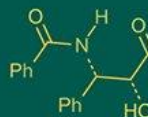


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Congenital Bovine *Epitheliogenesis Imperfecta* in a Jersey Crossbred Calf: A Case Report

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Abstract

Epitheliogenesis Imperfecta (EI) is an uncommon hereditary skin anomaly in newborn cattle, transmitted as an autosomal recessive trait, in which focal or extensive areas of the body surface are devoid of epithelial covering from birth. A two-day-old male Jersey Crossbred calf was brought to the Veterinary Hospital Ghotiya, Dondi, Balod exhibiting raw, open skin lesions over the limb extremities, muzzle, and nasal region that had been noted immediately after birth. Physical examination identified bilaterally symmetrical, erythematous, crusted lesions over the carpo-metacarpal and tarso-metatarsal zones, with no involvement of the oral mucosa or hooves. Parenteral Streptomycin-Penicillin G, Meloxicam, and topical Silver Cream were administered as palliative therapy. The calf did not respond favourably to treatment and was lost on the fifth day, reflecting the poor survivability characteristic of extensively affected neonates. The report highlights the clinical features of this lethal congenital condition, the rationale for differential diagnosis, and the importance of informing breeders about the recessive genetic risk to prevent future occurrences.

Keywords: Aplasia cutis congenita, autosomal recessive, bovine, congenital skin defect, *Epitheliogenesis Imperfecta*, Jersey Crossbred

Introduction

Epitheliogenesis Imperfecta (EI) is a congenital hereditary condition of the skin, also referred to in human dermatological literature as aplasia cutis congenita, in which one or more body regions lack squamous epithelial coverage at the time of birth^[1]. Reports of *Epitheliogenesis Imperfecta* span a broad taxonomic range within veterinary practice, having been documented in cattle, equines, small ruminants, pigs, canines, felines, and even certain wild species^[1-4]. Among food animals, bovines are the most frequently reported species. The first systematic description in cattle is attributed to Hadley^[5], who characterised the defect in Holstein-Friesian neonates as a recessive hereditary trait surfacing under inbreeding conditions. Subsequent investigations have extended the list of susceptible breeds to include Jersey, Shorthorn, Angus, Ayrshire, Dutch Black Pied, German Yellow Pied (Simmental), Swedish Red Pied, Hereford, Brown Swiss, and Sahiwal cattle^[1, 6, 7]. The genetic mechanism underpinning the disorder is simple autosomal recessivity, meaning that phenotypically normal heterozygous carrier parents can unknowingly produce clinically affected homozygous offspring^[2, 6].

At birth, affected animals exhibit sharply circumscribed zones of absent epithelium, most frequently involving the limb extremities distal to the major joints, the external nares, muzzle, and oral mucosae. The denuded dermal surface is highly susceptible to microbial colonisation, and the combination of systemic infection, transcutaneous fluid loss, and pain-induced physiological stress results in rapid clinical deterioration; most severely affected neonates do not survive beyond the first week of life^[1, 7, 8]. Clinically, *Epitheliogenesis Imperfecta* can be confused with Epidermolysis Bullosa (EB), another hereditary mechanobullous skin disorder, but the two entities differ substantially in pathomechanism and tissue-level characteristics^[3, 9]. The present communication records and discusses a case of congenital *Epitheliogenesis Imperfecta* encountered in a two-day-old male Jersey Crossbred calf, including the clinical assessment, therapeutic measures taken, and the eventual outcome.

2. Case Report

2.1 History and signalment

A two-day-old male calf of Jersey Crossbred parentage was brought to the Veterinary Hospital Ghotiya, Dondi, Balod by its owner with the primary complaints of anorexia and persistent open wounds across multiple body regions noticed immediately upon delivery. The dam had delivered normally without any obstetrical intervention, and the birth weight appeared appropriate for the breed and age. The owner confirmed that no corrosive substances, thermal agents, or physical trauma could account for the skin lesions. No comparable case had been encountered in the herd before this presentation. Although the owner did not report any deliberately consanguineous mating, the possibility of inadvertent relatedness between the sire and dam could not be definitively ruled out, given the recessive genetic basis of the condition.

