ABSTRACT OF THE MAIN THESIS

Clinical and Experimental Studies on Functional Tricuspid Insufficiency in Dogs

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Tricuspid insufficiency is a clinical term generally indicating the incomplete obstruction of the tricuspid valve of the right ventricle by various causes. In practice, it is caused either by an organic change of the tricuspid valve or by the centrifugal displacement of this valve free from any organic change.

For instance, insufficient closure is induced by right ventricular dilatation or congestive right heart failure which appears after the displacement of the chordae tendineae, papillary muscle, or the site of attachment of the valve in the organ of attachment of the tricuspid valve. The former is so-called organic tricuspid insufficiency, and the latter functional tricuspid insufficiency. Clinically, the differential diagnosis of one from the other seems extremely difficult.

Functional tricuspid insufficiency is mainly induced by displacement of the tricuspid valve. As causes for the displacement of the tricuspid valve, right ventricular dilatation, stenosis of the pulmonary artery, embolism of the pulmonary artery, pulmonary circulatory disturbance, pulmonary congestion, and mitral insufficiency might be considered. Such venous or pulmonary circulatory disturbance most frequently occurs in filariasis of dogs. Filariasis is said to be found in 70~80% of the adult dogs in Japan. The incidence of canine filariasis is quite high in Japan, as compared with any

other country. Right ventricular dilatation, embolism of the pulmonary artery, and pulmonary circulatory disturbance due to canine filariasis leading secondarily to functional tricuspid insufficiency have been found at a considerable frequency. Only a few reports, however, are available on the pathophysiological studies of this type of insufficiency.

From a clinical standpoint, general clinical tests, blood tests, blood biochemical tests, urinalysis, roentgenography, electrocardiography (ECG), phonocardiography, and the measurement of intracardiac pressure were conducted in 13 dogs suspected of functional tricuspid insufficiency and selected from among 48 dogs affected with heart disease. These tests were followed by autopsy. The results of these tests and autopsy were summarized, and the pathological changes of tricuspid insufficiency were interpreted theoretically. The clinical diagnosis of this disease was discussed. Attempts were also made to clarify the mechanism of development of functional tricuspid insufficiency by pulmonary arteriography and other means for the studies of heart specimens.

In the 13 clinically suspected cases, the results of the tests were summarized and findings characteristic to this disease pointed out. In the general clinical tests, congestive right heart failure, or carotid vein pulsation, liver congestion, intracardiac murmur, edema, and ascites were important keys for the diagnosis of this disease. In the general blood tests, it was important to detect microfilariae for the confirmation of congestive right heart failure caused by canine filariasis. Qualitative changes of the circulating blood appeared to be more important for making prognosis than for making clinical diagnosis. The results of urinalysis and blood chemical tests failed to provide characteristic findings for the clinical diagnosis of this disease. In roentgenological studies, bilateral enlargement of the ventricles and atria, dilatation of the pulmonary artery, and embolism by canine filariae were important findings for the clinical diagnosis of tricuspid insufficiency. In ECG, dilatation of the

right ventricle and atrium was a reliable clue for the diagnosis of this disease. In phonocardiographic studies, systolic or diastolic intracardiac murmur, especially regurgitation murmur, at the tricuspid ostium was reliable. In the test for intracardiac pressure, it was important for the evaluation of abnormalities in the hemodynamics of this disease to find that the left ventricle and aorta decreased in pressure, while the right ventricle, right atrium, vena cava, and pulmonary artery increased unusually in pressure. These changes in pressure were especially important for expressing the regurgitation of the blood stream through the incompetent tricuspid valve. At autopsy, findings were obtained to confirm the results of each clinical test. When canine filariae infest the right ventricle abundantly, they invade the tricuspid ostium causing tricuspid insufficiency. When they coil around the chordae tendineae, the blood stream passing through the right ventricle is stagnant, which leads to right ventricular dilatation. The tricuspid valve is displaced distally to cause insufficient closure.

Judging from the functional disturbances observed in clinical examination and the autopsy findings, it seemed that the blood stream within the right ventricle might regurgitate into the right atrium or vena cava in the systolic phase in these cases, causing pulsation of the carotid vein. So-called congestive right heart failure thus developed. This phenomenon progressed into somatic circulatory failure, tissue circulatory insufficiency, and distrubances in the function of the liver, kidney, and lung. These changes were aggravated through the so-called vicious cycle and made the life of the dog short.

