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NUTRITION AND METABOLIC DISEASES IN DAIRY CATTLE- A REVIEW

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Abstract- Metabolic disorders are a key problem in the transition period of dairy cows as the cow suffers from negative energy balance due to low dry matter intake. Main challenge and problem appears to be adaptation of rumen at different physiological stages, which leads to onset of various metabolic diseases. Nutrition plays a pivotal role in preventing metabolic disorders post calving and through lactation. Metabolic disorders such as ketosis, fatty liver syndrome, hypocalcaemia etc can have a significant effect not only on a cow's lactation performance, but also on fertility performance. These types of problems tend to be associated with higher yielding animals, which are controllable by good feeding practice, both in lactation and during the dry period. So an understanding and day to day update of these metabolic diseases becomes essential and critical for successful venture of dairy farm.

Keywords- Metabolic disorders, nutrition, lactation, dry period.

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Introduction

Among domestic farm animals, the metabolic diseases achieve their greatest importance in dairy cows. The high producing dairy cows always verge on abnormality because the breeding and feeding of dairy cattle for high milk yield is etiologically related to the diseases of metabolism so common in these animals. In dairy cows, the incidence of metabolic diseases is highest in the period commencing at calving and extending until the peak of lactation is reached, and this susceptibility appears to be related to the extremely high turnover of fluids, salts and soluble organic materials during the early part of lactation [1].

The biological cycles of milk production and reproduction determine dairying profitability thus making management decisions dynamic and time-dependent. The dry period in cows looks apparently to be a resting phase between lactations, in reality considerable fetal growth, mammary tissue remodeling and high nutritional demands occur [2]. During past two decades, the average annual milk production per cow has increased significantly due to advancement of our knowledge and techniques in genetics, breeding, nutrition and management. As the production of milk has increased per cow, metabolic diseases have become more common because huge amount of nutrients are drained from the cows in milk, which is difficult to replenish back through feed dry matter intake, which is less and fixed for cattle especially in first three weeks of parturition[3]. Thus, proper nutrition and management of these high-producing cows become increasingly complex and critical.

The recognition of the importance of the period from late pregnancy until early lactation has led to the development of the concept of the transition period, which is commonly defined as the period from 3 weeks before to 3 weeks after calving [4]. Nutrient requirements of the fetus reach maximal levels three weeks prepartum, yet dry matter intake (DMI) decreases by 10 to 30%. Within three weeks of the onset of lactation, milk yield, milk proteins, fat and lactose increase rapidly and exceed feed intake [5]. Moreover, the diet of most dairy cows changes rapidly at calving from being mainly forage-based to concentrate-rich diets giving a challenge to rumen papilla which are adapted to high fiber diet during dry period. Postpartum milk production and the requisite nutritional adaptations induce a physiological state of negative energy balance (NEB). Thus, the conclusion reached by many is the existence of an overall antagonism between milk yield and reproduction [6].

Because the disorders mentioned above are interrelated and can occur as a complex in one animal or in a herd, herd health management strategies designed to prevent these diseases are urgently needed. So an understanding and day to day update of these metabolic diseases becomes essential and critical for successful venture of dairy farm.

Fatty liver

Fatty liver is a common metabolic disorder of dairy cows during transition period. Up to 65% of dairy cows are affected by moderate or severe fatty liver during early lactation [7]. Fatty liver or fat cow syndrome does not occur due to extra fat or extra feed in ration as the name implies, but in fact occurs when the cow cannot meet its energy demand in first four weeks of calving due to less dry matter intake and negative energy balance. It also occurs as a secondary disease of other production diseases that depress appetite or increase body fat mobilization. The clinical symptoms comprise depression, lack of appetite and weight loss, and the cows are weak and apathetic [8]. Most cows suffer from nonspecific clinical signs

including reduced rumen motility and decreased milk yield. Risk factors for fatty liver in dairy cows may be nutritional, managemental, genetic and usually cows with BCS above 4.5 are in high risk due to over conditioning in dry period [9]. In obese cows, lipolysis of adipose tissue is increased more during peripartal period than in cows with normal body condition. Obese cows have a greater decrease in feed intake around calving and, therefore, have a more severe negative energy balance [10]. During the negative energy balance body fat is mobilized into the bloodstream in the form of non-esterified fatty acids (NEFA) and severity of negative energy balance will be proportional to severity of fatty liver. NEFA are taken by the liver in proportion to their supply, but the liver does not have capacity sufficient to oxidize and use all amount of NEFA for energy. Therefore, cows are predisposed to accumulate NEFA as triglycerides within the liver when large amounts of NEFA are released from adipose tissue [11].