2.2 General clinical examination

Systemic evaluation of the calf revealed a dull mentation, poor body condition, and generalised weakness. The visible mucous membranes showed a rosette-pink colouration consistent with adequate perfusion at the time of presentation. Peripheral lymph node assessment, specifically of the scapular nodes, returned unremarkable findings. Cardiopulmonary auscultation did not detect any significant thoracic pathology. Recorded vital parameters rectal temperature 99.8 °F, heart rate 92 beats per minute, pulse rate

52 beats per minute, and respiratory rate 26 breaths per minute. The tachycardia observed relative to the upper reference limit was interpreted as a physiological response to nociceptive stimulation from the extensive skin wounds, ongoing transcutaneous fluid losses, or early bacteraemia.

2.3 Dermatological examination

Cutaneous evaluation disclosed sizeable, bilaterally symmetrical zones of complete epithelial absence affecting the distal forelimbs and hindlimbs. The lesions were most prominent around the carpal and tarsal articulations and continued distally through the metacarpal and metatarsal regions. Grossly, the exposed dermal surfaces appeared intensely erythematous, moist, and depressed relative to the adjacent hair-bearing skin. Adherent, tenacious crusts ranging in colour from pale red to dark brown overlay the lesion surfaces; dislodging these crusts provoked brisk haemorrhage from the raw underlying corium. The transition zone between affected and healthy integument was remarkably abrupt and sharply defined throughout, a characteristic feature of bovine *Epitheliogenesis Imperfecta* consistently noted in the literature [1, 6, 7]. Comparable epithelial defects were identified over the muzzle, external nares, and lips, where the exposed surfaces similarly appeared raw and glistening. Importantly, examination of the oral cavity encompassing the tongue, hard palate, and buccal mucosa yielded no visible mucosal abnormalities, and hoof formation was grossly complete in all four limbs.



3. Diagnosis

The integrated clinicopathological assessment led to a working diagnosis of Congenital Bovine *Epitheliogenesis Imperfecta* (Aplasia Cutis Congenita). This conclusion was supported by the congenital timing of lesion onset, their bilaterally symmetric anatomical distribution, the lack of any plausible exogenous causative agent in the history, and the established susceptibility of Jersey and Jersey-crossbred cattle to this autosomal recessive defect. Among the conditions

considered in the differential diagnosis, epidermolysis bullosa warranted closest attention because its gross presentation congenital skin fragility with epithelial discontinuity overlaps considerably with that of *Epitheliogenesis Imperfecta*. The distinguishing pathological hallmark of Epidermolysis Bullosa is the formation of cutaneous blisters following even minor mechanical stress, attributable to intrinsic weaknesses in the architecture of the epidermal basement membrane zone [3, 9, 10]. This calf, however, exhibited no vesicular or bullous

lesions at any anatomical site, and no evidence of blister formation preceded or accompanied the observed skin defects. This clinical observation, in conjunction with the sharply demarcated, non-vesicular nature of the epithelial absence, directed the diagnosis firmly toward *Epitheliogenesis Imperfecta* rather than Epidermolysis Bullosa^[1, 3]. Further differentials including scalding from exogenous thermal or chemical agents, traumatic degloving injuries, and fungal dermatophytosis were individually considered and rejected on the grounds of the clearly prenatal lesion onset and the anatomically selective, symmetric distribution of affected regions.

