Some Clinical and Microscopical Findings in an Icteric Foal

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Some clinical and microscopical findings of the liver, spleen, kidneys, lungs, heart, and lymph nodes of a foal dying of hemolytic icterus on the eleventh day after birth were described. In the liver, cholestatic jaundice and hemosiderosis were quite distinct. Bile pigments bright, light greenish-yellow in color appearing to be dilute were very frequently seen in the sinusoids and Disse's spaces, but not in the bile canaliculi. Hemosiderosis was also marked in the spleen and lungs. Extramedullary hematopoiesis was often observed in the liver and spleen.

Many papers have been published on hemolytic icterus of newborn foals, but only few of them bear the pathological descriptions of dead cases. In Japan, there have been no reports on the pathological findings of this disease. In this paper, some clinical and microscopical findings of the liver, spleen, kidneys, heart, lungs, and lymph nodes of a newborn foal which died on the 7th day after onset of the disease with typical clinical, hematological, and immunohematological findings are chiefly described.

History and Clinical Signs

A Thoroughbred female foal was born apparently normal on April 20, 1971. In a short time after birth, she got up and nursed actively. On April 24, the attendants of the farm noticed that she was very depressed and anorectic.

In the first clinical examination, the body temperature of the foal was 40.3°C. The heart beat was accentuated, resulting in pulse frequencies of 150-160 per minute, and quick shallow breathing was observed. Her mucous membranes were yellowish-white in color. She spent much time lying down. Erythrocyte count was 2.8 millions. She was treated with blood transfusion, glucose and electrolyte solutions, thiamine, steroid hormone, and antibiotics.

The blood findings obtained on the morning of April 30 were as follows: erythrocyte count, 1.49 millions; packed cell volume, 7%; minimum and maximum red cell fragilities, 0.85 and 0.46; marked anisocytosis and erythrophagocytosis on the blood film; icterus index, 120 units; sideroleukocyte count, 0.5 per ten thousand leukocytes; leukocyte count,
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8,700; differential leukocyte count, shift to the left (stab. 21.5 %, seg. 63.5 %, l.y. 13.5 %, and mon. 1.5%); high hemolysin titer of mare's serum to foal's erythrocytes, positive for mare's serum at 1:2.048; and strongly positive for direct Coombs' test. She died on April 30.

Microscopical Findings

Postmortem examination showed icteric coloration, anemia and the hydropic appearance of all the organs.

The liver, spleen, kidneys, heart, lungs, and lymph nodes (mesenteric and inguinal) were examined microscopica-

Fig. 1. Liver. Disorder in arrangement of hepatic cords is marked
Sinusoids are dilated extremely. Hematoxylin and eosin (H.E.) staining, x 40.

Fig. 2. Liver. Dilated sinusoids contain fibrinoid fibril-like substances and polymorphs.
Kupffer cells show active phagocytosis of hemosiderin or bile pigments. Bile canaliculi are filled with bile pigments. Hepatic cells show granular degeneration. H. E. staining, x 200.
Materials were collected from them, fixed in 10% formalin solution, and embedded in paraffin. Paraffin sections were stained with hematoxylin and eosin, and Prussian blue. Microscopically, the most striking changes were observed in the liver.

Liver: In short, cholestatic icterus and hemosiderosis were extremely notable.

Bile thrombi in bile canaliculi and bile pigment deposition in the protoplasm of hepatic cells were extremely marked extending over the entire hepatic lobule. Most of the bile pigments were dull greenish-brown in color, but a few were bright, light greenish-yellow and appeared to be dilute. The latter were found in no bile canaliculi.

Disorder in arrangement of the hepatic cell cords was considerably intensive. In general, hepatic cells showed swelling and often formed vacuoles in the granulating protoplasm. The hepatocellular degeneration was more intensive in the centrolobular part in general, where swelling of cells and loosening or reticulation of the protoplasm were mainly observed. The intrahepatocellular vacuoles often contained bile pigments and occasionally hyaline substances. Hepatic cells occasionally showed intralobular necrosis solitarily or centrolobular necrosis microfocally. The necrosis were accompanied by a neutrophil reaction in most cases.

The sinuses were markedly dilated in general. In the sinusoidal lumina, there were often regular or irregular network structure due to the existence of fibrinoid fibril-like substances. Most of these substances were suggestive of debris of the erythrocytic cell membrane. The sinuses contained a very few erythrocytes, including few normoblasts, in addition to a few neutrophils and sometimes free bile pigments. They rarely contained relatively large spherical hyaline substances some of which had bile pigments in the interior, and very rarely irregular lamellar structures some of which were taken for endothelial cells.

