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A Case of Isolated Intrauterine Cutaneous HSV

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BACKGROUND

In the United States, genital infection with HSV is quite common. An estimated 65% of pregnant women in the United States harbor herpes simplex virus (HSV) 1 or 2 with a genital infection [2]. Neonatal HSV is defined as an infection in a newborn within 28 days of birth and has potentially devastating consequences [1]. Untreated, neonatal HSV carries a 60% mortality rate. While infection rates differ geographically, rates as high as 60 per 100,000 live births have been reported in the United States [1]. Alternatively, intrauterine infection rates are reported at 1 in 300,000 deliveries. Most neonatal HSV cases are the result of exposure in the genital tract during delivery, however, intrauterine and post-natal infections do also occur. Intrauterine HSV infection is frequently excluded from descriptions of neonatal rates and previously have been thought to exhibit signs and symptoms limited to skin, the central nervous system, and eyes [2]. Recurrent genital herpes infections are the most common form of neonatal infection, however, infants born to mothers with newly acquired HSV during pregnancy are at the highest risk of obtaining the virus [2,3]. Neonatal transmission arises when the gravid mother is shedding the virus, however, most genital HSV infection occur initially without any signs or symptoms in the mother [3]. Furthermore, a large percentage of neonatal cases do not present with the classic findings involving skin, central nervous system, and eyes. Due to this the clinician must have a high index of suspicion and start empiric therapy as delay in treatment can significantly affect the clinical outcome for the neonate[2].

CASE PRESENTATION

A male infant was born at 35 5/7 weeks gestation by spontaneous vaginal delivery due to premature labor in a 22-year-old gravid one woman who received prenatal care. Pregnancy was complicated by Covid-19 infection at 12 weeks gestation but was otherwise uncomplicated. APGAR scores at birth were eight and nine due to associated coarse breath sounds and tachypnea. He was placed on heated high flow nasal cannula and transferred to the NICU. On initial exam the infant was noted to have erythematous, denuded skin on the right side of the neck, shoulder, inguinal region, scalp, and back.

The lesions were in various stages of healing and none appeared vesicular or bullous in nature. Several areas of hypopigmentation were noted on the lower back. **(Figure 1a, Figure 1b, Figure 1c)**. Physical exam was otherwise normal aside from the skin findings. White blood cell count was 6200 mm3 with 14% neutrophils, 27% bands, 49% lymphocytes, and 10 percent monocytes. Hemoglobin was 13.0, hematocrit 38.7, and platelets 316. Complete metabolic panel was significant for glucose of 110 and CO2 of 24.

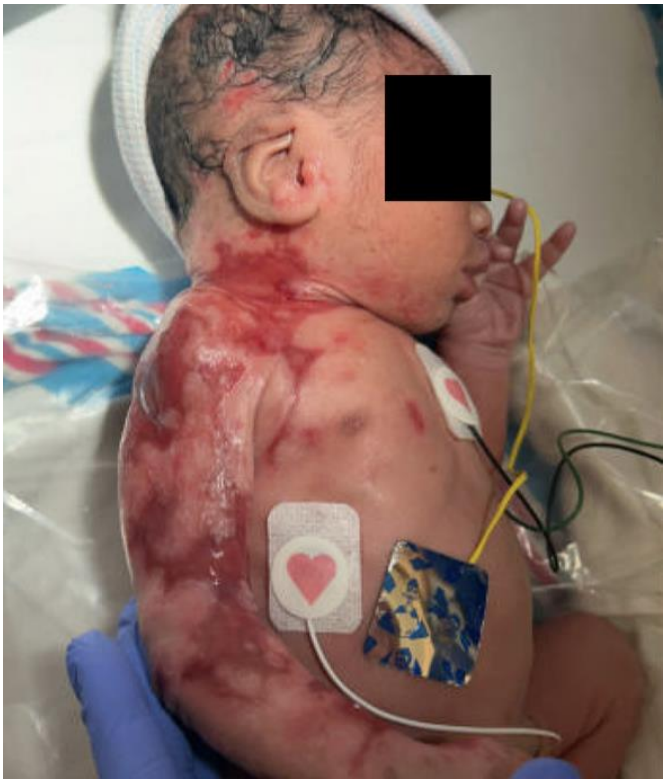
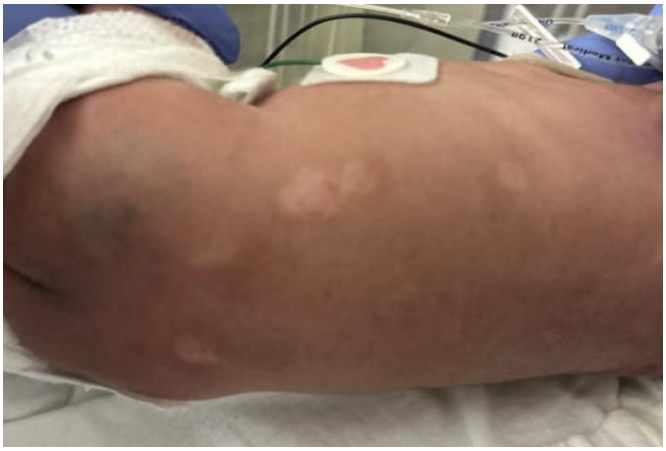


