

PostScript

CORRESPONDENCE

Cystic atrioventricular node tumour: not a mesothelioma

Cystic tumours of the atrioventricular node (sometimes called "benign mesotheliomas of the atrioventricular node") are rare lesions associated with sudden cardiac death.^{1,2} A recent case provided several informative points regarding the nature of this lesion and best practice methodology within the coroner's necropsy.

A 31 year old man, training for a marathon, collapsed and died suddenly without previous medical history. No system, including the heart, revealed pathology on macroscopic examination at necropsy. Toxicology and non-cardiac tissue histology were negative. However, tissue sampling of the cardiac conduction system revealed an 11 mm diameter cystic tumour of the atrioventricular node that blended into the nodal tissue and proximal His bundle (fig 1). Immunohistochemistry showed no reactivity with standard mesothelial markers (calretinin, thrombomodulin, and Wilms' tumour 1), although reactivity for pan-keratin (AE1/AE3), cytokeratin 5/6 (CK5/6), cytokeratin 7, epithelial membrane antigen, BerEp4, and carcinoembryonic antigen was noted. Mucin histochemistry, and staining for p53, Bcl-2, cyclin D1, CK20, and thyroid transcription factor 1 was negative. Mib-1 (Ki-67) staining showed minimal proliferation (2%). Previously reported immunohistochemistry studies showed no reactivity with HBME-1 and antibody to factor VIII receptor antigen, and positive staining for secretory component and CA19.9.^{3,4}

The immunohistochemistry profile appears to support epithelial differentiation, and designation of this tumour as a form of

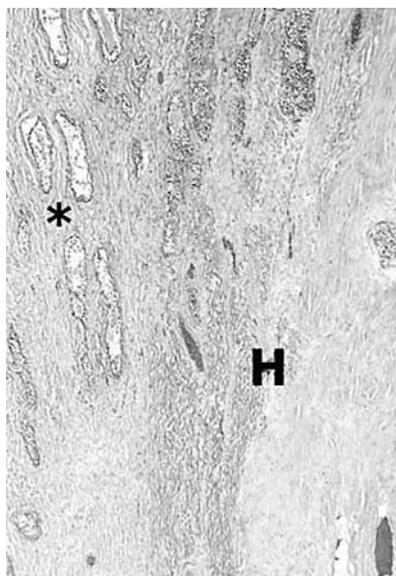


Figure 1 Irregular glands (*) are seen running into the His bundle (H) tissue (original magnification, $\times 200$; haematoxylin and eosin stain).

endodermal heterotopia. This probably reflects embryonic developmental factors, with differentiation towards an upper foregut phenotype. Certainly, these results provide confirmation that the lesion should not be regarded as mesothelial. Furthermore, immunohistochemistry could also argue against a

Learning points

- The atrioventricular node tumour shows no evidence of mesotheliomatous differentiation
- The atrioventricular node tumour may have no macroscopic clues to its presence
- Detailed conduction system sampling is possible from largely unfixed cardiac tissues within 24 hours of necropsy, thereby satisfying the coroner's needs

neoplastic aetiology, thereby supporting the concept of a congenital tissue rest, capable of slow proliferation.

The precise incidence of this tumour cannot be determined given the infrequency of detailed examination of the cardiac conduction system at necropsy (in patients with and without cardiac disease) and the lack of macroscopic clues to its presence. Cases of sudden cardiac death have shown that this tumour is associated with fatal cardiac dysrhythmia, although partial/heart block has also been reported.^{1,3} Patients with a more atrial based site appear to have a better presentation with heart block,^{3,5} and in rare cases there is the possibility of surgical ablation.³ This case presented with sudden death, probably as a consequence of a re-entrant tachycardia/ventricular fibrillation. No preceding syncopal history was derived from relatives.

Investigation of these cases usually requires careful cardiac tissue sampling.¹ Recent adverse publicity with regard to the necropsy has disinclined some pathologists from carrying out postmortem histological investigations. In this case, the standard tissue blocks around the atrioventricular node, sinoatrial node, His bundle, and regional samples from both ventricles were taken after transfer of the heart to our centre from the primary hospital. The residual cardiac tissues were then returned, within 24 hours, to be reunited with the body, allowing for appropriate funeral arrangements without delay. Despite the perceived complexity of dissection of the conduction system, it is possible to sample tissues appropriately without creating problems for the coroner or relatives. Indeed, failure to take adequate histology will automatically mean the failure to make a vital diagnosis of relevance to an inquest.

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Permission was given for this case to be reported

References

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- 2 Burke AP, Virmani R. Tumors and tumor-like conditions in the heart. In: Silver MD, Gotlieb AI, Schoen FJ, eds. *Cardiovascular pathology*. New York: Churchill Livingstone, 2001:585-6.
- 3 Paniagua JR, Sadaba JR, Davidson LA, et al. Cystic tumour of the atrioventricular nodal region: report of a case successfully treated with surgery. *Heart* 2000;**83**:E6.
- 4 Arai T, Kurashima C, Wada S, et al. Histological evidence for cell proliferation activity in cystic tumor (endodermal heterotopia) of the atrioventricular node. *Pathol Int* 1998;**48**:917-23.
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BOOK REVIEW

The Pathology of Human Viral Infections and Associated Conditions

Authored by J F Boyd. Glasgow: Published by University of Glasgow Press, 2004, £150.00 (softback), pp 2374. ISBN 0852616414

This very large work describes the pathology of human virus infections and associated conditions in three volumes, with a total of 2360 pages. Section A (most of volume 1) contains a comprehensive description of general pathology, including descriptions of viral structure and replication etc., clinical aspects, gross pathology, histopathology, and inflammation. Section B deals comprehensively with a long list of human (and some animal) viruses and describes the current knowledge of these in the context of human infection, and with a focus on pathology.

It is fascinating to contemplate the various angles from which the pathogen-host interaction can be viewed. Most commonly, it is viewed from the angle of the individual viral agent, and so this interaction is routinely described in text books on virology, which often include descriptions of the effect of a virus on an individual tissue. But the present approach is refreshing and rightly reminds us that the virus is only one side of the equation, and it is in the actual pathology that the pathogen-host interaction can be most clearly seen.

Advantages of the book are its unique focus on the pathology of virus infection. It is extremely comprehensive and will be very useful for reference. The disadvantages are that it is soft bound and produced in black and white. This is unfortunate because the figures do not appear in photographic quality. Being a microbiologist/virologist, I would not find this a problem, but I imagine that histopathologists may wish for better graphics.

The strength of this three book set appears to me to be its novel approach to the topic of pathology of human virus infection. Although its attractiveness to a general audience will be affected by its sheer breadth, I can see it finding a niche among virologists and pathologists with an interest in virus infection.

J R Kerr