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Transition Cow Management for Improving Health, Productivity and Reproductive Efficiency in Dairy Cows

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Abstracts:

The transition period, extending from three weeks prepartum to three weeks postpartum, represents the most critical phase influencing health, productivity, and reproductive efficiency in dairy cows. During this time, profound metabolic, endocrine, and immunological adjustments occur as cows shift from gestation to lactation. Reduced dry matter intake coupled with increased nutrient demands predisposes animals to negative energy balance, hypocalcemia, and impaired immune function. Consequently, major postpartum disorders such as retained fetal membranes, metritis, mastitis, milk fever, ketosis, left displacement of the abomasum, and laminitis commonly arise, leading to reduced milk yield, delayed uterine involution, prolonged days open, and increased culling rates. Effective transition management integrates controlled-energy nutrition, mineral balance optimization including DCAD strategies, hygienic calving practices, early disease detection, and timely therapeutic intervention. Precision monitoring tools and metabolic profiling further enhance early diagnosis of subclinical conditions. Strengthening metabolic resilience and immune competence during this window is essential to improving herd health, maximizing productivity, and ensuring sustainable dairy production systems.

Keywords: Retention of fetal membranes, metritis, mastitis, ketosis, left displacement of abomasum, laminitis, milk fever.

Introduction:

The transition period, spanning approximately three weeks before to three weeks after calving, represents the most critical and biologically demanding phase in the productive life of a dairy cow. During

this interval, the cow undergoes an abrupt physiological transformation from a pregnant, non-lactating state to one of intense metabolic activity required to support colostrum synthesis and peak milk production. This transition is not merely a change in production status; it is a complex systemic reprogramming involving endocrine shifts, metabolic adaptation, immune modulation, and profound alterations in nutrient partitioning. The success or failure of this adaptive process determines subsequent health, productivity, and reproductive efficiency. At parturition, dry matter intake typically declines, while energy, glucose, amino acid, and mineral demands increase dramatically. The resulting negative energy balance stimulates adipose tissue mobilization, hepatic fatty acid oxidation, and ketone body production. Concurrently, calcium demand rises sharply for colostrogenesis and lactogenesis, challenging mechanisms of calcium homeostasis. If compensatory pathways fail, metabolic disorders such as hypocalcemia and ketosis develop. These metabolic disturbances are not isolated biochemical events; they impair immune competence, reduce neutrophil function, and delay uterine involution, thereby increasing susceptibility to postpartum infections such as retained fetal membranes, metritis, and mastitis. Furthermore, alterations in rumen fermentation associated with high-concentrate diets predispose cows to subacute ruminal acidosis, laminitis, and displacement of the abomasum. Thus, metabolic, infectious, and inflammatory disorders during the transition period are interlinked components of a broader systemic imbalance.

