Chronic Perforation of the Aortic Arch by Kirschner Wires

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Case Report

A 70-year-old woman sought medical care in our hospital due to dizziness, weakness of the right arm, which occurred suddenly and resolved completely within 4 hours. She reported similar symptoms approximately 9 months ago which also resolved spontaneously within 3 days. The physical examination showed no pathological findings except for a dysdiadochokinesia and slight impairment of fine motor skills.

Due to her history of breast cancer, cerebral metastases were initially suspected. However, this differential diagnosis was subsequently excluded based on the results of head magnetic resonance imaging (MRI) and lumbar puncture. Instead of neoplasia, the head MRI revealed minuscule subacute infarcts located at the left hemisphere and the parietal lobe, which potentially accounted for the symptoms.

Next, duplex ultrasound and computed tomography (CT) angiography of the neck and head as well as transesophageal echocardiography were performed to search for the sources of cerebral embolism. Duplex ultrasound showed minor arteriosclerotic changes at the carotid bifurcation. However, CT angiography detected two Kirschner wires penetrating the aortic arch. These wires were used to stabilize a right clavicle fracture 13 years ago. Since transesophageal echocardiography showed normal cardiac function and no atrial thrombi, we suppose that the intraluminal Kirschner wires most likely represent the source of the cerebral embolization.

►Fig. 1 shows the intraluminal location of the Kirschner wires in horizontal, sagittal, and frontal CT slices. Chest CT with contrast agent reveals completely normal ascending aorta and aortic arch without dissection or periaortic accumulation of fluid. There was also no pericardial or pleural effusion, no pneumothorax or hemothorax.

We decided against a surgical intervention for two reasons. First, the patient had no chest complaints and the perforation of the aorta had likely been well tolerated for many years. Second, in case thrombotic material or calcification had been formed on the wires occult to our diagnostic imaging, a removal of the wires could release them and cause further embolization.

Instead, the patient received anticoagulation with apixaban and was scheduled for follow-up every 6 months. Until now, the chest CT findings described earlier have not changed for more than 2 years and the patient remains without new symptoms.

►Fig. 2 presents the three-dimensional reconstruction of the chest CT which shows the spatial relation of the Kirschner wires to the aortic arch at their entry sites and the location and course of the wires at the right sternoclavicular joint.
Fig. 1  Intraluminal location of two Kirschner wires as shown in (A) frontal, (B) horizontal, and (C) sagittal computed tomography slides.

Fig. 2  Three-dimensional reconstruction of chest computed tomography. (A) Entry sites of the wires in the aortic arch. (B) The location and course of the wires at the right sternoclavicular joint.
Discussion

We present a case of Kirschner wires penetrating the aortic arch. The unusual setting in this case is the late occurrence of neurological symptoms and the lack of classic complications such as bleeding. This setting raises several questions and requires further discussion.

Although being rare, migration of Kirschner wires into the aorta, the right ventricle, or the trachea has been reported in the literature before. The common primary sites of the wires include the sternoclavicular joint, the clavicle, the acromioclavicular joint, although intracardiac migrations from more distal regions such as the pelvic bone, the femur, and the finger have also been reported.

To prevent migration, the Kirschner wires are usually bent at one end to form a J shape. In all cases where a migration was reported, the wires were either not bent or broken. A migration of the Kirschner wires in the present case is also possible. However, all wires were bent and are intact (Fig. 2B), and the location of the wires has been completely unchanged for more than 2 documented years. Hence, it is possible that the aortic arch was perforated during the reduction of the clavicle fracture and this complication was unnoted at the time of the initial procedure and had been well tolerated for the next 13 years. Alternatively, considering that the ascending aorta had a diameter of approximately 33 mm in the last CT scan, the wires could have only touched the aorta at the time of wire placement and the aorta’s pulsation and potential enlargement may have caused the penetration. In mice, internalization of foreign material into the aorta without hemorrhagic complications has been reported. Such a mechanism may also explain the late occurrence of neurological symptoms which may then have been associated with the actual time point of the wires penetrating the intima, releasing the embolic material.

Since perforations of chest organs or great vessels mostly result in severe complications, migrating wires are immediately removed in most cases. To our knowledge, this is the first report in which the penetration of the aorta by Kirschner wires only caused minor and transient neurological symptoms. Because the location of the wires has been unchanged for more than 2 years and the patient remains asymptomatic under therapy with apixaban, we suggest that a conservative approach in this case is preferable to surgical removal of the wires.

References