NEFA can be metabolized by many maternal and fetal tissues for energy production and fat synthesis. Circulating NEFA is a significant contributor to milk fat synthesis in the mammary gland postpartum and can contribute to almost half of milk fat. During neutral or positive energy balance when plasma NEFA are not elevated, NEFA contributes only minimally to milk fat synthesis[12]. Non-esterified fatty acids are also largely metabolized by hepatic tissue. Uptake of NEFA from plasma by the liver is proportional to the plasma concentration and the rate of blood flow and is about 25% of NEFA passing through hepatic circulation. During the transition to lactation and associated surge in plasma NEFA, rate of blood flow increases. This combination results in an increase uptake of NEFA into hepatic tissue in postpartum dairy cows [13].

The incidence of fatty liver is strongly associated with the incidence of especially ketosis and displaced abomasum because these disorders are related to severe negative energy balance [14]. In severe cases of fatty liver, milk production and feed intake are decreased but however fatty liver is often a reversible condition especially in early stages when liver can cope up with fatty infiltration[15]. Nutritionally it can be prevented by improving the metabolic state of cows in the peripartal period by supplying an extra source of blood glucose and by decreasing mobilization of NEFA from adipose tissue and are presented underneath in [Table 1].

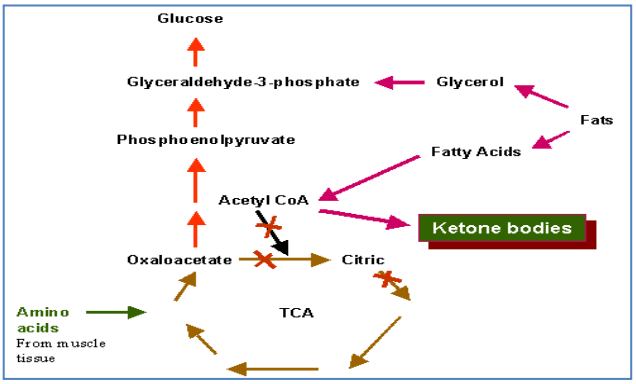
Table-1 *Preventatives for different levels of fatty liver in lactating dairy cows*

Preventative	Effect	Reference
Glycerol	+/0	DeFrain [16]
Insulin	+/0	Hayirli [17]et al., 2002
Ammonium propionate	+	Furll and Leidel[18], 2002
Choline	0	Pinotti [19]et al., 2003
Glucagon	++	Nafikov [20]et al., 2002
Insulin	+/0	Hayirli [17]et al., 2002
Monensin	++	Duffield [21]et al., 2003
Propylene glycol (bolus dose)	++	Christensen [22]et al., 1997
Sodium borate	+	Basoglu [23]et al., 2002

[1]The number of + indicates slight, moderate, or strong effectiveness as preventative or treatment for fatty liver; 0 indicates no beneficial effect.

Ketosis

Ketosis is the imbalanced use of body fat by a cow common in early lactation and is characterized by hypoglycemia and hyperketonemia. The three main ketone bodies fond are acetoacetate, beta-hydroxybutyrate and acetone. This disorder occurs both subclinically and clinically with high prevalence of subclinical ketosis in first 2 weeks of lactation due to negative energy balance and less dry matter intake [14]. Ketosis occurs primarily when a cow is in negative energy balance immediately after calving. To support the energy demands, the body mobilizes fat reserves resulting in release of NEFA in blood.