These results of clinical examination are probably quite important for the clinical diagnosis of functional tricuspid insufficiency. The mechanism of development of this disease, however, has not yet fully been understood.

Attempts were therefore made to clarify the mechanism of development of functional tricuspid insufficiency in clinical cases. Hemodynamic studies were conducted on animals with experimental pulmonary stenosis caused by pulmonary arteriorrhaphy and leading to functional tricuspid insufficiency.

The experimental materials used were 12 adult mongrel dogs showing no abnormalities on general clinical examination, blood or urine tests, roentogenography, ECG or phonocardiogram. Under fluothane anesthesia accompanied by positive-pressure respiration, thoracotomy was performed between the left 3rd and 4th ribs. In order to produce stenosis, the main pulmonary arteries were then sutured in such varying manner as to reduce the caliber to about 1/3, 1/2, or 2/3 the normal size or to close the vessel completely. Changes in blood pressure before and after stenosis were observed by the aid of cardiac catheterization.

When stenosis was produced on the main pulmonary artery in such manner as to make the caliber of the artery less than 2/3 the original size by suture, neither stagnation of the blood stream in the right ventricle nor functional tricuspid insufficiency due to right ventricular dilatation was noted. On the other hand, when the main pulmonary artery was reduced in caliber to less than 1/3 the original size by suture in order to produce stenosis, acute circulatory failure occurred to the lung, which led to death. Therefore, the caliber of the main pulmonary artery was reduced to about 1/3 the original size by suture, and changes caused by the resulting stenosis were observed in chronological sequence. Stagnation of the blood stream in the right ventricle definitely caused dilatation of the right ventricle. Then the tricuspid valve was dislocated centrifugally and functional tricuspid insufficiency resulted. In the phase of cardiac systole, regurgitation was noticed in the blood stream passing from the right ventricle to the right atrium. Such regurgitation phenomenon reached the vena cava, producing pulsation of the jugular vein. This phenomenon was confirmed by injecting an opaque medium by way of a catheter inserted into the heart and examining the regurgitation of the opaque medium by roentgenography. The hemodynamics in this condition consisted of a rise in pressure of the venous system, such as pressure of the right ventricle, of the right atrium, and of the vena cava. Pressure fell in the left ventricle. Observation on

chronological changes in pressure after the occurrence of stenosis in the pulmonary artery revealed no dilatation of the right ventricle 30~50 minutes after suturing of this artery to reduce the caliber to 1/2 of the original size.

When closure was carried out up to 2/3 of the original caliber, pressure began to change within 5~10 minutes and regurgitation was confirmed in the blood stream. These changes in blood pressure agreed approximately with those noticed in clinical spontaneous cases.

Experimental stenosis of the pulmonary artery, however, was anticipated to be accompanied by compensatory changes with the lapse of time, judging from the anatomical structure of the artery. Such experimental conditions might be different from the clinical spontaneous case of functional tricuspid insufficiency. Then, it was assumed necessary to make observation on the chronic course of experimental functional tricuspid insufficiency. This observation was made on 6 experimental dogs considered to be healthy be general clinical examination. In these dogs, the main pulmonary artery was sutured to about 2/3 closure by the method of pulmonary arteriorrhaphy. Observation on the course of disease was continued for 80~90 days.

As a result, general clinical examination revealed the appearance of pulsation of the jugular vein, systolic intracardiac murmurs detected by auscultation, general malaise, and refusal to exercise. These symptoms were in close agreement with the clinical findings of functional tricuspid insufficiency. The hemodynamics of these cases consisted of congestion of the circulating blood within the right ventricle due to stenosis of the pulmonary artery, leading to a rise in pressure of the right ventricle, dilatation of the right ventricle, and centrifugal displacement of the tricuspid valve. The development of functional tricuspid insufficiency was thus confirmed.

When changes in pressure were observed, pressure increased in the right atrium and owing to the regurgitation of the blood stream from the right

ventricle to the right atrium. These hemodynamics changes were quite similar to those in the clinical spontaneous case and the experimental case of acute functional tricuspid insufficiency. At autopsy conducted after the experiment was over, many of the findings obtained were similar to those from the clinical spontaneous cases of this disease.

Studies on the clinical spontaneous cases of functional tricuspid insufficiency revealed that canine filariae had infested the heart or pulmonary artery in most of the cases, causing stenosis or embolism and eventually functional tricuspid insufficiency.

