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PATHOLOGICAL ASPECT IN PATIENTS INFECTED WITH DENGUE HEMORRHAGIC FEVER

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The pathologic manifestation in dengue hemorrhagic fever was obtained mostly in patients infected with dengue virus, who followed a 3-4 days fever-course, then underwent into shock and died within 24-48 hours thereafter. The pathologic changes prior to this critical period and during the recovery stage has not as yet been known. The pathologic lesions in those who died were grouped into three categories as follows

I. VASCULAR LESION there was no significant structural alteration in the capillary walls under light microscopic study. There is some perivascular edema and mild perivascular infiltration by mononuclear cells.

II. LESION SUGGESTING A PHYSIOLOGIC SEQUELAE

a. Leakage of blood elements mainly red blood cells outside of capillary channels: Complexed mechanisms of such leakage were proposed as 1) defective clotting mechanism by deficiency in blood clotting factors such as fibrinogen, prothrombin complex deficiency, lowering of platelets due to productive failure or increased destruction. 2) Intravascular coagulation which was suggested by the

hematologic evidence namely decreased levels of fibrinogen, platelets, and some increase in amount of fibrin degradation product and transitory decrease in platelet survival time in the blood of patients infected with dengue virus. Pathologically fibrin and platelet thrombi were occasionally demonstrated in some fatal adolescent cases, but very rarely in pediatric patients. 3) Some unknown vasculopathy which leads to capillary leakage for erythrocytes.

b. Leakage of water, electrolytes and plasma proteins which is brought about by permeability factors whose release are probably triggered by the immune mechanism. The factors include anaphylatoxin (C3a, C5a), and kinins among others.

c. Visceral change. Hyperplasia of immunocytes namely plasma cells and lymphocytes is quite distinct in spleen, lymph nodes and liver. Phagocytosis of lymphocytes and erythrocytes is prominent in these same organs. Some liver changes such as focal necrosis and Councilman-like body formation, are reminiscent of early lesions in monkey liver experimentally infected with yellow fever.

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