Excessive release of NEFA from fat depots may overwhelm the capacity of the liver to use the fatty acids as a source of energy and converted to ketone bodies [24]. Feed intake is naturally depressed by about 20% around the time of calving. Simultaneous with reduced feed intake, plasma NEFA and liver triglyceride concentrations are also increase. All factors exacerbating negative energy balance and depression of feed intake around calving increase the risk of ketosis, especially in over conditioned cows. Negative energy balance, high blood levels of NEFA and ketosis reduce the response capacity of white blood cells so that invading bacteria can compete the immune system of the cow [25]. Hypoglycemia alone is not likely to exacerbate periparturient immunosuppression, but hyperketonemia appears to have multiple negative effects on immune functions. Impaired immune functions around calving make cow more prone to infectious diseases. In addition, prolonged and pronounced negative energy balance in early lactation adversely affects reproductive performance of cows and increases the risk of other metabolic diseases [26].

There are four main types of ketosis: primary ketosis, secondary ketosis, butyric acid ketosis and underfeeding ketosis [9]. Classical or primary ketosis (also called production ketosis) generally occurs in cows during the first 2 to 4 weeks of lactation [27]. It develops when the glucose demand exceeds the gluconeogenesis capacity of the liver resulting in increased ketogenesis, and thus in high concentrations of ketone bodies in blood, milk, and urine. The disorder is mainly seen in over conditioned cows. Secondary ketosis results from another disease that depresses feed intake and increases body fat mobilization [28]. Silage with high butyrate concentrations results in an increased concentration of beta-hydroxybutyrate in the blood and such silage is consumed in lower amount than normal silage leading to butyric acid ketosis. Underfeeding ketosis occurs in cows that are fed insufficiently and to obtain the deficit energy, fat is mobilized leading ultimately to ketosis. Underfed animals are deficient in glucogenic precursors and this condition then leads to increased ketogenesis [9]. Most accepted ketosis treatments attempt to increase blood sugar levels is usually giving about 500 ml of a 50 percent glucose. As a result, most veterinarians recommend intravenous injection of glucose with the incorporation of insulin as a part of the therapy[29]. Also, some veterinarians supplement corticosteroids for a few days following treatment to boost blood glucose levels. Thin cows (BCS<3) or over conditioned cows (BCS>4) should be avoided. Compounds like niacin and propylene glycol decrease fatty acid mobilization from adipose tissue have been useful to prevent of fatty liver and ketosis [30].

Rumen acidosis

Ruminal acidosis is an important nutritional metabolic disorder common in field especially in high yielding cows with high grain ration [31]and takes a huge toll of profit from dairy enterprise often unnoticed by farmer. It costs double loss to farmer as productivity and health of dairy animal is compromised along with feed loss as dairy farmer is feeding costly grain ration of which considerable amount passes undigested in feces. Ruminal acidosis when occurs in chronic form is called as Sub-acute ruminal acidosis (SARA). Sub-acute or sub-clinical ruminal acidosis (SARA) is considered to be one of the major threats to the welfare of

lactating dairy cows and may affect up to 20% of cattle in early to mid lactation [26]. Subclinical rumen acidosis is defined as a condition where rumen fluid pH is below 6 while acute rumen acidosis is when rumen pH is below 5.5 associated with rumen motility that is weak or ceased [32]. Often Ruminal acidosis and SARA are confused with each other but they actually vary in their occurrence as ruminal acidosis classically occurs when an animal consumes excess of grain or due to lactate accumulation while SARA is due to excessive VFA production that exceeds ability of rumen papillae to absorb them. This reduced ability of VFA absorbance is commonly seen at calving when dry cow is switched to grain ration (in dry period rumen papilla are adapted to high fiber feed and are smaller and less distinct in size) because neither rumen papilla nor ruminal microbes are adapted to high grain ration [33]. During dry period the ruminal microbial population is set up to digest high fiber feed and starch digesters like *Megasphera elesdeni* and *Bacteriodes ruminicola* are not dominant which leads to accumulation of lactic acid and ultimately fall of rumen pH leading to acidosis [34]. Dairy cows experiencing an episode of SARA will reduce their feed intake in order to reduce the acid load in their rumen. Cows will start eating again when ruminal pH is above 5.6. This results in erratic feed intake that often goes unnoticed in individual cows experiencing SARA, particularly in large dairy herds where cows are housed and fed in groups. Other clinical signs often observed during SARA may include reduced rumination (cud-chewing), mild diarrhoea, foamy feces containing gas bubbles and appearance of undigested grain [35]. Reduced ruminal efficiency, liver and lung abscesses, and laminitis all can be related to SARA. One of the way for field diagnosis of SARA is to see the hair around the skin of rectum i.e. perianal region, hairless shiny skin will indicate SARA due to chronic acid damage to skin. Low rumen pH can cause rumenitis, metabolic acidosis, lameness, hepatic abscesses formation, pneumonia, and even death [36]. It can be prevented by balancing the diet for starch and effective fibre. Also sudden changes of feed and slug feeding of grain and/or molasses should be avoided. Roughages should be provided with or immediately after feeding grain/molasses. Feed buffers such as sodium bicarbonate or ionophores also counteract acidosis [33,37].

