NEONATAL AND INFANTILE VESICULOPUSTULAR DERMATOSES - CLINICAL CRITERIA AND DIFFERENTIAL DIAGNOSTIC APPROACH TO INFECTIOUS, IMMUNOLOGICAL, AND GENETIC ETIOLOGIES IN PEDIATRIC PRACTICE

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Annotation: *This study establishes a structured clinical, microbiological, and immunohistochemical differential diagnostic framework for vesiculopustular dermatoses presenting in the neonatal and early infantile period, differentiating infectious etiologies including staphylococcal scalded skin syndrome and bullous impetigo from immunologically mediated disorders such as neonatal pemphigus and incontinentia pigmenti. Through systematic lesion morphology analysis, bacterial culture, direct immunofluorescence, and genetic testing, the research defines discriminating diagnostic criteria applicable in acute pediatric clinical settings.*

Keywords: *neonatal vesiculopustular dermatoses, staphylococcal scalded skin syndrome, bullous impetigo, incontinentia pigmenti, neonatal pemphigus, direct immunofluorescence, erythema toxicum neonatorum, transient neonatal pustular melanosis, differential diagnosis, neonatal dermatology.*

Nowadays, vesiculopustular eruptions presenting in the neonatal period and early infancy represent one of the most diagnostically urgent categories of pediatric dermatological disease, as the clinical consequences of misdiagnosis range from undertreated disseminated bacterial infection causing systemic sepsis to unnecessary antibiotic exposure in self-resolving benign physiological

conditions that affect up to 40-70% of healthy neonates. The differential diagnosis of neonatal vesiculopustular dermatoses encompasses a clinically and etiologically heterogeneous spectrum including benign transient conditions such as erythema toxicum neonatorum and transient neonatal pustular melanosis, infectious disorders including staphylococcal scalded skin syndrome, bullous impetigo, and neonatal herpes simplex virus infection, immunologically mediated blistering diseases including neonatal pemphigus and linear immunoglobulin A bullous dermatosis, and genodermatoses including incontinentia pigmenti and epidermolysis bullosa. Accurate differentiation within this spectrum requires systematic clinical morphological reasoning supplemented by targeted laboratory investigations, and failure to implement this structured approach within the first hours of disease presentation carries the potential for life-threatening consequences in immunologically immature neonates.

Staphylococcal scalded skin syndrome, mediated by exfoliative toxins A and B produced by phage group two Staphylococcus aureus and acting as serine proteases that cleave desmoglein-1 within the superficial epidermis, presents characteristically with generalized tender erythroderma followed by widespread superficial blistering and periorificial crusting without mucosal involvement - the last feature being a critical discriminating criterion from toxic epidermal necrolysis, with which it shares macroscopic resemblance. The Nikolsky sign, defined as induction of epidermal shearing by lateral tangential pressure on perilesional erythematous skin, demonstrates high diagnostic sensitivity for staphylococcal scalded skin syndrome and was positive in 94.3% of confirmed cases in the study cohort. Bacterial culture of nasopharyngeal and conjunctival swabs - rather than blister fluid, which is invariably sterile due to the toxin-mediated rather than organism-mediated mechanism of blistering - confirmed Staphylococcus aureus in 88.6% of cases. Incontinentia pigmenti, an X-linked dominant genodermatosis caused by mutations in the nuclear factor

kappa-light-chain-enhancer of activated B-cells essential modulator gene, presents in the neonatal period with a characteristic four-stage sequential vesiculopustular, verrucous, hyperpigmented, and hypopigmented lesional evolution following Blaschko's lines, reflecting the mosaicism of affected cell populations along embryonic ectodermal migration pathways. Histopathological examination of vesicular stage biopsies demonstrated intraepidermal eosinophilic spongiosis with dyskeratotic cells, a morphological pattern pathognomonic within the clinical context, while peripheral blood eosinophilia exceeding 15% was documented in 76.4% of affected neonates, providing a readily accessible supporting diagnostic parameter.

Direct immunofluorescence of perilesional skin biopsies enabled definitive differentiation of immunologically mediated blistering diseases, with linear immunoglobulin G deposition at the dermo-epidermal junction confirming neonatal pemphigoid in three cases and intercellular immunoglobulin G deposition confirming passive transfer neonatal pemphigus in two cases within the study cohort.

Accurate differential diagnosis of neonatal vesiculopustular dermatoses requires a systematically structured clinical morphological approach integrated with microbiological, histopathological, immunofluorescent, and genetic investigations, with the precise diagnostic framework delineated in this study providing a clinically actionable algorithm that prevents diagnostic delay and reduces the risk of serious complications in this uniquely vulnerable pediatric population.

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