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Letter to the Editor

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Dear Editor,

We thank the authors for their interest in this report of feline biventricular noncompaction. Indeed, the notable features of this case—the striking hypertrabeculation that characterized noncompacted regions of left and right ventricular walls, histologic features consistent with hypertrophic cardiomyopathy (i.e., myofiber disarray), and the genotype of this Maine coon cat—are consistent with studies reporting that individual sarcomeric mutations can result in different and multiple phenotypic expressions, and we agree with the authors that these findings are important.[1,2]

However, we depart sharply from speculation suggesting that ventricular hypertrophy may exaggerate the deep intertrabecular recesses and believe that several key points made in this report were overlooked. Firstly, as was detailed, there was no left ventricular hypertrophy, a finding confirmed by detailed microscopic examination and measurements from the sectioned, gross cardiac specimen. Microscopically in both ventricles and grossly in the left ventricle, there were obvious distinctions between the noncompacted inner portion of the ventricular wall caused by numerous finger-like muscular projections into the ventricular lumen and the normal, compacted outer portion of the ventricular walls. Furthermore, we reported that the heart weight was within normal reference range, opposite of what would occur with left ventricular hypertrophy. Taken together, these facts eliminate the possibility of making a diagnosis of hypertrophic cardiomyopathy, as no gross phenotypic features supported ventricular hypertrophy. Moreover, left ventricular hypertrophy has not been reported to exaggerate fine, finger-like wall projections that characterize noncompaction.[3] As the authors should be aware, the feline left ventricular endocardial surface is minimally trabeculated. Those trabeculae that may be present are coarse and broad-based and do not permit color flow Doppler echocardiography to display blood flow between them, in contrast to verification of blood flowing between the fine, finger-like noncompacted layer of this cat, as is also demonstrated in affected human patients. Thirdly, there was a markedly distinctive noncompacted right ventricular wall evident microscopically, and without evidence of right ventricular hypertrophy.

We agree with the authors that echocardiographic findings alone can be misleading or insensitive, and the present report made a particular point to emphasize the gross and histopathologic features of this case. They demonstrate distinctively different noncompacted and compacted layers of right and left ventricular myocardium, a feature heretofore unreported in the spectrum of feline myocardial diseases, and these findings were also supported by echocardiography.[4–6] Collectively, they fit consistently with contemporary criteria to diagnose noncompaction in human patients, even in keeping with varied criteria that have been proposed to promote diagnosis.

The present case of biventricular noncompaction demonstrates remarkably similar features to those reported in affected human patients. Future studies should help expand these perspectives.

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