Laminitis

Laminitis is a pathophysiologic disturbance of the microstructure of the dermis or corium of the claw. Due to its prevalence, lameness is probably the major animal welfare issue affecting the species[38]. The highest incidence of lameness is seen in high-production, intensively managed dairy cattle. It is probably equal in importance to reproductive inefficiency to which it is now known to be closely related causing huge economic losses. Inflammation is primarily associated with a dysfunction of the digital vasculatory system that results in hypoxia and malnutrition of the sensitive laminar structure in the hoof wall[39]. The etiology of the circulatory disturbance is not fully understood and there are some possible explanations related to nutrition. Due to mechanical stretching of the attachment between the inner and outer laminar structures of the hoof wall, which has been affected by inflammation, the claw bone can rotate and sink inside the hoof. Depending on the severity of the initial lesion, the mobility of the claw bone inside the capsule and counter pressure on the sole from hard floors, the sole corium can suffer contusions and secondary lesions of the sole area can develop [40].

Laminitis has been associated with nutrition, and specifically with ruminal acidosis either in its acute or sub-acute (SARA) form. Exact relationship between laminitis and SARA is not known [41]. One of the theories states that damage of the ruminal epithelium induced by acidosis allows absorption of histamine and endotoxins into the blood which comes from the lysis of gram negative bacteria in rumen due to low pH. Histamine and endotoxins cause vasoconstriction and inflammation of lamella of hoof causing laminitis [33]. Cows fed the higher level of crude protein may have increased incidence and duration of lameness. It is considered that products of degradation of protein excess in the rumen may be the causative agents for lameness. It has also been shown that nutritional supplements such as biotin and zinc can help reduce lameness through improving claw horn quality [42]. Biotin is essential for two major metabolic pathways in keratinisation, keratin protein synthesis and lipogenesis. Other vitamins also may play important roles in maintaining claw integrity, including vitamin A and vitamin E.

Other trace minerals that impact claw condition include iodine, selenium, copper, manganese and Cobalt [43]. It can be prevented by feeding for stable rumen conditions i.e. addition of buffers, feeding of good quality roughages, etc. Foot baths containing 2–5% copper sulphate particularly in wet conditions is beneficial for lame animals[33]. Nutritional supplements like biotin and zinc (Zn) can help reduce lameness through improving claw horn quality [42]. Biotin is essential for two major metabolic pathways i.e. in keratin protein synthesis and lipogenesis. Other vitamins like vitamin A and vitamin E also may play important roles in maintaining claw integrity. Zinc, as a component of many enzyme systems, has a role in formation of structural proteins necessary for keratinazation [42] and help to improves claw integrity by speeding epithelial tissue repair. Other trace minerals that impact claw condition include iodine (I), selenium (Se), copper (Cu), manganese (Mn) and Co.

Displaced abomasum

The abomasum normally lies on the floor of the abdomen but can become filled with gas and rise to the top of the abdomen, when it is said to be 'displaced'. Dislocation can be to the left (left-displaced abomasum) or to the right (right displaced abomasum) in the stomach in relation to the normal placing [44]. Approximately 80-90% of the incidences are left-displaced abomasums. It occurs when the abomasum moves to the left of its normal position, becoming trapped between the rumen and the left abdominal wall. In some cases it will also twist. Stretching results in constriction of the entrance to and exit from the abomasum and it fills with gas. LDA is suspected when tapping the lower left side of the cow produces a hollow 'ping', indicating the presence of gas. The disease is most frequent in high producing cows in early lactation and most of the cases are seen in the first 4 weeks postpartum [45].

