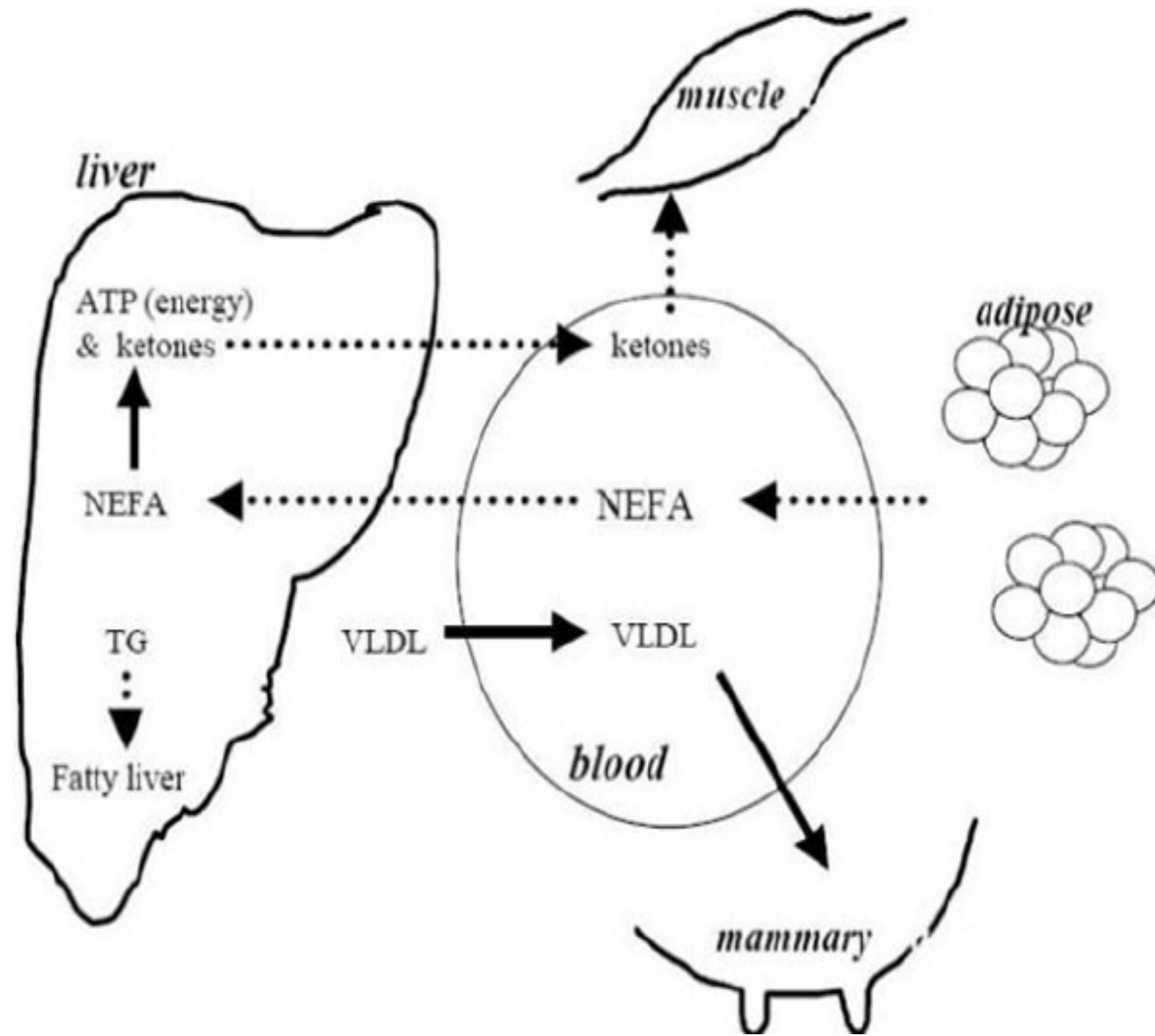


# Fat Cow Syndrome (Lipid mobilization syndrome)

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# Definition

It is a multifactorial condition occurring in dairy cows following parturition characterized by:

- ❖ progressive depression
- ❖ failure to respond to treatment of other predisposing diseases.

# Causes

- (1) Mobilization of excessive quantities of fat from body depots to the liver.
- (2) It occurs either because of :
  - a deprivation of feed in fat beef cattle and those **bearing twins**
  - a sudden demand of energy in postpartum period in well-conditioned cows.

# Clinical signs

- It occurs in the postpartum period. Most affected cows are either obese or very well-conditioned with a long amount of omental and subcutaneous fat.
- (1) Depression, anorexia, weight loss and weakness that can lead to recumbency.
  - (2) Decreased rumen motility and decreased milk production.
  - (3) Other signs vary and are related to other concurrent diseases such as metritis, retained fetal membranes, mastitis, parturient paresis and displaced abomasum.

