HYPERTROPHIC CARDIOMYOPATHY IN PARMA WALLABIES *(NOTAMACROPUS PARMA).*

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Introduction

Macropodidae (e.g., kangaroos, wallabies etc.) are increasingly common in UK zoos, however scientific literature describing cardiomyopathies in this family is scarce. Reported conditions include:

- Toxoplasmosis in red kangaroos (*Osphranter rufus*) and Bennett's wallabies (*Notamacropus rufogriseus*)
- Takotsubo-like cardiomyopathy in a Bennett's wallaby
- Primary hypertrophic cardiomyopathy (HCM) in an Australian rock kangaroo (*Macropus robustus*), a Matschie's tree-kangaroo (*Dendrolagus matschiei*), and two Bennett's wallabies

Specific aetiologies for primary HCM in these species are unknown; genetic factors likely influence the condition (e.g., like MYBPC3 genetic mutations in Ragdoll and Maine Coon cats).

Parma wallabies are a near-threatened species, and research into their predisposed conditions is vital to their conservation. To date, HCM in Parma wallabies has not been described.

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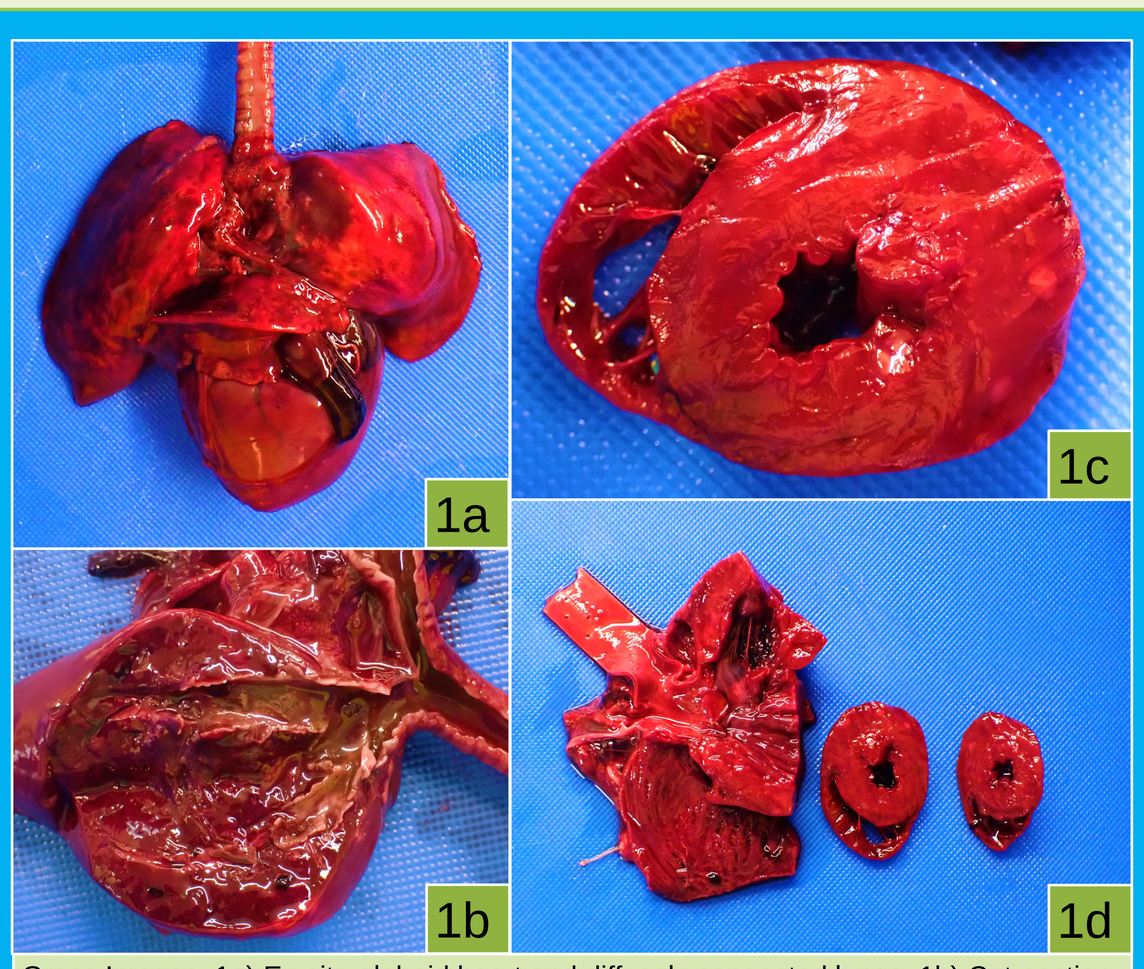
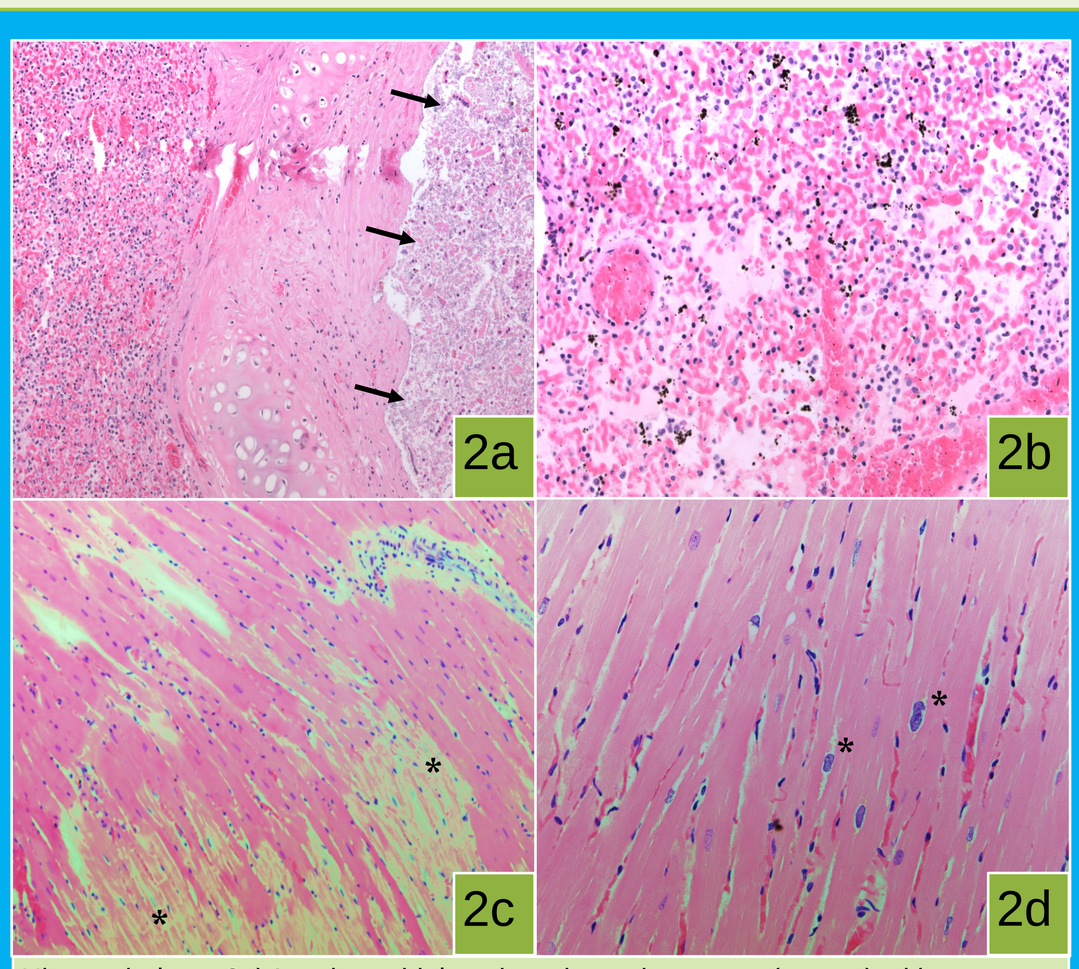
Materials & Methods

