

猫の心臓右心室乳頭筋の異形成の1症例

誌名	Japanese journal of veterinary science
ISSN	00215295
著者名	宇根,ユミ 谷本,忠司 菅沼,常德
発行元	Japanese Society of Veterinary Science
巻/号	49巻6号
掲載ページ	p. 1133-1134
発行年月	1987年12月

農林水産省 農林水産技術会議事務局筑波産学連携支援センター
Tsukuba Business-Academia Cooperation Support Center, Agriculture, Forestry and Fisheries Research Council
Secretariat



Papillary Muscle Dysplasia of the Right Ventricle in the Heart of a Cat

Yumi UNE, Tadashi TANIMOTO, Tsunenori SUGANUMA¹⁾, Tetsuo HYOHDOH²⁾, Kinji SHIROTA, and Yasuo NOMURA

Department of Veterinary Pathology and Veterinary Radiology¹, School of Veterinary Medicine, Azabu University, Sagami-hara, Kanagawa 229, Hyohdoh Veterinary Clinic², Asahi-ku, Yokohama 241, Japan

(Received 7 August 1986/Accepted 4 September 1987)

Jpn. J. Vet. Sci. 49(6): 1133-1134, 1987

KEY WORDS: cat, heart, muscle dysplasia.

Congenital heart disease (CHD) is believed to be less common in cats [3, 5, 7] than in dogs [13] and humans [14]. Based on the morphological analysis, it has been reported that the common CHD in cats is the malformation of mitral valve complex, dysplasia of tricuspid valves, ventricular septal defect, stenosis of aortic valves and persistent common atrioventricular canal [7]. There have been no reports on the papillary muscle dysplasia in animals.

A 7-month-old male Japanese cat showed right-sided heart failure with prominent ascites and diarrhea. Anorexia and depression appeared on the day before death. The systolic regurgitant murmur was auscultated. Laboratory examinations revealed mild anemia. Radiological examinations proved marked approach of the right ventricular curve to the sternum and severe expansion of the posterior vena cava to approximately 2.4 cm in diameter. The heart was extremely enlarged with rounded right ventricular margin and dislocation

of the apex cordis to the left. The cardiothoracic ratio was 91.6%.

At necropsy, subcutaneous edema, ascites, hydrothorax and hydropericardium were noted. The posterior vena cava and pulmonary artery were markedly dilated. The lung and liver revealed severe congestion. The right atrium and ventricle were prominently dilated (Fig. 1), showing thin atrial wall and irregular endocardial thickening with calcification. Right atrioventricular orifice was widely expanded, measuring 24 mm in diameter. Three leaflets were not plastered on the wall of right ventricle. In the tricuspid valve, there were no incisions between the leaflets, and the length from the orifice to the free border of each leaflet was uniform, approximately 5 mm. The leaflets were transparent except the rough zone. The large papillary muscle in the right ventricle was hypertrophic and cylindrical, measured about 3 mm in diameter, had a blunt top, and directly attached to the midportion of angular and parietal cusps. No rough zone and basal chordae tendineae were observed between the papillary muscle and tricuspid valves (Fig. 2). The small papillary muscle arose from slightly upper portion of halfway of the septal wall and also attached directly to the free edge of the septal cusp lacking chordae tendineae. Elongated subarterial papillary muscle was



Fig. 1 Dilatation of right atrium and ventricle and marked congestive edema of the lung.

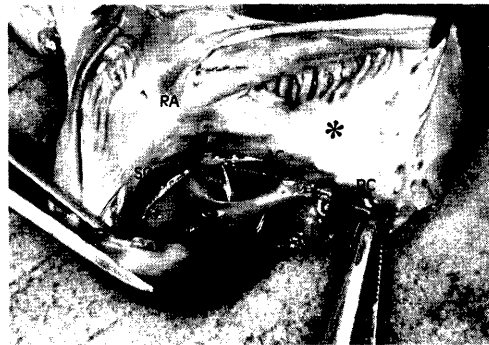


Fig. 2. Right atrium (RA) and ventricle (lower half). The RA endocardium is markedly thickened with calcification (*). Hypertrophied large papillary muscle (LPM) directly attaches to the midportion of angular (AC) and parietal (PC) cusps without chordae tendineae. Arrowhead indicates the area where the subarterial papillary muscle attaches to the leaflets. SC: septal cusp.

also directly attached to the midportion of angular and septal cusps. Two adult heart worms were present in the right ventricle. The endocardium of the right ventricle was rough. No changes were observed in papillary muscles of the left ventricle.

Histopathologically, the endocardium of the right atrium revealed irregular fibrous thickening. In addition focal calcification, necrotic and fibrotic foci were seen in the subendocardial myocardium of the right atrium. The tricuspid valvular tissue consisted of the fibrosa and spongiosa as well as normal valvular tissue. The immature valvular tissue, mainly consisting of the spongiosa without proper structure, was not seen in the leaflets. The large papillary muscle of the right ventricle had no conducting system.

