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Verrucoid lesions of mitral valve in a dog with features of inflammatory myofibroblastic tumor

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Abstract

We report a mitral valve lesion detected at autopsy in a 9-year-old male German Shepherd dog suffering from mild mitral regurgitation. Gross examination of the heart showed exophitic, noncontiguous lesions involving the atrial aspect of both mitral leaflets. Microscopic evaluation of the mitral lesions disclosed a diffuse proliferation of myofibroblasts with little atypia, arranged loosely and rather randomly, within a myxoid stroma and associated with inflammatory cells identified as CD138+ plasma cells, CD68+ macrophages, and eosinophils. The myofibroblastic proliferation we describe is quite similar to previously described inflammatory myofibroblastic tumor (IMT), a very rare lesion, exceptionally found in the human heart.

Keywords: Dog; Mitral valve; Cardiac tumor; Inflammatory myofibroblastic tumor

We report a mitral valve lesion detected at autopsy in a 9-year-old male German Shepherd dog suffering from mild mitral regurgitation. The dog was humanely euthanized because of an extensive chondroblastic osteosarcoma of the right radius. Gross examination of the heart showed exophitic, noncontiguous lesions involving the atrial aspect of both mitral leaflets, of 0.9 cm maximum diameter (Fig. 1A). No other cardiac abnormality was found. Microscopic evaluation of the mitral lesions disclosed a diffuse proliferation of myofibroblasts (smooth muscle actin and vimentin immunoreactive, negative for desmin, S100 and cytokeratin AE1/AE3) with little atypia, arranged loosely and rather randomly, within a myxoid stroma and associated with inflammatory cells identified as CD 13 8+ plasma cells, CD68+ macrophages and eosinophils (Fig. 1B,C). The exophitic mitral valve lesions were completely covered by hyperplastic endocardial cells without erosions. Stains for microorganisms including bacilli and fungi (PAS, Grocott, Warthin-Starry) were not contributory. Mitral valve between lesions did not show any relevant microscopic alteration. The myofibroblastic proliferation we describe is quite similar to previously described inflammatory myofibroblastic tumor (IMT), a very rare lesion, exceptionally found in the human heart with possible involvement of the mitral valve [1] and, so far, never described in canine heart. Only a splenic inflammatory pseudotumor has been previously described in a 12-year-old dog [2]. We excluded other neoplastic (i.e., myxoma, fibrona, fibrosarcoma), inflammatory (bacterial endocarditis), or degenerative (myxomatous mitral valve disease) lesions because of the presence of large spindled myofibroblasts with bland atypias, inflammatory cells, and irregular vascular network. Differential diagnosis with papillary fibroelastoma and healed endocarditis was also based on the absence of papillary fronds and elastosis of valve erosion, fibrous thickening, and calcifications, respectively.

In humans, IMT may present with an inflammatory syndrome, be associated with malignant tumors of other sites [3], be asymptomatic, or cause syncope and sudden death [4]. Gross features include polypoid aspect, vertucoid mass, or multifocal noncontiguous lesions. Also, histology may have variable (predominantly myxoid, inflammatory or fibrous) pattern. In the present case, the myofibroblastic lesion was associated with a malignant bone tumor, but the relationship between the valve tumor and the osteosarcoma of the radius is unknown.

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Fig. 1. (A) Gross morphology of dog mitral valve. The irregular, verucoid proliferations involve the atrial aspect of both mitral leaflets; no erosion is evident (bar=1 cm). (B) Microscopic view of spindle cell proliferation, associated with inflammatory cells in the anterior mitral leaflet (hematoxylin-eosin staining). (C) Immunohistochemical staining (immu-noperoxidase, hematoxylin counterstaining) shows smooth muscle actin-positive myofibroblasts.

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