Two adult male Parma wallabies from two separate herds were submitted to IZVG pathology for full post-mortem examination between 2020 and 2023. Both died spontaneously without detectable clinical signs of heart failure.



Gross Images: 1a) Ex-situ globoid heart and diffusely congested lungs. 1b) Cut-section of lung with diffuse pulmonary congestion and oedema. 1c) Transverse LV-IVS-RV section demonstrating an LV-to-RV ratio of 6:1 (>3.5:1 = left ventricular hypertrophy and circular foci of myocardial pallor. 1d) Dissected heart with two transverse sections through heart ventricles with left sided hypertrophy.



Histopathology: 2a) Intrabronchial aspirated gastric content (arrows) without associated luminal inflammation, lungs (HE, 40x). 2b) Haemosiderin-pigment and laden macrophages scattered throughout the parenchyma, lung (HE, 200x). 2c) Multifocal cardiomyocyte disarray and associated degeneration (asterisks), left cardiac ventricle (HE, 100x). 2d) Hypertrophy-related cardiomyocyte anisokaryosis (asterisks) and anisocytosis, left cardiac ventricle (HE, 400x).

Results

Gross examination:

- Globoid hearts, with marked concentric left ventricular hypertrophy and marked diffuse pulmonary congestion with oedema in both cases.
- The second case also exhibited agonal aspiration of gastric content and multifocal, small (1-3mm) diameter circular areas of LV myocardial pallor.
- Heart wall measurements were LV-12mm, IVS-11mm and RV-2mm, with an LV-to-RV ratio of 6:1 (>3.5, indicating left ventricular hypertrophy).
- Luminal diameters were LV-11mm and RV-8mm.

Histological examination:

- Cardiac lesions were histologically limited to mild cardiac myofibre disarray and moderate myofibre hypertrophy (anisokaryosis/anisocytosis).
- Pulmonary lesions included bronchioloalveolar oedema with minimal to mild accumulations of haemosiderin-laden macrophages ("heart-failure cells").

Conclusions

- A specific aetiology for HCM was not determined in either case. Concurrent diseases (e.g., bilateral thyroid hyperplasia, or aortic atherosclerosis) were notably absent.
- The gross and histologic changes in both specimens are consistent with primary (idiopathic) HCM, as previously described in other macropods.

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