Figure 1a: Day of Life One



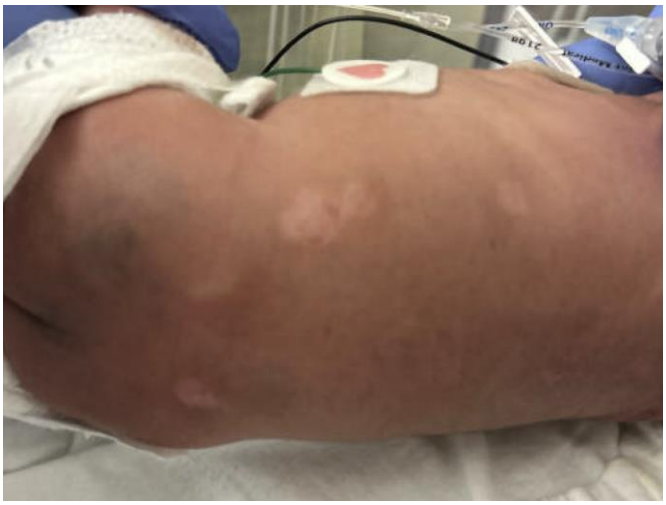


Figure 1b: Day of Life One

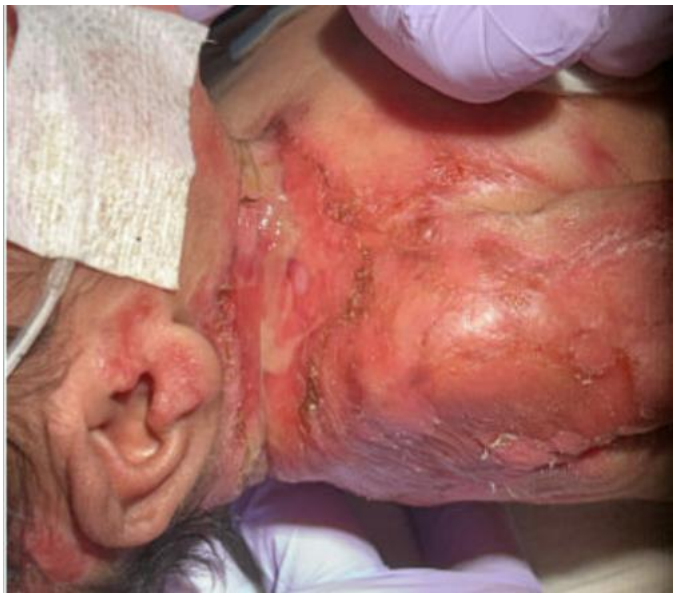


Figure 1c: Day of Life One

Initial infectious workup including cultures, HSV polymerase chain reaction (HSV PCR) and lesion swab were negative for causative agent. Initial lesion biopsies demonstrated dense inflammatory cell infiltrate and denuded epidermis without viral histopathology. Maternal serologies were negative for GBS, RPR, rubella, HIV, Hepatitis B and C, gonorrhea, and chlamydia during pregnancy. At eleven days of life, the patient had an eruption of vesicular lesions in the groin, axilla and face. The new lesions were noted to be remote from the original skin lesions. Vesicles

