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HEARTWORM DISEASE IN DOGS: ITS THERAPY AND PATHOPHYSIOLOGY

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Heartworm (HW) disease is a circulatory disease in dogs caused by infection with filarid worms, *Dirofilaria immitis*. Although it is classical and the most well-known disease in dogs, many dogs fall victim to it at present time. Many studies have been performed on the prophylaxis, diagnosis and therapy for the disease, but there have been only a few investigations on pathophysiology. In the present study, the author describes a therapy for HW disease (surgical heartworm removal with flexible alligator forceps), and its pathophysiology, especially caval syndrome(CS).

1. Therapy for HW disease: The flexible alligator forceps was developed in our laboratory in 1985. This forceps can remove HWs from the pulmonary arteries and right atrium through the jugular vein (without thoracotomy). The removal efficiencies of HWs with this forceps were above 90% both in dogs with pulmonary HW disease and those with CS. After HW removal, the clinical signs, laboratory data, cardiopulmonary function values, and proliferative lesions of pulmonary arterial intima improved rapidly, indicating the efficacy of the treatment. Pulmonary arterial pressure fell immediately after HW removal, and elevated immediately after HW insertion. Therefore, the presence of HWs in the pulmonary arteries contributed to pulmonary hypertension in HW disease. Moreover, most dogs having an ominous prognosis after HW removal had a severe pulmonary thromboembolism involving dead HWs. Pulmonary thromboembolism with dead HWs related most significantly with pulmonary hypertension. The treatment of pulmonary thromboembolism might be the most important remaining issue to be addressed, and treatment producing obstructive pulmonary arterial lesions with dead HWs, such as adulticidal treatment, might possibly be avoided.

2. The pathophysiology of HW caval syndrome (mechanism of HW migration from the pulmonary arteries to the right atrium): Dogs with HW-CS suddenly show signs such as prostration, anemia, caval murmur, jugular pulsation, circulatory disturbance, liver and renal failure and hemoglobinuria. Dogs die acutely, if they cannot receive adequate treatment. The migration of adult HWs from the pulmonary artery towards the right atrium is the beginning of this syndrome. However, the inducer of HW migration has been unknown. We found HW migration in microfilaremic HW-infected dogs after administration of milbemycin D, an HW prophylactic having a microfilaricidal

activity. From the measurement of cardiopulmonary function values at the shock phase, we considered that HW migration might be induced by a decrease in blood flow in the right heart system. Therefore, we attempted to reduce cardiac output with administration of a beta-blocker (metoprolol). After its administration, cardiac output and heart rate decreased, and HWs migrated from the pulmonary artery to the right atrium. Because pulmonary thromboembolism involving recently dead HWs were found in almost all dogs with CS, we considered that the death of HWs might be related to the migration of live HWs. We administered extracts of HW body fluid intravenously to HW-infected dogs, who went into shock, cardiac output decreasing rapidly, and HWs migrating to the right atrium. In addition, since dead HWs produce pulmonary arterial obstruction, we inserted dead HWs or HW-like silicone tubes into the pulmonary arteries using flexible alligator forceps. Some days after insertion, pulmonary arterial pressure elevated gradually, cardiac output decreased, and HWs migrated toward the right atrium. In dogs with naturally acquired CS, a decrease in cardiac output from heavy worm burden and a release of body fluid into circulation as well as pulmonary thromboembolism might be inducers of the HW migration.

The very name of the disease indicates that the most important parasitic site of HWs is the venae cavae. In the present study, dogs with CS showed signs of severe venous congestion. After removal of live HWs from the tricuspid valve area, the valve functions improved, and tricuspid regurgitation and signs of venous congestion disappeared. Therefore, the circulatory disturbance in this syndrome is due to tricuspid valve dysfunction caused by the presence of HWs at the tricuspid valve orifice. Another characteristic of CS is intravascular hemolysis. Dogs with CS have severe liver injury. They exhibit abnormal lipid metabolisms owing to liver injury, such as low plasma lecithin-cholesterol acyltransferase activities, high cholesterol contents in low density lipoprotein and erythrocyte membranes, and high mechanical fragility of erythrocytes. Hemolysis with chemical and immunological ,mechanisms and disseminated intravascular coagulation were denied. In CS, the abnormal blood flow in the area of the tricuspid valve orifice caused by the presence of HWs might lead to the breakdown of the mechanically fragile erythrocytes.

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