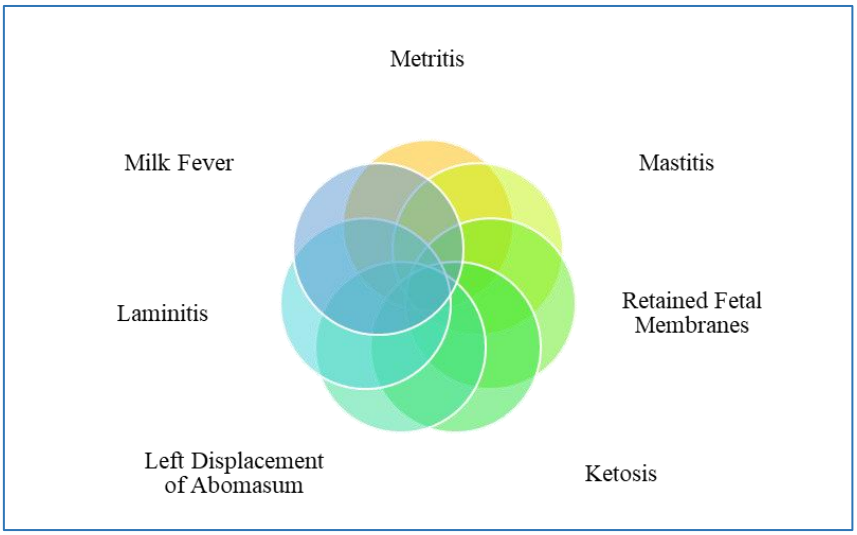

Fig. 1. Major disease during transition period

The economic consequences of inadequate transition management are substantial. Postpartum diseases reduce milk yield, increase culling rates, prolong days open, elevate treatment costs, and compromise lifetime productivity. Even subclinical conditions such as subclinical ketosis or subclinical endometritis can silently impair reproductive performance and herd profitability (Amalendu, 2003; Sahu et al., 2025). Therefore, effective transition cow management extends beyond treatment of clinical disease; it requires anticipatory nutritional planning, mineral balance optimization, controlled body condition management, hygienic calving environments, early disease detection, and strategic postpartum monitoring. Improving health, productivity, and reproductive efficiency in dairy cows demands an integrated approach that recognizes the transition period as a window of vulnerability but also of opportunity. By understanding the physiological basis of postpartum disorders and implementing evidence-based preventive strategies, it is possible to enhance metabolic resilience, strengthen immune

defense, support uterine recovery, and ultimately optimize both milk production and reproductive outcomes. The transition cow, therefore, stands at the center of sustainable dairy herd management, where precise biological stewardship translates directly into improved animal welfare and economic performance.

Major Postpartum Diseases and Their Management:

1. Retained Fetal Membranes (RFM):

Retained fetal membranes (RFM) are defined as failure of expulsion of the fetal membranes within 12 hours after calving, although some authors extend the time limit up to 24 hours postpartum. Primary retention results from failure of detachment of cotyledons from the maternal caruncles, whereas secondary retention occurs when membranes have separated but are not expelled due to mechanical difficulty such as uterine atony (Noakes, et al., 2018; Sahu et al., 2024a). Clinically, affected cows may initially show normal pulse, temperature, and milk yield, with fetal membranes hanging from the vulva; however, in prolonged or severe cases, anorexia, fever, increased pulse rate, straining, reduced milk yield, foul odor, and discolored or dry placental tissue may be observed, indicating secondary infection (Sahu et al., 2024b). Diagnosis is based on history of recent calving and persistence of fetal membranes beyond the normal expulsion period, along with clinical examination to assess uterine tone and systemic condition.

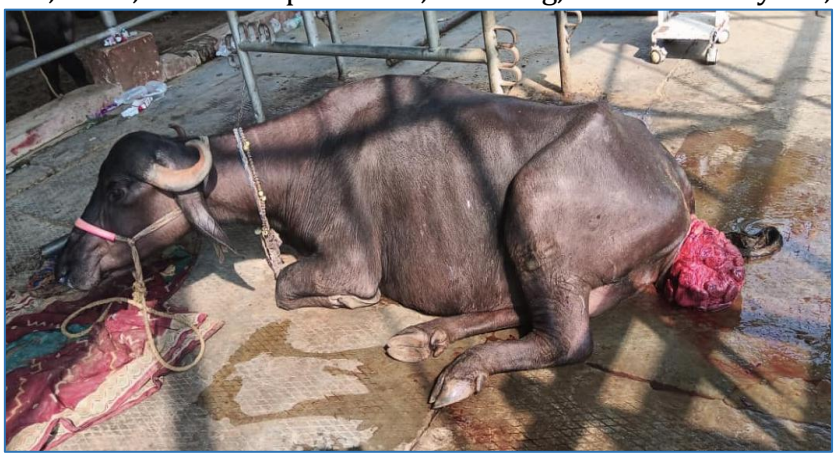

Fig. 2. Postpartum prolapse of uterus secondary to retention of fetal membrane

Treatment includes administration of oxytocin (75–100 IU intramuscularly) immediately after calving to stimulate uterine contractions, although its effectiveness diminishes after 24 hours. Indigenous or uterotonic preparations may be used to promote uterine tone and expulsion. If membranes fail to detach, careful manual removal may be performed under epidural anesthesia (5–7 ml lignocaine), with strict aseptic precautions; the placenta is grasped and twisted gently like a rope while the lubricated hand separates cotyledons from caruncles by rolling and squeezing motions, ensuring complete removal to prevent infection, though manual removal is contraindicated in febrile animals due to risk of septicemia. Intrauterine therapy using suitable preparations for 3–5 days and parenteral antibiotics such as streptopenicillin or oxytetracycline may be administered when active infection is suspected. Supportive therapy including NSAIDs, antihistamines, and liver extracts may be used. In cases associated with uterine atony due to hypocalcemia, oxytocin combined with calcium borogluconate may be beneficial. A newer therapeutic approach involves intraumbilical injection of collagenase solution prepared with collagenase, calcium chloride, sodium bicarbonate, and saline, which facilitates separation of cotyledons by promoting