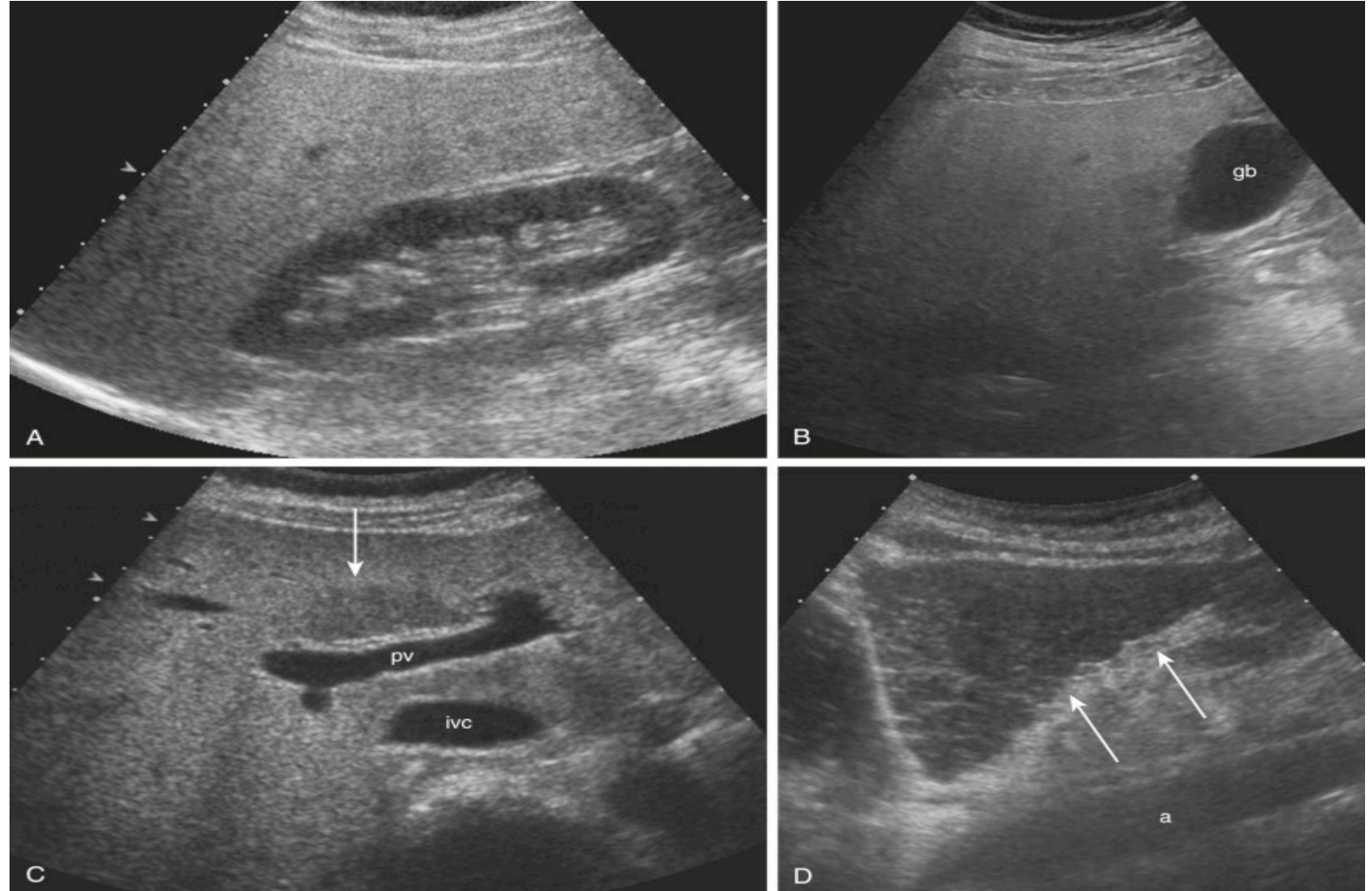


## **Clinical pathology**

- (1) Hepatic lipidosis but serum fatty acids (FFAs) will be increased.
- (2) Decrease in triglycerides, cholesterol, albumin, magnesium and insulin.
- (3) Serum enzymes: significant increases occur in the serum' activities of lactic dehydrogenase (LDH), aspartate aminotransferase (AST), alkaline phosphatase (ALP) and acid phosphatase (ACD).
- (4) Blood ketones: in severely ketotic cows there is a positive correlation between blood ketone body concentrations and the degree of fatty liver.
- (5) Liver biopsy and analysis: liver biopsy can be used to determine the severity of the fatty liver and the concentration of triglycerides.



(6) Ultrasonography of the liver: to evaluate fatty infiltration in dairy cattle.



(7) Hemogram: indicates leukopenia, neutropenia and lymphopenia.

(8) Necropsy findings: In severe fatal cases, the liver is grossly enlarged, pale yellow, friable and greasy.

(9) The histological change includes fatty cysts, enlarged hepatocytes and compression of hepatic sinusoids.

