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Case Report

Sudden death of a child from myocardial infarction due to arteritis of the left coronary trunk

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ABSTRACT

An eight-year-old Japanese boy developed abdominal pain, followed by convulsion and loss of consciousness. He was taken to an emergency room but could not be resuscitated. At autopsy, the left main coronary trunk (LMT) demonstrated an increase in caliber with severe luminal narrowing, and the left anterior descending branch (LAD) subsequent to the LMT showed severe stenosis. Microscopically, the intima of the LMT demonstrated severe fibrosis and infiltration of lymphocytes and histiocytes suggesting vasculitis, and the small lumen was occupied by a fresh thrombus. The LAD showed significant intimal thickening with strong lymphocytic inflammation at the edge of the thickening. The left ventricle showed widespread myocardial infarction in the recovery stage. There were no findings of atherosclerosis, vasculitis or fibrocellular changes in the ascending aorta or intravisceral arteries other than the LMT and the LAD under investigation. The increase in the caliber of the LMT and the limitation of arteritis to the LMT and the subsequent branch suggested Kawasaki disease (KD), but it was atypical that the patient had no clinical history consistent with KD. The present case showed no findings suggesting classical polyarteritis nodosa (cPAN) at the acute or scar stage in the other vessels being investigated, and cPAN in childhood is rare compared to KD. A nonspecific inflammatory reaction (single organ vasculitis, SOV) was also considered as a possible cause, but it is difficult to determine whether the cause of the coronary stenosis in the present case was SOV because the sampling of arteries was insufficient. If forensic pathologists make unusual findings suggesting vasculitis at autopsy, the collection of a sufficient number of vessels of various sizes is warranted.

1. Introduction

Coronary diseases in children are uncommon, but forensic pathologists should keep them in mind because the patients can suffer out-of-hospital cardiopulmonary arrest and the antemortem diagnosis is difficult [1]. We report here the sudden and unexpected death of a child from subacute myocardial infarction caused by stenosis of the left coronary main trunk. Marked inflammatory reactions of the intima and the media were observed in the left trunk, and the differential diagnosis of the coronary arteritis became an issue.

2. Case report

An eight-year-old Japanese boy began to feel unwell and developed abdominal pain in an after-school day care center. While lying down, he convulsed and lost consciousness. He was taken to an emergency room but could not be resuscitated. A month prior to this, he had complained of feeling ill, and had experienced one week of nausea and vomiting at that time. He had no clinical history suggesting Kawasaki disease (KD), e.g., eruptions with desquamation in the extremities, oropharyngeal reddening, conjunctivitis, or cervical lymphadenopathy. He died suddenly and unexpectedly during the after-school care service, and responsibility for management became a problem, so a forensic autopsy was ordered by legal authorities.

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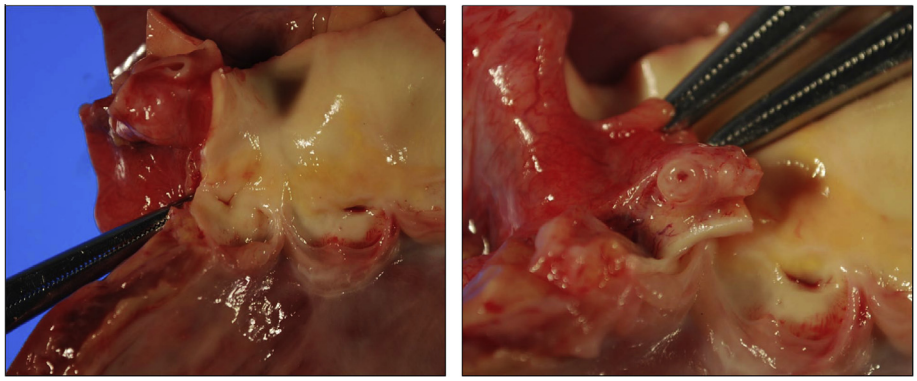

Fig. 1. The left main coronary trunk with an increase in diameter (6 × 5 mm) and severe luminal narrowing.

