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Five dogs with spontaneous PAA and 4 clinically healthy dogs were used. These 5 dogs with spontaneous PAA were divided into 5 cases, case 1; early stages of PAA and cases 2 to 5; late stages of PAA.

Microscopically, atrophic and non-atrophic areas were observed from both biopsy specimens and necropsy samples collected from the pancreas of case 1. Atrophic areas were characterized by destroyed acinar structures, whereas in non-atrophic areas acinar cell structures could still be observed. The microscopic appearances of the pancreases in cases 2 to 5 coincided with those recognized in previous reports. In case 1 atrophic areas were located near the pancreatic duct and on the periphery of the lobuli. In the atrophic areas, metaplasia of ductal cells and hyperplastic changes of ducts were observed. In the non-atrophic area of case 1, almost all acinar cells of biopsy specimens had zymogen granules, but acinar cells of necropsy samples which had zymogen granules were rare. Furth-

ermore, the cytoplasm of acinar cells in the non-atrophic areas of case 1 were characterized by diffusely distributed vacuoles and atrophy.

Apoptosis of acinar cells was seen in non-atrophic areas of cases 1 and 3. In addition, apoptotic cells showed a positive reaction to TUNEL staining.

On immunohistochemical examination of the pancreatic islet cells in the non-atrophic area of case 1, a decrease in the proportion of pancreatic islets cells was found compared to the controls. In the atrophic areas of all cases, the appearance of islets of Langerhans and the ratio of islet cell structures was very different from that of the controls.

These results suggested that atrophy of the acinar cells in PAA develops near the pancreatic ducts and the periphery of the lobuli, and the possibility that apoptosis plays a role in the pathogenesis of PAA. In addition, it appeared that atrophy of acinar cells might have an effect on the composition of the islet cells.

#### Clinicopathological studies of serum adenosine deaminase activity in cattle

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The purpose of this study was to clarify the clinical usefulness of serum adenosine deaminase (ADA) activity in cattle.

The serum levels of ADA in 135 clinically healthy cattle and 128 cattle with various diseases were measured by an automated enzymatic method. Other blood chemical parameters that are measured routinely in clinics and bovine leukosis virus antibodies (BLVab) of the diseased cattle were assayed at the same time. Moreov-

er, 85 livers of diseased cattle were examined for routine histopathology.

The results obtained were follows :

Serum ADA levels of normal cattle were  $5.4 \pm 2.6$  IU/l (mean  $\pm$  SD), which was lower than that of humans. Serum ADA levels of healthy milk cows were lowest during pregnancy. There was no statistically significant difference between milk cows and castrated Holsteins.

Serum ADA levels were high in most cattle

with various hepatic diseases and leukosis. Cattle with leukosis showed high serum ADA activity with or without BLVab. There was no correlation between ADA activity and the histopathological findings of degeneration and the degree of fibrosis in the diseased livers. However, serum ADA activity was significantly elevated accompanied by increase of mononuclear cells infiltration.

In conclusion, the elevation of serum ADA activity in bovine liver diseases appeared to be a result of invading mononuclear cells as well as disruption of hepatocellular integrity. The results of this study suggest that the measurement of serum ADA activity might be a useful aid in the diagnosis of bovine hepatic diseases and leukosis.

#### Clinical application of ultrasonographic examination to the canine neonatal brain

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Ultrasonography (USG) is widely used in veterinary medicine as a diagnostic imaging technique. However, little intracranial USG and no pulsed-wave Doppler analysis has been reported in veterinary medicine. Therefore, to obtain ultrasonographic images of brain abnormalities in neonatal dogs, the following experiments were done using 12 neonatal and 2 adult dogs.

Intracranial USG, using the B-mode, color-flow Doppler mode and pulsed-wave Doppler mode was performed on normal neonatal dogs from the day of birth to 50 days of age and on 2 normal adult dogs. Intracranial USG was also performed on 4 dogs with experimentally induced intracranial disease and 2 puppies with mild ventriculomegaly. Pulsed-wave Doppler imaging was performed on the rostral cerebral artery to evaluate blood flow velocity and the resistive index (RI).

It was possible to image the brains of neonates up to approximately 40 days of age. The changes noted in the brain with age were the size of the brain, the formation of sulci and the curve of the rostral cerebral artery. Compared

to the adult brain, the neonatal brain had a less clear distinction between gray matter and white matter with smoother sulci. Blood flow velocity increased as puppies grew older and it was higher than that of adults at 15 or 20 days after birth. The RI was not stable within 24 hours after birth; however, it was stable between 0.65 and 0.70 from 1 day to 30 days old. Then the RI tended to decrease gradually to around 0.50, which was the average level of the normal adults. In the dogs with experimentally induced hemorrhage in the brain parenchyma and ventricle, the hemorrhagic lesions were imaged as hyperechoic areas. One day after intraventricular hemorrhage, mild enlargement of the ventricle and a hyperechoic mass suspected to be a clot were visualized. In puppies with spontaneous mild ventriculomegaly, enlargement of the ventricle and the 3rd ventricle were observed. USG of the brains from puppies with canine herpetic encephalitis, which were fixed in buffered formalin placed in a water bath, revealed enlargement of the lateral ventricle and hyperechoic lesions that resulted from intraventricular hemorrhage.