had a positive HSV PCR. Biopsy demonstrated necrotic keratinocytes with positive HSV immunohistochemical staining. The final diagnosis was made 14 days after birth and therapy was started with IV acyclovir. The infant received IV therapy for two weeks and was transitioned to oral acyclovir for suppressive therapy at discharge for a duration of 6 months. By six months, the infant was seen in office and demonstrated healed lesions **(Figure 2a, Figure 2b)**. There was a recurrence of infection necessitating re-initiation of therapy for a brief period in the outpatient setting. The patient demonstrated no clinical or laboratory evidence of systemic involvement. Repeat blood HSV PCR was negative, as was HSV PCR of the cerebrospinal fluid. These findings suggest isolated cutaneous HSV infection. Isolated cutaneous infection usually presents at 1-2 weeks of life. This case is unique due to the presence of cutaneous manifestations at birth.

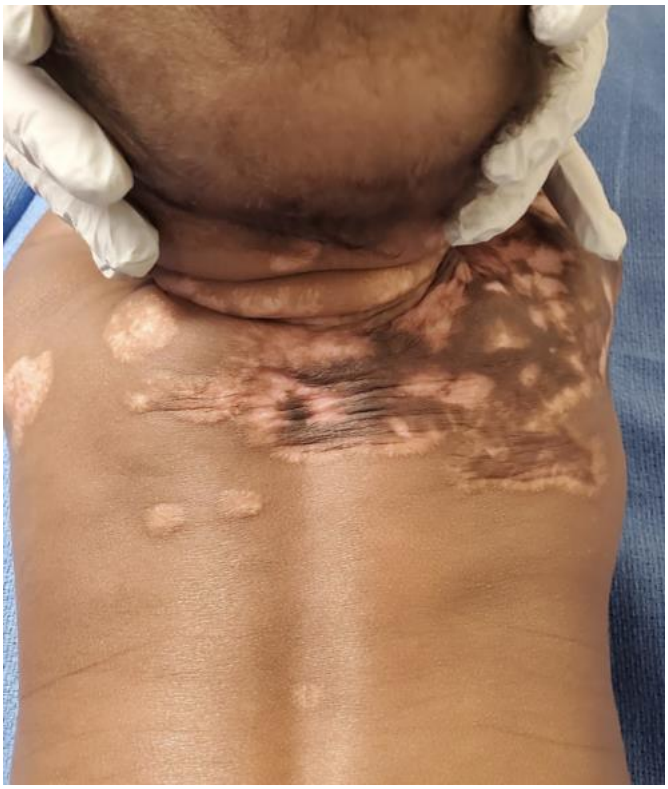


Figure 2a: 6 months

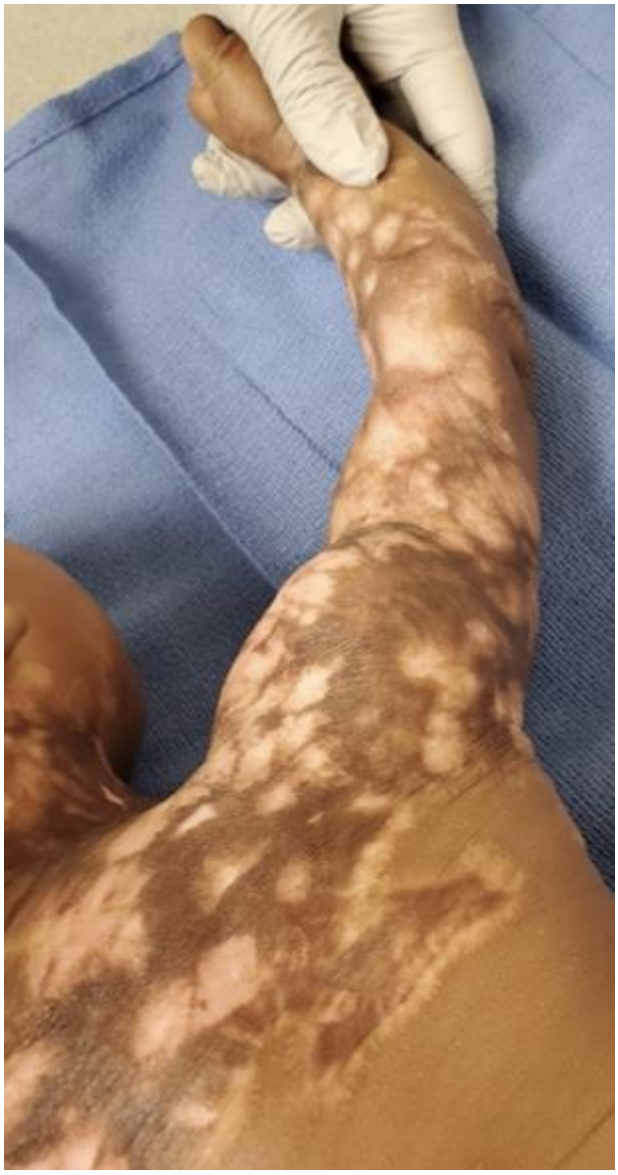


Figure 2b: 6 months

DISCUSSION

Neonatal HSV infection is infrequent and can present similarly to other cutaneous lesions as well as other congenital infections [2]. The classic presentation of an intrauterine HSV infection includes cutaneous, ophthalmologic, and central nervous system symptoms at birth, however, these manifestations are rare and the full triad is only found in roughly thirty percent of reported cases [1,2]. The most common

presentation of intrauterine HSV infection in the neonate is a myriad of cutaneous lesions, however lesion presentation is inconsistent making it one of the main reasons for delayed diagnosis. Lesions may manifest as the typical vesicular lesion, but may also exhibit cutaneous ulcers, pustules, plaques, or cutis aplasia [4-7]. Due to the variability of presentation, lesions can be mistaken for TORCH infections, epidermolysis bullosa, cutis aplasia congenita, a varicella syndrome, or incontinentia pigmenti [4,8,9]. Similar to our case, there are three other cases reported that describe the presentation as skin denudation. This is believed to be a manifestation of scarring as sequela of vesicular eruptions that occurred earlier in the pregnanc [2]. As we discovered, the other cases also received false negative viral cultures and only on the re-culture of fresh vesicles was the definitive diagnosis made [10]. Another infrequent presentation with few cases reported is a zosteriform eruption in the distribution of a dermatome, confusing the disease with a congenital varicella syndrome [6].