Nutrition is a major risk factor in the etiology of displaced abomasums, but the precise cause is still unclear. Low feed intake and increased negative energy balance in prepartum have been found common cause to the increased risk of displaced abomasums in cows [9]. Cows fed high concentrate diets in early lactation and diets with smaller particle size are also at increased risk of displaced abomasums [37] along with sudden changes in the diet. Feeds like silage increase the incidence of disease as compared to those fed hay, probably because silage is often finely chopped and risk could be minimized if cow eats a kilogram of straw daily [9]. Volatile fatty acids (VFA) in the abomasum also have been reported to reduce abomasal motility. Effects of VFA on motility may be exacerbated by low ruminal absorption of VFA during the transition period [45]and hypocalcaemia also may playing a role. Due to reduced feed consumption, slow contractions and inadequate filling of the rumen, which therefore does not reach the ventral abdominal wall, empty space appears for movement of abomasum. Then usually more fatty acids escape absorption in the rumen and reach the abomasum. Those VFA, along with hypocalcaemia and accumulated gases, contribute to reducing abomasum contractility and development of atony [46]. Thus, inadequate feed consumption and insufficient rumen fill along with reduced motility and strength of abomasal contractions together contribute to the onset displaced abomasums [46].

The incidence of this disease also increases if low concentrate ration in dry period is given because rumen papillae have low absorptive capacity and rumen microbes are also not adapted to high grain ration. Thus too rapid increase of concentrates after (especially ration high in non-structural carbohydrates) calving may reduce roughage intake and potentially increase the risk of displaced abomasums [9]. BHBA and aspartate aminotransferase (ASAT) activity in the blood may be used to predict the development of LDA [47]. It can be prevented by maintaining the forage to concentrate ratio of the diet fed in late gestation and early lactation [48]. TMR that are easily sorted by cows may affect the ratio of forage to concentrate of total feed consumed by an individual cow and will contribute to DA [45]. When a TMR is not fed, grain intake after calving should be increased slowly (0.25 kg/day) until peak grain intake is achieved[48].

Parturient hypocalcaemia

Milk fever is a metabolic disturbance usually of dairy cows, characterized by reduced blood calcium levels and is most common in the first few days of lactation (usually within 72 hours) when the demand of calcium for milk production exceeds the body's potential to mobilize calcium reserves[49]. Fever is a misnomer, as body temperature during the disease is usually below normal. Low blood calcium level interferes with muscle function throughout the body, as contraction of muscle fibers is dependent on it causing general weakness, depression and death. It is more common in older dairy cows, which have reduced ability to mobilize calcium from bone and in high milk producing breeds in later lactation cycle due to exhausted reserves [1].

The lack of Ca in the diet does not lead to any changes in health or production for a long time because being compensated by body Ca reserves in the skeleton by parathyroid hormone but its temporary less functioning during dry period fails calcium mobilization as parathyroid passes through a period of inactivity. More often hypocalcaemia exists in a subclinical form with very few or no symptoms (subclinical hypocalcaemia) incurring greater losses. Hypocalcaemia was defined as the content of total Ca in blood below 2 mmol/L with or without clinical signs of paresis [8], which is approximately equivalent to 1 mmol/L of ionized Ca [50]. The disease is characterized by pronounced hypocalcaemia which is accompanied by hypophosphataemia in most cases, and by less serious hypermagnesaemia [51].

Generally, incidence of milk fever occurs from 0–10%, but some time it may exceed 25% for calving cows. In some studies conducted on milk fever the cows calving incidence has reached 80% [52]. Mostly dietary factors, which increase the incidence, are providing dry cows a high daily intake of calcium, unfavorable ratio of Ca/P and diets high in phosphorus at the onset of lactation also increase the incidence of milk fever[8]. High dietary level of phosphorus decreases the production of 1,25 dihydroxy vitamin D_3 and thus reduces the intestinal calcium absorption mechanisms [53]. Pre-partum diets high in cations like sodium and potassium (particularly potassium) are associated with an increased incidence of milk fever while diets of high in anion, especially chlorides and sulfides are associated with decreased incidence of the disease. The addition of anions to the diet of dairy cows prior to parturition effectively reduced the incidence of milk fever by inducing a metabolic acidosis, which keeps parathyroid hormone in active state thus reducing incidence of fever milk [54].