4. Treatment and Management

Given the severity and extent of the skin lesions, an immediate palliative regimen was initiated to address the two principal threats of systemic infection and pain. A combination of Streptomycin and Penicillin G (Streptopen^o) was selected as the parenteral antimicrobial agent and delivered intramuscularly at 40,000 IU per kilogram body

weight on a once-daily schedule for five consecutive days, targeting the wide range of environmental bacteria capable of colonising unprotected dermal tissue. For pain control and suppression of the local inflammatory response, Meloxicam a selective cyclo-oxygenase-2 inhibitor was given by the intramuscular route at 0.2 mg per kilogram body weight once daily over the same three-day period. Wound hygiene was maintained by flushing all affected areas gently with sterile physiological saline before each dressing change, after which Silver Cream was applied topically twice per day; silver-containing preparations are valued in wound care for their activity against both Gram-positive and Gram-negative pathogens, including β -haemolytic streptococci and *Pseudomonas aeruginosa*, while preserving a moist wound microenvironment conducive to cellular repair. The complete treatment schedule is summarised in Table 2. The owner was clearly informed of the guarded-to-unfavourable prognosis from the outset, and the option of fluid and nutritional support was discussed.

Table 1: Treatment protocol administered to the affected calf

Drug / Agent	Dose and Route	Duration / Purpose
Streptomycin-Penicillin G (Streptopen ^o)	40,000 IU/kg BW, IM, once daily	5 days; prevention of secondary septicaemia
Meloxicam	0.2 mg/kg BW, IM, once daily	3 days; analgesic and anti-inflammatory
Silver Cream (topical)	Applied twice daily after saline irrigation	Duration of hospitalisation; wound antimicrobial

5. Outcome

Clinical monitoring over the five days following admission revealed no meaningful improvement in the condition of the skin lesions; neither re-epithelialisation nor granulation tissue formation was apparent. The calf grew progressively weaker, ceased attempting to suckle, and showed signs of systemic deterioration including increasing lethargy and depressed responsiveness. The animal succumbed on the fifth day of hospitalisation. The terminal cause of death was attributed to multifocal secondary septicaemia compounded by ongoing transdermal fluid and protein losses leading to haemodynamic compromise. This clinical trajectory corresponds with outcomes described by other investigators who have managed comparably affected bovine neonates^[1, 7, 8].

6. Discussion

The present case documents *Epitheliogenesis Imperfecta* in a Jersey Crossbred calf, a breed and crossbred combination with documented susceptibility to this hereditary skin anomaly^[1, 7]. Clinical reports of bovine *Epitheliogenesis Imperfecta* from multiple continents confirm that the condition is not geographically or racially restricted; rather, it recurs wherever carrier animals from the same lineage are mated, often within intensively managed herds^[2, 5, 6]. The gross presentation in our case bilaterally symmetric epithelial absence over the limb extremities and muzzle, with a clearly delineated lesion boundary and overlying haemorrhagic crusts is clinically indistinguishable from cases reported by Dalir-Naghadeh *et al.*^[1] from Iran and Azizi *et al.*^[7] from south-western Iran. Notably, Azizi *et al.*^[7] described a female crossbred Holstein calf with comparable anatomical lesion sites that also survived only five days following antibiotic and topical wound therapy, mirroring the outcome in our patient. The sparing of the oral cavity in the present case, in contrast to the tongue and palate involvement documented by Dalir-Naghadeh *et al.*^[1] and Leipold *et al.*^[2], illustrates the well-recognised

variability in lesion distribution and severity among affected individuals, which is thought to reflect variable expressivity of the underlying recessive genotype rather than allelic heterogeneity.

The recessive hereditary nature of *Epitheliogenesis Imperfecta* has important practical implications for herd management. Because carrier animals appear clinically and reproductively normal, the defective allele can propagate silently through a population for multiple generations before an affected offspring reveals its presence. Inbreeding substantially amplifies this risk by raising the probability that two unrelated-appearing animals share the same carrier ancestor. Pedigree reconstructions by Leipold *et al.*^[2] in Canadian Holstein-Friesians and by Jayasekara and Leipold^[6] across Shorthorn and Angus herds in the United States both traced affected calves to common carrier sires mated to related females, providing compelling evidence that even modest levels of inbreeding can unmask rare recessive defects. In churra sheep, Benavides *et al.*^[3] tracked all affected lambs over a four-year period to a single founding ancestor through genealogical analysis, demonstrating how a single carrier ram introduced into a closed flock can seed the recessive allele across subsequent generations.