proteolysis and has been reported to be effective within 36 hours in a high proportion of cases. Early management is essential to prevent complications such as metritis, delayed uterine involution, and impaired fertility.

2. Metritis:

Metritis is an acute postpartum uterine infection involving the endometrium and frequently the deeper muscular layers of the uterus, typically occurring within 10–14 days postpartum and characterized by bacterial contamination of the uterus combined with impaired immune clearance. It is clinically manifested by an enlarged, atonic uterus and the presence of fetid, red-brown watery discharge progressing to viscous off-white purulent exudate, often accompanied by reduced milk yield, anorexia, fever, depression, and delayed uterine involution (Noakes, et al., 2018; Sahu et al., 2026a). In clinical endometritis, purulent vaginal discharge is observed after 21 days postpartum, while mucopurulent discharge may persist beyond 26 days postpartum; subclinical endometritis is diagnosed after 40 days postpartum in the absence of visible discharge but with ≥5% polymorphonuclear cells in endometrial cytology samples. Diagnosis is based on postpartum history, vaginal examination, uterine enlargement on rectal palpation, nature of discharge, and where required, cytological evaluation of endometrial samples (Sahu et al., 2026b).

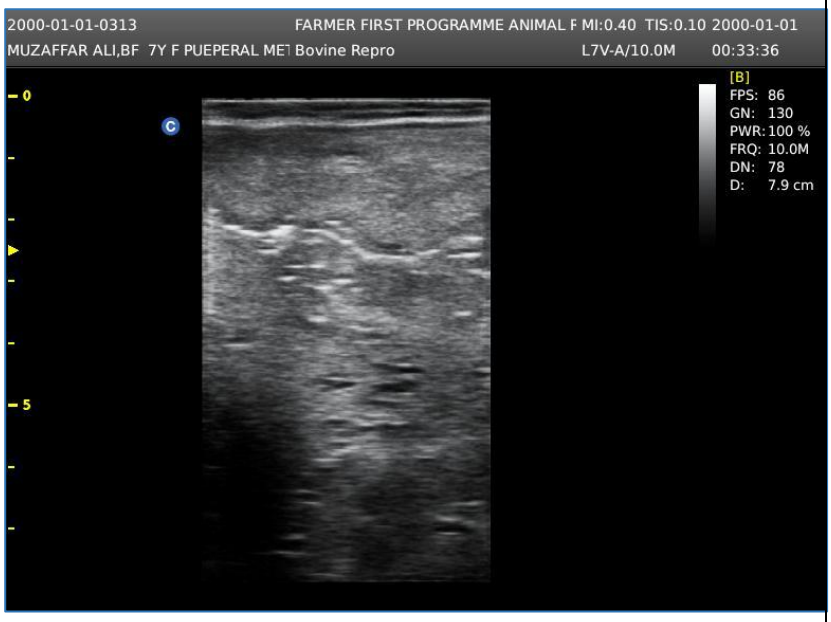

Fig. 3. Echogenic content and thickening of endometrium in buffalo

Treatment includes systemic antibiotic therapy such as penicillin, ceftiofur, or ceftriaxone in clinically affected cows, and intrauterine antibiotic therapy using agents like oxytetracycline or combinations such as levofloxacin with ornidazole or metronidazole where indicated. Hormonal therapy using prostaglandin F2α analogues such as cloprostenol (500 µg IM) or dinoprost (25 mg IM) may be administered to promote uterine involution and luteolysis, particularly when a functional corpus luteum is present. Combined hormonal and intrauterine antibiotic therapy (e.g., oxytetracycline intrauterine infusion with cloprostenol IM) may enhance therapeutic response. In selected cases, immunomodulatory approaches such as intrauterine E. coli lipopolysaccharide, autologous serum or plasma infusion, PMN extracts, oyster glycogen, or leukotriene B4 have been utilized to stimulate neutrophil migration and uterine defense mechanisms. Preventive measures include maintaining hygienic calving environments, minimizing dystocia and retained fetal membranes, ensuring adequate nutritional and mineral balance

during the transition period, and early postpartum monitoring for timely intervention.