The most predisposed breeds for milk fever are Jersey and Guernsey, followed by Holstein and Brown Swiss. In the case of Jerseys, possible reasons are considered to be the higher milk production per unit of body weight [55]and a higher content of Ca in the colostrum [27]. In addition, the number of receptors in the intestine is about 15% lower in Jerseys than in Holstein's milk fever due to well-timed treatment and relatively inexpensive; the disease was not considered as a factor of economic importance in dairy production for a long time [8]. However, later findings confirmed its close relationship with other metabolic disorders like dystocia, retained placenta, metritis, uterine prolapse, ketosis, mastitis and displaced abomasum. Due to increased susceptibility to other health disorders and possible complications, the production life of cows that experienced MF was reduced by an average of 3-4 years [56].

Subclinical hypocalcaemia influences in the same manner as the clinic one, but to a lesser extent. As it is more common in the herd, adverse effects of subclinical hypocalcaemia on herd economy can be equal or even greater than the effects of Milk Fever due to its broader influence on feed intake, secondary disease conditions, and milk production during early lactation[53]. Nutritionally it can be corrected by maintaining the dietary cation-anion difference in the diet (adding excess anions) during the late prepartum and early postpartum period. It is believed to enhance calcium resorption from bone and absorption from GI tract [57]. Another strategy is to reduce potassium content of diet. Addition of vitamin D_3 and its metabolites is also effective in preventing parturient paresis [58].

Hypomagnesaemia

Magnesium (Mg) homeostasis is not under direct hormonal control but is mainly determined by absorption from the GI tract, excretion through kidneys and varying requirements of the body for pregnancy, lactation, and growth. If Mg secretion in milk (0.15 g of Mg removed with each liter of milk) and its endogenous losses exceeds absorption from the rumen hypomagnesaemia occurs [59]. Ruminants absorb Mg less efficiently than non-ruminants (35% vs 70% of intake).

Rumen is the primary site of absorption of Mg and low pH enhances Mg absorption while high pH above 6.5 decreases its solubility and rumen absorption. Grass tetany generally occurs when the dietary intake of total Mg is not particularly low, but factors, which increase the animal's requirement for Mg or reduce the availability of dietary Mg are present [60]. Lush green pastures are rich in potassium, sodium and NPN compounds. Sodium and potassium have positive DCAD thus alkalize the rumen environment resulting in decreased Mg absorbance. Further degradation of NPN compounds in lush green pastures exceed the capacity of rumen microbes to incorporate in microbial protein resulting in ammonia build up which increases the rumen ph resulting in decreased Mg absorption [61, 62].

Clinical hypomagnesaemia in cows with plasma Mg concentrations below 0,4 mmol/L is manifested as grass tetany [63]. The Mg concentrations in forage and subsequently in the blood of cattle are influenced strongly by high amounts of fertilizer K and, to some extent, fertilizer N. Absorption of Mg by plants is reduced by high levels of K in the soil [60]. Mg deficiency results in reduced appetite, which decreases total nutrient intake [62], thus chronic hypomagnesaemia results in reduced feed intake and milk production. It may progress quickly into acute hypomagnesaemia, which terminates in convulsions, coma and death. One-third of animals with clinical symptoms die [60].

Marginal form of hypomagnesaemia (Mg <0,85 mmol/L) also significantly reduces the mobilization of Ca from the skeleton around calving [37]due to which hypomagnesaemia becomes a risk factor for milk fever through inhibition of Ca mobilization by interrupting PTH secretion [64]. If one or more cases of grass tetany occurred in a herd, immediate consideration should be given to provide magnesium supplements (magnesium salts in water, magnesium licks, magnesium capsules, etc.) or shifting at-risk animals to new paddock containing more legumes, mature herbage [48]. Additional 10-15 g of Mg into each pregnant cow and 30 g into each lactating cow per day usually prevents hypomagnesemic tetany. The biggest problem in getting the extra Mg into the animal is because most of the magnesium salts are unpalatable and least soluble. Magnesium is readily acceptable in grain concentrates and inclusion of 60 g of magnesium oxide into just 0.5-1 kg of grain will be effective[59].