Activation of sinusoidal endothelial cells was outstanding. Hemosiderosis was extremely notable. The hemosiderin-laden endothelial cells also frequently took bile pigments and/or sometimes erythrocytes. Endothelial cells taking only bile pigments were occasionally pointed out.

Disse's spaces were markedly dilated, frequently bringing about narrowing of the sinusoidal lumina, and often contained dust-form proteinic coacervates or bile pigments. When the bile pigments were compared, the bright, light greenish-yellow pigment which appeared to be dilute was pointed out more frequently and readily than the dull greenish-brown one (see the above-mentioned).

The central veins rarely had the bright, light greenish-yellow bile pigment in the lumen.

The interlobular septa were edematous. In their blood vessels the walls evidently showed edematous loosening and swelling were occasionally observed. In the septa, the light greenish-yellow bile pigment was present occasionally in tissue spaces and rarely in the vascular and bile-ductal lumina. Hemosiderin-laden cells were rarely found in the septa (in the hepatic capsule as well).

Spleen: Engorged with the blood.

Hemosiderosis was conspicuous. Erythrophages were observed occasionally. The follicles were edematous and very poor in cellular elements. Edematous swelling of the capillary walls was pointed out. Some small arteries evidently revealed edematous loosening and swelling. Extraduillary hematopoiesis was relatively intense. It was difficult to find bile pigments.

Kidneys: The interstitium manifested edematous loosening. Small arteries often showed edematous loosening and swelling. Epithelial cells of the renal
tubules underwent granular degeneration and were sometimes accompanied by vacuolation. The tubular epithelia occasionally had a deposition of dull greenish-brown pigment suggestive of bile pigment. Hemosiderin-laden cells were very rarely found in the interstitium. Neutrophils were present very sparsely in the capillary lumina.

Heart: Cardiac muscle fibers manifested vesicle formation in the protoplasm (hydropic degeneration). Neutrophils were present very sparsely in the capillary lumina.

Lungs: Edematous. In the alveolar septa there were many hemosiderin-laden cells among which macrophages probably phagocytizing bile pigment-laden cells were rarely found.

Lymph nodes: The inguinal lymph node developed simple acute inflammation. Neutrophils were very rarely found in the sinuses of the mesenteric lymph node. In these lymph nodes siderophages and erythrophages were observed. There was extramedullary hematopoiesis.

**Discussion**

In the microscopical observation of the present case, interesting changes were seen in the liver, which showed intense intrahepatic cholestatic jaundice and hemosiderosis. Jubb and Kennedy described that icterus in this disease was evident by the second day and increased in severity.

The bile pigment that was bright, light greenish-yellow in color and that appeared to be dilute was more noticeable than the bile pigment dull greenish-brown in color. On the other hand, a fibrinoid fibril-like substance appearing to be debris of erythrocytic cells was often seen in the sinusoids. It is probable that these findings may be a result of hemolysis.

Occasionally, hepatic cells showed intralobular necrosis solitarily or centrolobular necrosis microfocally. Doll described that sections from the liver of an icteric foal dying at 7 days of age showed central necrosis involving one-fourth to one half to the area of some lobules, and that these histopathological lesions were severer in foals which died later. It is probable that both prolonged anemia and toxic effects of jaundice may have contributed to the production of these lesions in the present case.

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子馬の溶血性黄疸症1例における2,3の
臨床ならびに組織学的所見

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生後4日目に発症し、7日間の経過で死亡した溶血性黄疸症の1例（サラブレッド種、母うす）について、肝、脾、腎、心、肺およびリンパ節（腸間膜、リンパ節および臓器リンパ節）の病理組織学的観察を行なった。

肉眼的には、全身性の強い黄疸および各臓器の貧血が目立ったほかは、著変はみられなかった。

顕微鏡的には、肝において高度の胆汁停滞性黄疸ならびにヘモジデロージスが観察された。胆汁色素には、鮮やかな帯淡緑黄色で稀薄な感じを与えるもののが、静脈洞内、ジェッセ腔内にしばしばみられた。ヘモジデロージスは肝および肺においても著明であった。また肝および肺で赤血球貪食像がしばしば観察された。

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