3. Mastitis:

Mastitis is an inflammatory condition of the udder irrespective of cause, characterized by physical, chemical, and microbiological changes in the milk and pathological alterations in the glandular tissue, leading to changes in milk color, consistency, and the presence of leukocytes. It may occur in per-acute, acute, sub-acute, or chronic forms. Per-acute mastitis is severe and may endanger life, presenting with high fever (106–107°F), anorexia, respiratory distress, painful and swollen udder, cessation of milk secretion, and blood-stained exudate, commonly caused by Staphylococcus aureus, Streptococcus species, Escherichia coli, and Pseudomonas aeruginosa. Acute mastitis involves swelling of the udder with yellow or brown milk containing flakes or clots, while sub-acute mastitis shows variable milk changes with minimal systemic signs; chronic mastitis results in fibrosis, hard nodular quarters, atrophy of glandular tissue, and persistent abnormal milk. Diagnosis is based on clinical examination, changes in milk, culture and antibiotic sensitivity testing, and screening methods such as the California Mastitis Test (CMT) (Amalendu, 2003).

Treatment includes isolation of affected animals, frequent milking of the infected quarter, removal of secretions, and use of intra-mammary antibiotic preparations; systemic antibiotics are indicated when systemic signs are evident. Parenteral therapy may include penicillin, streptomycin, cloxacillin, ampicillin, cephalosporins, gentamicin, erythromycin, or tetracycline depending on sensitivity (Amalendu, 2003). Supportive therapy such as intravenous fluids containing glucose, antihistamines, corticosteroids, NSAIDs, and udder fomentation may be employed. In refractory cases, drying off the affected quarter or chemical drying using silver nitrate solution may be considered. Preventive management includes teat dipping with iodine or chlorhexidine solutions after milking, maintaining milking hygiene, proper milking order (healthy cows first), regular screening, dry cow therapy at drying off, clean bedding, adequate nutrition including vitamin A and trace minerals, and culling of chronic non-responsive cases.

4. Milk Fever:

Milk fever, or parturient hypocalcemia, is an acute metabolic disorder of dairy cows occurring most commonly within 24–72 hours after calving due to failure of calcium homeostasis at the onset of lactation. The sudden demand for calcium for colostrum and milk synthesis exceeds the cow's capacity for bone resorption and intestinal absorption, leading to hypocalcemia. Clinically, affected cows show progressive muscle weakness, depression, anorexia, ruminal stasis, cold ears and extremities, subnormal temperature, reduced defecation and urination, and varying degrees of recumbency. In advanced cases, cows may lie in lateral recumbency with the head turned toward the flank, exhibit dilated pupils, weak pulse, and decreased responsiveness. Diagnosis is based on recent calving history, characteristic signs of flaccid paralysis, and rapid response to calcium therapy; biochemical confirmation is made by detecting reduced

serum calcium levels (Chakrabarti, 2007).

Treatment should be initiated immediately once clinical signs are evident, as early intervention greatly improves prognosis. The primary therapy consists of calcium supplementation, preferably in the form of calcium gluconate or calcium borogluconate, which is considered the drug of choice and is commonly available as a 25% solution. The recommended dose is approximately 1 ml/kg body weight of 25% calcium borogluconate given as a single administration, with about 50% of the calculated dose delivered slowly by intravenous route and the remaining portion administered intramuscularly or subcutaneously; when larger volumes are required, the subcutaneous route is preferred (Chakrabarti, 2007). Alternatively, 10% calcium gluconate may be administered intravenously at a rate of 0.5–1.5 ml/kg/hour. Once the animal stabilizes, the preparation may be diluted with an equal volume of 0.9% sodium chloride and given subcutaneously twice daily if needed. After initial stabilization, oral calcium preparations such as calcium gluconate powder or calcium carbonate may be provided to maintain circulating calcium levels. Administration of 10–15 ml of 40% glucose and multivitamin supplementation may also be beneficial. Complications such as hyperthermia or hypoglycemia should be managed appropriately, and animals not responding to calcium therapy require reassessment. Supportive care is essential, including maintaining the animal in sternal recumbency to prevent bloat and aspiration and managing prolonged recumbency carefully to avoid muscle damage and pressure sores (Chakrabarti, 2007).