Retained placenta

Placenta is an essential organ for prenatal transfer of nutrients and oxygen from the dam to the fetus. It normally drops within short time post partum. If the placenta is not expelled within certain time (12 h post calving) is defined as being retained placenta (RP) and it creates a number of problems like pulling of microorganisms into the uterus causing inflammation, fever, weight loss, decreased milk yield, longer calving intervals and may result in an open cow during the next year and if the infection is so bad the animal may actually die. RP causes great economic losses, mainly due to decreased milk yield and calf crop[65].

Cows in a greater negative energy balance prepartum are 80% more will likely suffer from RP [66] and develop less immune response. Thus, higher energy consumption during the last weeks of the dry period and transition period management reduces the risk to great extend[55]. Inadequate antioxidant status or "oxidative stress" of the cow leads to poor functioning of immune system and increased risk of retained placenta. Se, vitamin E and beta-carotene are important dietary antioxidants whose low level increase incidence of RP [37]. Blood lymphocyte proliferation was higher in cows supplemented with beta-carotene, and phagocytic activity of blood neutrophils was enhanced as well as intracellular killing by blood neutrophils. Therefore, dietary beta-carotene can enhance peripartum host defense mechanisms by enhancing lymphocyte and phagocyte function [67]. In hypocalcaemia, muscles become weak and there is absence of uterine contractions in hypocalcemic animals does not contribute to the expulsion of fetal membranes [46]. Cows with retained placenta were 3 times more likely to develop mastitis than animals without retained placenta [11]. Supplementation of

diets with antioxidants (Vit. E and Selenium) to meet the requirements is crucial during the periparturient period. When the diet contained at least 0.12 mg/kg of Se and 1000 IU of dietary vitamin E per cow/day, the incidence of RP is lower [48].

Use of blood and milk analyses to evaluate nutritional and disease status

Recognition of subclinical diseases is difficult, the condition may be confirmed by analyzing blood, milk or sometimes urine [9]. Blood and milk analyses as tools to evaluate nutritional and disease status of animals in heard help making decisions for improvement of nutritional strategies and production management. Metabolic profile testing (MPT) refers to the use of a battery of tests for the diagnosis of subclinical nutritional and metabolic disease in dairy cows on a herd basis. Blood samples from 5-7 animals per production group are tested for various analytes [68]. MPT is most useful in the diagnosis and management of periparturient disease. Specific nutrient imbalances, either deficiency or excess, in the diet of late-gestation cows have been related to increased prevalence of milk fever, hypomagnesemia, retained placentas, downer cow syndrome, ketosis and displaced abomasum are present in [Table-2] [69].

Table-2 *Nutrient imbalances (deficiency or excess) and their associated metabolic and reproductive disorders*

Potential disease process	Nutrient status		Associated disease
	Deficiency of:	Excess of:	
Milk fever	Ca, Mg, Protein	Ca, P, Na, K, Vit.D.	RP,DYST,MAST,KET
Hypomagnesima	Mg	K, protein	M.F, Tetany
Ketosis	Energy, protein		MF,PR,FCS,LDA
Displaced Abomasum	Fiber, energy	Energy(grain)	MET,MAST,MF,KET
Downer cow syndrome	P,K	Protein, K	MF,FCS
Metritis	Ca, Co, vit. D, Se	Energy	RP, FCS
Retained placenta	Se, vit. E, vit. A, Cu, I, P, Protein, energy	Energy, K	FCS,MF,KET

FCS = fat cow syndrome, KET = ketosis, LDA = left-displaced abomasum, MAST = mastitis, MET= delayed uterine involution/metritis, MF = milk fever, RP = retained placenta

Summing up nutritional strategies and corrections to reduce metabolic diseases incidences

Although metabolic disorders are not easily categorized as to their cause, nutritional strategies have been developed to help prevent many of these disorders [70]. Problems in application of the strategies in the field still exist and their limitations with possible negative consequences on milk production economy in some circumstances. Strategies are based on major factors directly or indirectly increasing the risk of diseases such as excessive fat mobilization during transition period, over conditioning at calving, low nutrient intake and environmental stress [9]. Strategies are expected to reduce morbidity and improve reproduction and production at same time. To prevent metabolic disorders in the periparturient period nutritional strategies must start before calving. According to a survey conducted by Curtis [71] consumption of nutrients before calving was directly related to the occurrence of metabolic disorders and directly or indirectly to the occurrence of reproductive disorders after calving [72]. As they appear to be interrelated consequently strategies to reduce one disease can help preventing others like strategies to reduce liver triglyceride accumulation at calving may decrease incidence of ketosis, etc.