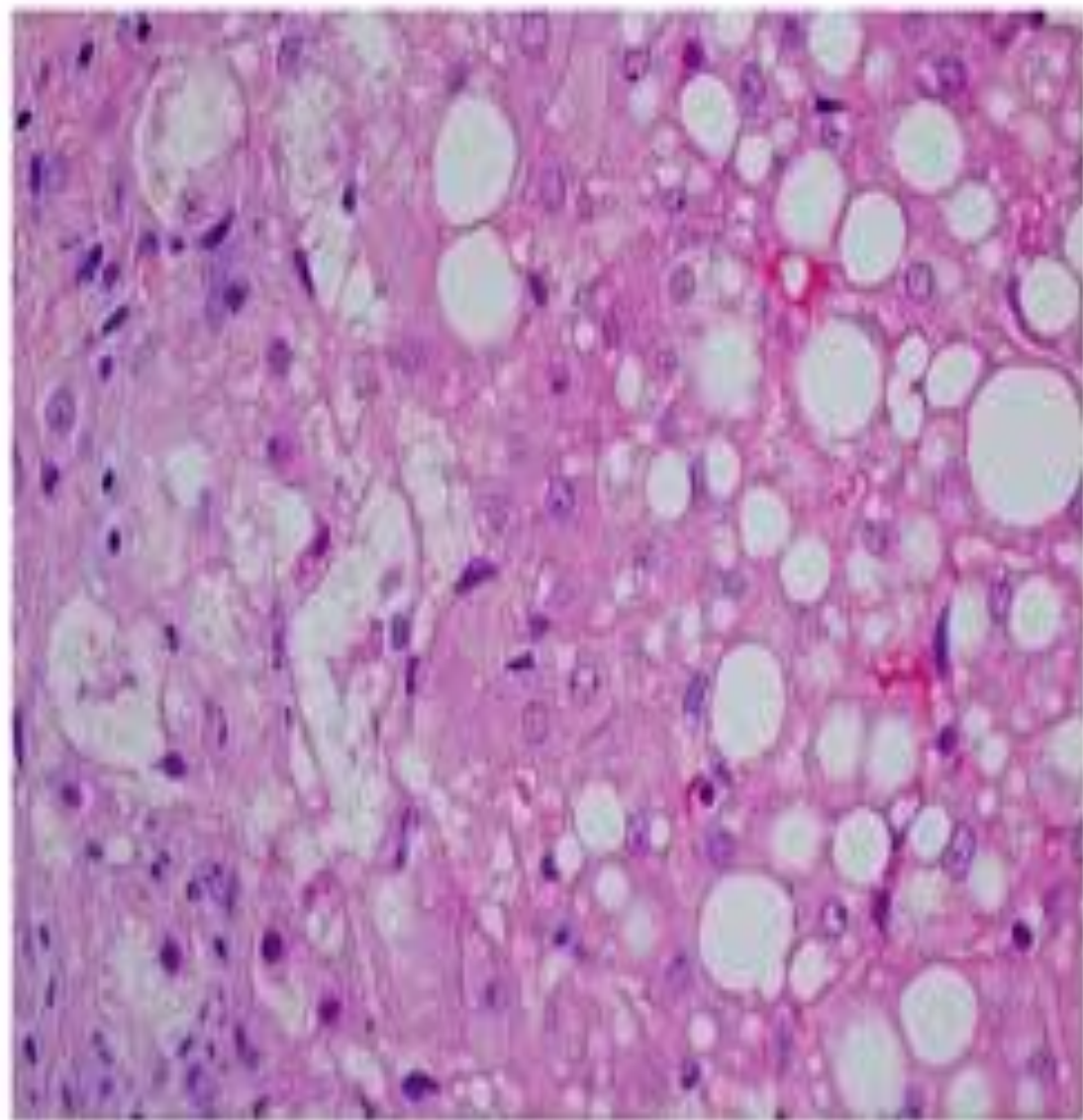




**Healthy Liver**



**Disease Liver**



# Differential diagnosis

It must be differentiated from those diseases which occur commonly immediately following parturition.

(1) **Left-side displacement of the abomasum**: it results in a secondary ketosis, and inappetance.

(2) **Retained placenta and metritis**: they may be accompanied by fever, inappetence to anorexia, ruminal atony and a foul smelling vaginal discharge.

(3) **Primary ketosis**: it occurs immediately after parturition or within several days. Inappetence, rumen stasis, ketonuria and a good response to glucose and propylene glycol.

# Prognosis

- The prognosis for severe fatty liver is unfavorable and there is no specific therapy.



# Treatment

- (1) Continuous IV infusion of glucose and electrolytes.
- (2) Intra-ruminal administration of rumen juice (5-10 L) from normal cows to stimulate the appetite of affected cows.
- (3) Corticosteroids such as dexamethasone at a dose of 20 mg every second day until recovery.
- (4) Propylene glycol given orally promotes gluconeogenesis.
- (5) Insulin (zinc protamine) at 200-300 pSC twice daily promote the peripheral utilization of glucose.
- (6) Injection of vitamin E and selenium.
- (7) Injection of choline chloride (25 g in 250 ml of sterile saline) given S/C.



## Control

- (1) Prevent pregnant cattle from becoming fat during the last third of pregnancy particularly during dry period.**
- (2) Metabolic profiles may be used as a means of assessing energy status of the animal.
- (3) Treatment of all diseases which occur immediately after parturition.
- (4) The use of propylene glycol will promote gluconeogenesis and minimize the mobilization of a depot fat.