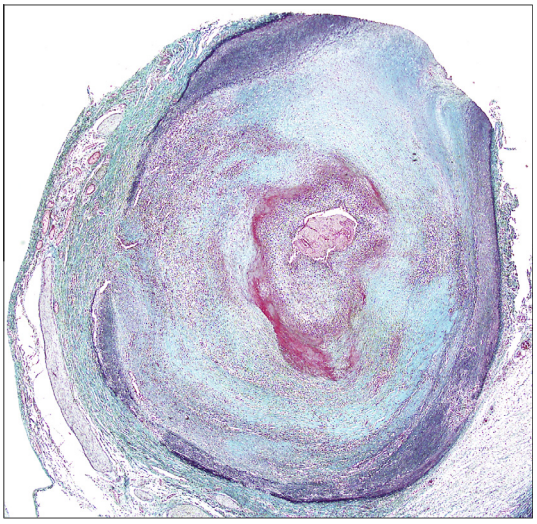

Fig. 2. Histological specimen of the left main coronary trunk. EM stain. 20×.

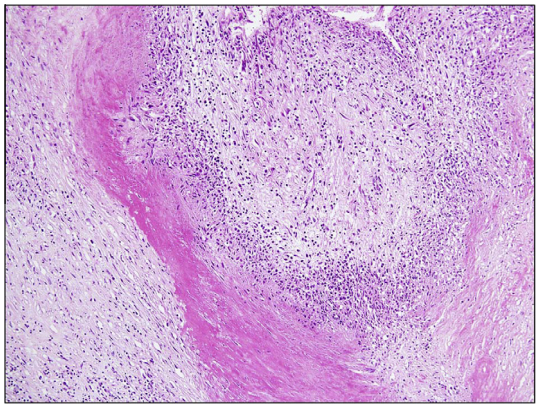

Fig. 3. Fibrous tissue and organized thrombus. HE stain. 100×.

Autopsy was performed approximately 24 h after the death. The boy was 128 cm tall and weighed 24 kg. Drug screening test using the Triage® Drugs of Abuse Panel was negative. The coronary system was left-dominant, and the left main coronary trunk (LMT) showed an increase in caliber (6 × 5 mm) with severe luminal narrowing (Fig. 1). Histologically, the LMT demonstrated severe annual-growth-ring-like intimal thickening with fibrosis, infiltration of lymphocytes and histiocytes, and an organizing thrombus (Figs. 2 and 3); the inflammation was severe at the subluminal layer, and a portion of the inflamed area formed a massive granulation tissue formation. The small lumen was occupied by a fresh thrombus consisting of platelets. The internal elastic lamina was partially disrupted. Lymphocytic and histiocytic inflammation was also seen around the vasa vasorum in the media, and focal disengagements of smooth muscle with fibrosis of the adventitia were also present. There were no fibrinoid necroses, neutrophilic infiltrations, eosinophilic infiltrations or multinuclear giant cells. Periodic Acid-Schiff (PAS) staining and Epstein-Barr virus-encoded small RNA in situ hybridization (EBER-ISH) of the LMT were negative. The left anterior descending branch (LAD) adjacent to the LMT showed significant intimal thickening with slight lymphocytic inflammation and disruption of the elastic laminae (Fig. 4).

There were no severe stenoses in any coronary branch other than those listed above (the numbers of histological specimens of coronary branches were as follows: one from the LMT, six from the LAD, five from the left circumflex branch, and 10 from the right coronary branch). Microscopically, slight intimal thickening with duplication of elastic laminae without active inflammation or scar formation was noted, but it was considered insignificant.

The subject's heart weighed 180 g and appeared oval. The left ventricle was widely dilated and showed widespread myocardial infarction, which was distributed over the anteroseptal wall at the basal side and the posterolateral wall at the apical side (Fig. 5). Microscopically, the myocardial infarction was at the recovery stage, showing granulations with collagen synthesis, recanalizations, and infiltrations of inflammatory cells consisting mainly of lymphocytes and histiocytes (Fig. 6). In some areas, there were foci of necrotic myocardium surrounded by large numbers of histiocytes and lymphocytes. Fresh bleeding was scattered.

The aorta showed no macroscopic intimal thickening or stenosis, and there were no findings of vasculitis or fibrocellular changes in the ascending aorta, brain, lung, liver, kidney, adrenal gland, spleen or pancreas. (Thoracic descending and abdominal aorta, extravisceral arteries including axillary, celiac, superior mesenteric, common iliac, pulmonary, carotid, intercostal, renal and testicular arteries, and intravisceral arteries of the skin, tongue, lung, testis, salivary gland, intestine and gallbladder were not sampled.)

3. Discussion

In the present case, the lesion in the left coronary trunk and the subsequent LAD had the appearance of vasculitis. The vasculitis seemed to be limited to the left main trunk and the adjacent left descending branch, with no involvement of the other branches of the coronary arteries or vessels in the other organs investigated.