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CONGENITAL PAUNCH CALF SYNDROME IN ROMAGNOLA CATTLE

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In the last decade genetic diseases have become a matter of considerable concern for the Romagnola breeders, with Spastic Paresis being the most commonly referred disorder (estimated prevalence of 0.6%).

We describe here a new congenital and possibly inherited defect observed in 12 Romagnola calves (10 were stillborn, two lived only some hours) admitted to our Department. Breeders seem to be finding this problem particularly worrisome. We have decided to call this defect Congenital "Paunch Calf" Syndrome because of the main clinical feature and because this is the name farmers use to describe the affected animals.

All the calves showed an enlarged and floating abdomen, denoting a considerable abdominal effusion. Moreover they all had facial deformities characterized by shortened and flattened face and in some cases by enlarged head. A disproportionate shortness of the limbs (rhizomelia) was evident in one case. Cleft palate was evident in four calves.

At necropsy all but one animal had marked subcutaneous oedema, especially in the ventral part of the abdominal wall. Different quantities of ascites fluid (in some cases up to 10 liters) were present in the abdominal cavity. The liquid ranged from yellow to red, with different grades of turbidity. The liver presented a moderate to severe diffuse fibrosis. It was moderately tough and enlarged with irregular and enhanced lobular pattern. One or more cysts, with serous or reddish fluid content, were observed on the peritoneal surface of the left lobe and/or of the hepatic hilus. On surface section, the parenchyma was irregularly separated by slight fibrous bands. Diffuse ectasia of the intrahepatic veins was also detected. Representative samples from the liver were collected and processed for histological examination. Five micron sections were stained with haematoxylin and eosin (HE), Masson-trichrome stain, Gomori and rhodanine techniques. Microscopical examination revealed an extensive distorsion of lobular architecture by widespread fibrosis in periportal areas and around centrolobular veins. In some lobules the fibrosis was extended to perisinusoidal spaces. Capsular fibrotic thickening and cellular degeneration or atrophy were detected in some cases.

Cardiac malformations were evident in 10 calves; they were characterized by atrial (one calf) and interventricular septal defect (eight calves), and patent ductus arteriosus (three calves).

A genetic cause is strongly suspected.

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