5. Ketosis:

Ketosis (acetonemia or hypoglycaemia) is a metabolic disorder characterized by relative or absolute deficiency of carbohydrates in hepatic cells, leading to increased beta-oxidation of fats and excessive production of ketone bodies, resulting in ketonemia, ketonuria, ketolactia, hypoglycaemia, and reduced liver glycogen content; the principal ketone bodies include acetoacetic acid, beta-hydroxybutyric acid, and their decarboxylation products such as acetone and isopropanol. Clinically, affected cows show reduced appetite, selective refusal of concentrates, progressive loss of body condition, decreased milk yield, dullness, dry feces, and a characteristic acetone odor in breath, milk, or urine; in nervous ketosis, signs such as hyperesthesia, abnormal licking, incoordination, bellowing, excitability, and even convulsions may be observed. Diagnosis is based on history of early lactation, reduced milk production, presence of ketone bodies in urine or milk, hypoglycaemia, and response to glucose therapy (Chakrabarti, 2007).

Treatment is primarily directed toward correction of hypoglycaemia and suppression of ketone body formation. Intravenous administration of 500–800 ml of 40–50% glucose (dextrose solution) is recommended as initial replacement therapy. To avoid repeated injections, oral hyperglycaemic agents such as glycerol (100 g twice daily for 2–3 days in adult cattle) or propylene glycol may be used as glucose

precursors; sodium propionate (100–200 g daily for 3 days) and lactates (initial 1 kg followed by 0.5 kg daily for 7 days) may also be administered due to their glucogenic effects, while sodium acetate (110–500 g daily) gives less satisfactory results (Chakrabarti, 2007). Hormonal therapy with glucocorticoids such as betamethasone or dexamethasone (up to 30 mg IM) is considered highly effective in field conditions, and triamcinolone may also be used; glucocorticoids reduce ketone body formation by enhancing glucose precursor availability in the liver. Prednisolone may be administered in larger animals (approximately 10 ml), and anabolic steroids such as durabolin, dianabol, or trenbolone have been reported to aid recovery. In recurrent cases, insulin therapy (approximately 200 IU in large animals) may be used to facilitate cellular glucose uptake, sometimes combined with glucocorticoids. Additional supportive treatments include chloral hydrate (initial 30 g orally followed by divided doses), vitamin B12, cysteamine, methionine, choline, and liver extract injections. Preventive measures focus on maintaining adequate energy intake, avoiding over-conditioning during gestation, ensuring proper concentrate-to-roughage balance, and preventing excessive negative energy balance during early lactation (Chakrabarti, 2007).

6. Left Displacement of Abomasum (LDA):

Left displacement of the abomasum (LDA) is a postpartum disorder in which the abomasum, normally positioned on the abdominal floor, becomes distended with gas and displaced between the rumen and the left abdominal wall, commonly occurring during the periparturient period. Predisposing factors include abomasal atony with increased gas production, concurrent metabolic diseases such as hypocalcemia, ketosis, mastitis, fatty liver, metritis, and retained placenta, rapid changes in abdominal topography around calving, pregnancy-related shifts in abdominal organs, and feeding of low-fiber, high-concentrate rations. Pathogenesis involves decreased abomasal motility and accumulation of ingesta and gas, allowing the loosely attached abomasum to move dorsally and become trapped on the left side. Clinically, affected cows exhibit intermittent anorexia, gradual decline in milk production, weight loss, abdominal discomfort with shifting weight between limbs, sluggish rumen motility, loose or scant feces, dehydration in later stages, and a characteristic high-pitched tympanic “ping” on simultaneous auscultation and percussion over the left abdominal wall. Diagnosis is based on history and clinical signs, confirmation of the typical left-sided ping, and ancillary procedures such as needle aspiration detecting abomasal fluid with acidic pH (Nandi et al., 2009).