When infestation of canine filariae occurred to the ostium of the tricuspid valve or that of the pulmonary arterial valve ostium, nothing was known at all about the mode of functional disturbance of the tricuspid or pulmonary arterial valve, or the subsequent disturbance of the intracardiac blood stream. Detailed hemodynamic studies may make it possible to clarify the pathophysiology of functional tricuspid insufficiency caused by canine filariae.

In order to study the mechanism of development of this disease secondary to filariae infestation, the following experiment was performed. In 6 clinically healthy adult dogs free from canine filariae infestation, the heart was removed after blood letting to be used as a heart specimen. The right and left atrium were then resected to make it possible to observe the tricuspid and mitral valve directly. An artificial pump was attached to the wall of the right and left ventricle. It was filled with physiological saline, so that the intracardiac pressure might be increased and decreased by increasing and decreasing pressure upon it, respectively. The opening and closing of the tricuspid or mitral valve induced by regulating the pump pressure was recorded by a 16 m/m cinecamera, along with observation by the naked eye. Intracardiac pressure, as well as pressure within the artificial pump, was recorded by an electric sphygmomanometer under synchronization with the cinecamera. Surviving canine filariae were collected from another

experimental dog. Ten to twenty of them were placed at the tricuspid ostium or pulmonary ostium of those experimental dogs, in which recording was made on changes in pressure and mode of opening and closing of the tricuspid valve.

As a result, the deposition of canine filariae at the tricuspid ostium disturb the closing of the tricuspid valve. The circulaling blood driven out of the right ventricle was observed by the naked eye to regurgitate into the right atrium through the spaces among the deposited canine filariae. The right ventricular pressure was low when canine filariae were deposited at the tricuspid ostium, causing an insufficient closure, and began to increase upon their removal. When canine filariae were placed in the pulmonary artery to cause embolism, this artery underwent stenosis. The circulating blood driven from the right ventricle was blocked, causing dilatation of the right ventricle. As a result, the tricuspid valve was displaced centrifugally, causing an insufficient closure. Regurgitation of blood from the right ventricle to the right atrium was confirmed. Since canine filariae forming an embolus in the pulmonary artery interfered with the outflow of the blood, pressure increased within the right ventricle. Removal of these canine filariae resulted in a normalization of the blood stream and a fall in pressure within the right ventricle.

Based on those experimental results from the heart specimens, it was confirmed that the functional tricuspid insufficiency occurring as the result of infestation of canine filariae at the tricuspid ostium or in the pulmonary artery was due to the disturbance of blood circulation through the pulmonary artery by these canine filariae.

General clinical examination, blood tests, blood biochemical tests, urinalysis, roentgenological studies, ECG, phonocardiography, and intracardiac pressure tests were conducted in 13 clinically suspected cases of functional tricuspid insufficiency.

As a result, the following characteristic findings were found to be available for the clinical diagnosis of this disease: pulsation of the jugular

vein, intracardiac murmurs, ascites, fluid in the pleural cavity, edema, weight loss, liver congestion, presence of microfilariae, bilateral enlargement of the ventricles and atria, embolism by canine filariae, rise in pressure of the right ventricle, coughing, rales, dyspnea, and hematuria.

In order to elucidate the mechanism of development of functional tricuspid insufficiency and its pathophysiology, experiments with the sutured pulmonary artery were conducted to study hemodynamics. Stenosis of the pulmonary artery caused dilatation of the right ventricle accompanied by congestive right heart failure. Since such dilatation of the right ventricle caused a centrifugal displacement of the tricuspid valve, functional tricuspid insufficiency occurred. These experimental results were similar to those obtained from observation made on the chronological sequence in chronic and acute experiments. They also showed a considerable agreement with hemodynamic changes in the clinical cases. In these cases, canine filariae probably played a major role in the development of this disease. The results of studies on the state of tricuspid insufficiency in the removed heart and hemodynamic changes in such state as embolism at the tricuspid valve ostium or in the pulmonary artery induced by canine filariae revealed the following mechanisms. When canine filariae were deposited at the tricuspid valve ostium or in the pulmonary artery, the tricuspid valve underwent mechanical insufficiency. When embolism was formed in the pulmonary artery, congestive right heart failure developed and functional tricuspid insufficiency occurred mechanically as the result of dilatation of the right ventricle. These mechanisms were confirmed and the state of disease was clarified.