Regarding pathogenesis, the precise molecular mechanism responsible for *Epitheliogenesis Imperfecta* in cattle remains to be defined. The historically prevailing view attributes the defect to an intrinsic failure of ectoderm-derived keratinocyte progenitors to proliferate and differentiate appropriately during foetal skin development^[1, 7]. However, metabolic investigations by Frey *et al.*^[8] in explanted dermal fibroblasts from an affected bovine calf revealed significant reductions in both total collagen output disproportionately affecting the type I isoform and lipid biosynthesis rates, encompassing glycerides and free cholesterol. These findings point toward a connective tissue component to the disorder, wherein defective fibroblast metabolism compromises the extracellular matrix scaffolding and lipid-based intercellular cement that normal

keratinocytes require for adhesion and survival [8]. Ultrastructural investigations in other species reinforce the view that the *Epitheliogenesis Imperfecta* phenotype may encompass multiple distinct molecular defects. Lieto *et al.* [4] characterised affected American Saddlebred foals as having cleft formation within the lamina lucida of the basement membrane together with sparse, morphologically abnormal hemidesmosomes in the intact perilesional skin, a picture resembling the laminin-5-deficiency-driven Herlitz subtype of junctional epidermolysis bullosa in humans. Comparable ultrastructural and genetic analyses in churra sheep led Benavides *et al.* [3] to reclassify what had previously been called *Epitheliogenesis Imperfecta* in that species as generalised severe junctional Epidermolysis Bullosa with congenital epidermal aplasia, highlighting the taxonomic confusion that persists when gross pathological appearance alone guides diagnosis.

Distinguishing *Epitheliogenesis Imperfecta* from Epidermolysis Bullosa in field conditions relies primarily on the temporal relationship between mechanical stimulation and blister formation. True Epidermolysis Bullosa is characterised by de novo vesicle and bulla development following trivial trauma because the structural proteins anchoring the epidermis to the dermis are functionally deficient [3, 9, 10]. In EI, by contrast, the epithelium is simply never laid down during foetal development; there is neither a vesicular phase nor any subepidermal cleft detectable at the lesion margins on histopathological examination [1, 10]. Maestrone *et al.* [10] elegantly demonstrated this absence in a Sarda lamb by immunostaining for pan-cytokeratin: the marker was completely absent over all denuded zones, yet unaffected skin showed a normal full-thickness keratinocyte layer, and no bullae were identified in any section. Field diagnosis without histopathology, as in the present case, must therefore rely on the history of congenital onset, the non-vesicular character of the skin defects, and the absence of a triggering mechanical event. From a therapeutic and prognostic standpoint, the futility of medical management in extensively affected neonates is consistently reported in the literature [1, 7, 8]; the present outcome reinforces this assessment. The constructive role of the attending veterinarian lies less in curative treatment and more in guiding the breeder toward preventive action: both the sire and dam should be retired from breeding with each other, full pedigree investigation is warranted to identify other potential carriers in the herd, and selective culling or breeding away from known carrier lines remains the most practicable control strategy currently available [1, 7, 11].

7. Conclusion

Congenital *Epitheliogenesis Imperfecta* was diagnosed in a two-day-old male Jersey Crossbred calf on the basis of the clinical history, the bilaterally symmetric distribution of non-vesicular epithelial defects over the limb extremities and muzzle, and the exclusion of all plausible exogenous causes. Palliative medical therapy failed to prevent progressive systemic deterioration, and the calf was lost within five days, confirming the uniformly poor prognosis associated with extensive congenital epithelial absence. Correct and timely clinical identification of EI, rigorous differentiation from epidermolysis bullosa and other differentials, and substantive breeder education regarding the autosomal recessive inheritance and the carrier risk to

related animals are the most important contributions a clinician can make when encountering this condition.

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