Medical management aims to restore abomasal motility and correct underlying metabolic disturbances and may include administration of calcium, parasympathomimetic drugs, fluid therapy, oral stimulants, and dietary correction with increased fiber intake; rolling the cow in lateral recumbency may temporarily reposition the abomasum but has limited long-term success. Surgical correction is considered definitive, with procedures such as right flank omentopexy or right paramedian abomasopexy providing stabilization and repositioning of the abomasum; decompression of gas prior to repositioning is

recommended (Nandi et al., 2009). Early diagnosis and correction are essential to prevent complications such as torsion (volvulus), severe metabolic derangements, and reduced productive performance.

7. Laminitis:

Laminitis is a metabolic–vascular disorder of the claw characterized by inflammation and structural damage of the laminar corium, frequently observed during early lactation and closely associated with transition-period metabolic disturbances (Nandi et al., 2009). It commonly occurs postpartum due to sudden dietary shifts toward high-concentrate, low-fiber rations that predispose cows to subacute ruminal acidosis, endotoxin release, and systemic inflammatory responses. Negative energy balance, excessive body condition loss, hypocalcemia, and concurrent diseases such as metritis or mastitis further contribute to vascular disturbances within the claw. Pathophysiologically, accumulation of lactic acid and endotoxins in the rumen increases histamine release and inflammatory mediators, resulting in vasodilation, impaired microcirculation, and damage to the sensitive laminar tissues. This leads to weakening of the suspensory apparatus of the distal phalanx, hemorrhage in the sole, sole ulcers, white line disease, and chronic claw deformities. Clinically, laminitis may present in acute, subacute, or chronic forms. Acute laminitis is characterized by sudden onset lameness, reluctance to move, arched back, shifting of weight between limbs, warm claws, and increased digital pulse. Subacute cases are more common postpartum and manifest as mild lameness, reduced feed intake, and decreased milk yield. Chronic laminitis results in abnormal hoof growth, ridged hoof walls, sole hemorrhages, double soles, and recurrent ulceration, leading to persistent lameness and reduced reproductive performance. Diagnosis is based on history of recent calving, dietary management changes, clinical signs of lameness, hoof examination, and presence of sole hemorrhages or ulcers; chronic cases may show characteristic hoof deformities.

Treatment focuses on correcting the underlying metabolic cause and relieving inflammation. Immediate dietary correction is essential, increasing physically effective fiber and reducing rapidly fermentable carbohydrates. Anti-inflammatory therapy using non-steroidal anti-inflammatory drugs (NSAIDs) helps reduce pain and inflammation. Corrective hoof trimming is critical to remove diseased horn tissue and redistribute weight away from affected areas. In cases of sole ulcers, therapeutic hoof blocks may be applied to the healthy claw to relieve pressure. Supportive care includes providing soft, dry bedding, minimizing standing time, and ensuring comfortable flooring to reduce mechanical stress. In early stages, improvement of rumen health through buffering agents and gradual dietary transition can prevent progression. Preventive management during the transition period, including controlled concentrate feeding, adequate fiber intake, avoidance of overcrowding, and regular hoof trimming, is crucial to reduce incidence and maintain productivity and reproductive efficiency.

Conclusion and Future Prospects:

Advancement in transition cow management increasingly emphasizes precision-based prevention

rather than reactive therapy, incorporating metabolic profiling, rumination and activity sensors, early disease biomarkers, optimized DCAD strategies, and genomic selection to identify risk before clinical expression. Because postpartum disorders such as hypocalcemia, ketosis, metritis, mastitis, LDA, and laminitis arise from interconnected metabolic and inflammatory disturbances, their control requires an integrated approach combining balanced nutrition, mineral regulation, immune support, hygienic calving management, and systematic monitoring. Strengthening metabolic resilience and uterine recovery during this critical window ultimately enhances milk yield, reproductive efficiency, animal welfare, and long-term sustainability of dairy production systems.

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