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. 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, thymomodulin, and Wilms’ tumour 1), although reactivity for pan-keratin (AE1AE3), cytokeratin 5/6 (CK5/6), cytokeratin 7, epithelial membrane antigen, BerEp4, and carcinoembryonic antigen was noted. Much 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.

The immunohistochemistry profile appears to support epithelial differentiation, and designation of this tumour as a form of 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. Patients with a more atrial based site appear to have a better prognosis.

Investigation of these cases usually requires careful cardiac tissue sampling. Recent adverse publicity with regard to the necropsy has disinclined some pathologists to supporting the non-cardiac tissue histology were negative. 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, thymomodulin, and Wilms’ tumour 1), although reactivity for pan-keratin (AE1AE3), cytokeratin 5/6 (CK5/6), cytokeratin 7, epithelial membrane antigen, BerEp4, and carcinoembryonic antigen was noted. Much 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.

The immunohistochemistry profile appears to support epithelial differentiation, and designation of this tumour as a form of 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. Patients with a more atrial based site appear to have a better prognosis.

Investigation of these cases usually requires careful cardiac tissue sampling. Recent adverse publicity with regard to the necropsy has disinclined some pathologists to supporting the non-cardiac tissue histology were negative. 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, thymomodulin, and Wilms’ tumour 1), although reactivity for pan-keratin (AE1AE3), cytokeratin 5/6 (CK5/6), cytokeratin 7, epithelial membrane antigen, BerEp4, and carcinoembryonic antigen was noted. Much 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.

The immunohistochemistry profile appears to support epithelial differentiation, and designation of this tumour as a form of 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. Patients with a more atrial based site appear to have a better prognosis.

Investigation of these cases usually requires careful cardiac tissue sampling. Recent adverse publicity with regard to the necropsy has disinclined some pathologists to supporting the non-cardiac tissue histology were negative. 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, thymomodulin, and Wilms’ tumour 1), although reactivity for pan-keratin (AE1AE3), cytokeratin 5/6 (CK5/6), cytokeratin 7, epithelial membrane antigen, BerEp4, and carcinoembryonic antigen was noted. Much 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.

The immunohistochemistry profile appears to support epithelial differentiation, and designation of this tumour as a form of 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. Patients with a more atrial based site appear to have a better prognosis.

Investigation of these cases usually requires careful cardiac tissue sampling. Recent adverse publicity with regard to the necropsy has disinclined some pathologists to supporting the non-cardiac tissue histology were negative. 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, thymomodulin, and Wilms’ tumour 1), although reactivity for pan-keratin (AE1AE3), cytokeratin 5/6 (CK5/6), cytokeratin 7, epithelial membrane antigen, BerEp4, and carcinoembryonic antigen was noted. Much 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.

The immunohistochemistry profile appears to support epithelial differentiation, and designation of this tumour as a form of 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. Patients with a more atrial based site appear to have a better prognosis.