Three basic physiological functions must be maintained during the periparturient period if disease is to be avoided: adaptation of the rumen and its microbes to lactation diets that are high in energy density, maintenance of a strong immune system and maintenance of normal calcium metabolism[46]. Whenever one or more of these functions are impaired, the incidence of both metabolic and infectious diseases increases.

Stability of rumen key to Success

Fully adapting the rumen flora to a high starch diet that will be fed after parturition requires about 3 to 4 weeks, and full development of rumen papillae requires about 5 weeks of concentrate feeding [46]. Thus, it becomes important to start increasing concentrates in the diet 3-4 weeks before calving and continue during first 1-2 weeks after calving to fully adapt rumen to lactation diet. If fresh cow is abruptly switched to a high starch lactation diet, the risk of developing rumen acidosis exists because the rumen and rumen microbes are adjusted to digest high fiber diet in dry period. When there is sudden increase in carbohydrates especially if the concentrate mix is rich in non structural carbohydrates leads to rapid lactate production and accumulation [33,26]. Feeding TMR, adding buffering agents such as Nabi carbonate or alkalinizing agents such as Mg-oxide are added to high concentrate ration to reduce the risk of acidosis [69].Na-bicarbonate should be supplemented particularly to corn silage-based diets at the rate of approximately 0,8 to 1% of DM [33].

A highly significant relationship between forage neutral detergent fiber (NDF) content in the diet and ruminal pH exists. NRC [72]recommended 19% of forage NDF a absolute minimum when formulating rations in the field. A system of "physically effective" NDF (peNDF) relates the ability of a feedstuff to stimulate chewing (cud) which stimulates more saliva due to chewing which acts as natural buffer for rumen due presence of high bicarbonate in it. The peNDF of a feed is the product of its physically effective fiber (pef) and NDF content. The diet should contain about 22% peNDF to maintain ruminal pH of 6,0 [33].Gradual adjustment of the rumen to a high concentrate diet can help avoiding serious drop in feed intake around calving and minimize body fat mobilization in early lactation as the microbes and rumen papilla get sufficient adaptation time. The plasma NEFA concentration is negatively correlated with DMI and depression in feed intake around the time of calving was largely responsible for fatty liver development [69]. Therefore, all nutritional measures that prevent drop in DMI before parturition may be useful in prevention of fatty liver. However, as the dry matter intake in transition is destined to be low due to less available space for rumen and hormonal effect one may increase nutrient density of ration. Increased energy and nutrient density of the diet may assure maintenance of the same intake of nutrients and energy despite lower DMI around calving, and decrease rate of lipid mobilization. Increase in nutrient density during the last 2–3 weeks prepartum by increasing concentrates in the ration has been referred to as "close-up" diet [9]and are kept rich in non-structural carbohydrates. According to the recommendations, NRC [72] energy content of the pre-calving diet should be from 5.2 MJ/kg of DM during the "far-off" dry period, to 6.8 MJ/kg of DM for the 3 weeks before parturition.

Laminitis is other important metabolic disorder especially in high yielders due to feeding of high grain ration with high non-structural carbohydrates influencing hoof health [33]. It is very evident that feeding diets that cause drop in rumen pH will result in increase in laminitis cases. To minimize the drop in rumen pH diet should contain at least 19% acid detergent fiber (ADF)or 25% total NDF, with non-fiber carbohydrate (NFC) between 35 and 40%(not exceeding 40%) or non-structural carbohydrate (NSC) levels between 30 to 35%, and minimize abrupt changes of ration dairy cow [9]. Increased energy content of the diet fed during the prepartum

period is associated with decreased incidence of displaced abomasum and for cows feeding on silage the risk can be almost eliminated if every cow eats 1 kg of straw daily [37].

Conflict of Interest: None declared

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