In feline species, there are three papillary muscles in the right ventricle [2]. The papillary muscles of the present case were normal in number and location [9], but lacked chordae tendineae. The tricuspid insufficiency of the present case appeared to have been due to the traction of the tricuspid valve by the directly attached papillary muscles, shortened leaflets and expanded orifice. Similar tricuspid valve insufficiency has also been described in tricuspid valve dysplasia [3, 4, 6, 7]. The papillary muscles, chordae tendineae, and most of the cusps are derived from the embryonic ventricular trabeculae [11]. The atrioventricular valves are thick and fleshy at first and become thin and fibrous with time. It is said that the chordae tendineae are initially thick and muscular, and transform into delicate fibrous strands later [12].

In some instances, an insufficient development of the chordae tendineae, which occurs frequently in the left ventricle, results in direct attaching of the papillary muscle to the leaflets [11]. Tricuspid valve dysplasia is defined as thickening or deformity of the valvular leaflets, adhesion of the leaflets to the ventricular wall, and stenosis of the atrioventricular orifice, and a diffuse to focal incomplete valvular tissue development [1]. In the present case, the valvular tissue showed complete differentiation, but there were no chordae tendineae. Thus, this case was diagnosed as "papillary muscle dysplasia of the right ventricle".

In human beings, papillary muscle dysplasias such as dwarfish change, excessive branching and direct attaching to the valve are considered to be a minor cardiac malformation accompanying Down's syndrome [8, 10] revealing 18th trisomy [10]. In the present case, chromosomal examination was not carried out.

REFERENCES

1. Becker, A. E., Becker, M. J., and Edwards, J. E. 1971. *Arch. Pathol.* 91: 167-178.
2. Crouch, J. E. 1969. Text-Atlas of Cat Anatomy. Lea & Febiger, Philadelphia.
3. Fox, P. R., Tilley, L. P., and Liu, S. -K. 1983. Heart disease. pp. 276-294. *In: Feline Medicine*, 1st ed. (Pratt, P. W. ed.), Am. Vet. Publ. INC., Santa Barbara.
4. Harpeter, N. K. 1977. *Adv. Vet. Sci. Comp. Med.* 21: 39-74.
5. Liu, S. -K., Tachjian, R. J. and Patraik, A. K. 1970. *J. Am. Vet. Med. Assoc.* 156: 1319-1330.
6. Liu, S. -K. and Tilley, L. P. 1976. *J. Am. Vet. Med. Assoc.* 169: 623-630.
7. Liu, S. -K. 1977. *Vet. Clin. North Am.* 7: 323-339.
8. Matsuo, N., Oshima, M., Naganuma, M., Shimizu, K., Okada, R., and Sperling, D. R. 1972. *Jpn. Heart J.* 13: 307-316.
9. McClure, R. C., Dallman, M. J., and Garrett, P. G. 1973. *Cat Anatomy*. Lea & Febiger, Philadelphia.
10. Naganuma, M. 1978. *Jpn. Circ. J.* 42: 1124-1130.
11. Netter, F. H. 1969. Anatomy. pp. 11-12. *In: Ciba Collection of Medical Illustration*, Vol. 5, (Netter, F. H., and Yonkman, F. H. eds.), Ciba Pharmaceutical Co., New York.
12. Netter, F. H. 1969. Embryology. pp. 126. *In: Ciba Collection of Medical Illustration*, Vol. 5, (Netter, F. H., and Yonkman, F. H. eds.), Ciba Pharmaceutical Co., New York.
13. Patterson, D. F. 1976. *Adv. Vet. Sci. Comp. Med.* 20: 1-37.
14. Watson, H. 1972. *Br. Heart J.* 34: 37-40.

要 約

猫の心臓右心室乳頭筋の異形成の1症例(短報): 宇根ユミ・谷本忠司・菅沼常德¹⁾・兵藤哲夫²⁾・代田欣二・野村靖夫(麻布大学獣医学部病理学講座, 放射線学講座¹⁾, 兵藤動物病院²⁾——右心不全を呈した7ヶ月齢の雄猫を病理学的に検索した。右心房および心室は高度に拡張し, 房室弁口は拡大していた。右心房内膜は, 不規則な肥厚と石灰化を示していた。右心室大乳頭筋は, 肥大伸張し, 腱索を欠いて直接, 三尖弁に付着していた。三尖弁弁帆は, 短く, 弁組織はよく分化していた。小乳頭筋は心室中隔のやや上方を起始部として中隔尖に付着し, 動脈下乳頭筋は伸張して中隔尖と角尖の間に直接付着していた。