Early identification of intrauterine HSV infection is essential as it can lead to devastating outcomes for the neonate such as spontaneous abortion, stillbirth, neurodevelopmental impairment, scarring, and even death [4]. The recommendation for diagnosis is recognition of the disease within 48 hours of birth, virologic confirmation, and exclusion of other pathological conditions [4,5]. 85% of reported cases of neonatal transmission of HSV can be attributed to HSV exposure during delivery or the peripartum period [6]. Post natal transmission is more commonly due to HSV-1 and is due to contact with hospital staff or family who are shedding the virus. Genital HSV infections in the mother can be subclinical and therefore incorrectly diagnosed by clinicians [2]. Intrauterine HSV infection is the rarest form of the neonatal disease and is estimated to be involved in one of every 300,000 deliveries. Only five percent of the neonatal cases occur from in utero transmission. When HSV transmission does occur in utero, there are two modalities of fetal exposure; trans-placental or ascending infection (which can occur without rupture of membranes).

The fetus can acquire the virus both from primary and recurrent HSV infection [6].

When concerning cutaneous findings are present at birth or within the first 48 hours of life, acyclovir should be started as it is critical in the treatment of intrauterine HSV infection and progression of disease. It cannot, however, prevent damage from in utero exposure. Once IV acyclovir is initiated in the perinatal period, the neonate should be placed on oral acyclovir for six months following discharge. Up to one half of patients affected can experience recurrence once IV acyclovir is discontinued so it is important to have suppressive therapy to prevent skin recurrence but also to prevent any neurodevelopmental consequences [7].

CONCLUSION :

Intrauterine HSV exposure can be difficult to diagnose. Occurrence is uncommon and has a variable presentation. Unlike the majority of other HSV infections, intrauterine infection does not necessarily have the quintessential zosterifrom rash. Furthermore, the age of the lesions may lead to false negative culture, serology, or biopsy results adding to the difficulty of diagnosing this infection. It is imperative that the clinician has a high index of suspicion and starts IV acyclovir early as it is critical to limiting progression of disease and improving outcomes. Once the diagnosis is confirmed, a suppressive dose of oral acyclovir should be continued for six months.

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