury. This anal herpetic infection appears to be early, given the clinical history of rectal bleeding beginning appropriately 24–36 h before death as noted by the mother and the absence of classic appearing herpetic vesiculopapular lesions. Instead of the typical herpetic mucocutaneous lesions, the anal herpes manifested as ecchymosis with no gross ulceration making premortem diagnosis of herpes difficult. Careful microscopic examination of the anus confirmed herpetic involvement of the anus by

the disseminated infection. This light microscopy demonstrated dermal hemorrhage with a leukocytoclastic-like vasculitis, which has been reported with herpetic infection (7). In addition, there were rare microscopic ulcerations with isolated viral cytopathic changes. Given the predominance of the dermal hemorrhage with the microscopic nature of the herpetic ulcerations and no gross vesiculopapular lesions, it was understandable how this early herpetic anal involvement with ecchymosis was misinterpreted as possible traumatic injury. Nevertheless, the correct interpretation of the perianal ecchymosis as nonabusive was critical in preventing false child abuse allegations.

Potential confusion at autopsy between nonabusive changes and abusive injuries represents pitfalls that may lead to serious injustices if not carefully evaluated. Some of these nonabusive artifacts are obvious to the pathologist, such as bruising of the sternum from cardiopulmonary resuscitation, postmortem scrotal drying simulating diaper rash, or postmortem insect activity resembling traumatic abrasions. Other autopsy findings are more challenging in terms of their significance as markers of child abuse with potential inaccurate interpretation if one is unaware of their true etiology. For instance, a recent case report describes anomalous suture defects of the parietal skull that simulate a traumatic fracture on gross examination, unless one is aware of this entity and performs microscopic examination (8).

When dealing with genital lesions in infants or children that raise the possibility of abuse, the pathologist must carefully assess the findings to ensure that nonabuse changes are excluded before diagnosing abusive trauma. As this case report demonstrated, a careful autopsy is necessary to rule out natural disease processes as the source of genital lesions. Other articles have documented nonabusive changes besides neonatal herpetic infection that are potentially confused with abusive genital injuries. One such nonabusive change is infantile perineal protrusion, which is described as a skin tag appearing lesion of the perineal median raphe area in infants that may be misinterpreted as sexual abuse (9). In addition, perianal hemorrhages and cutaneous excoriations are associated with other natural diseases, including perineal streptococcal infection, midgut volvulus with intestinal malrotation, and ectodermal dysplasia clefting syndrome (10).

This case report introduces yet another potential source of confusion for perianal hemorrhages that raise the possibility of sexual abuse, namely unsuspected neonatal herpetic infection with early anal involvement. This confusion is particularly likely if the newborn infant is discharged home following delivery without a neonatal herpetic diagnosis and subsequently presents back to the hospital with new onset of perianal hemorrhages. Such a delay in diagnosis of neonatal herpes following delivery has been documented in other cases, including 16-day-old and 4-day-old newborn infants (2). Consequently, pathologists must consider the possibility of neonatal herpetic infection in newborn infants who later develop perianal hemorrhages to prevent misdiagnoses of abusive injuries that may lead to false accusations of caregivers.

Conclusion

A female newborn infant was born by Cesarean section for a fetal distress with the maternal and paternal history negative for herpes infections. Prior to delivery, the mother was febrile with a vaginal swab positive for Group B Streptococcus growth. Following delivery, the newborn infant developed a fever with abdominal discoloration. She was treated with bacterial antibiotics and discharged home 5 days later, but returned back to the hospital with septic shock and died 24 h later. Perianal ecchymosis was noted, which raised the possibility of sexual abuse. An autopsy demonstrated unsuspected disseminated neonatal herpes with early involvement of the anus, producing perianal hemorrhages that simulated traumatic injury. This case report highlighted the importance of considering neonatal herpetic infections in perianal hemorrhages that are suspicious for abusive sexual injuries. Other nonabusive changes simulating sexual abuse have also been described, including infantile perineal protrusion, perineal streptococcal infection, midgut volvulus with intestinal malrotation, and ectodermal dysplasia clefting syndrome. Consequently, the pathologist must carefully exclude other nonabusive changes when evaluating genital lesions to ensure the correct diagnosis of abusive injuries.

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