Recanalisation after coil embolisation of persistent ductus arteriosus

Toshihiro Ino, Kei Nishimoto, Mataichi Okubo, Keijiro Yabuta

Abstract
A 5 year old girl underwent recanalisation after coil embolisation of a persistent ductus arteriosus. Recanalisation is uncommon after coil embolisation and may be related to shrinkage of the coil, a change in its position, and ductal shape.

Keywords: persistent ductus arteriosus; coil embolisation; recanalisation

Transcatheter coil embolisation techniques have recently been applied to the occlusion of small to medium sized ductus with acceptable short and intermediate term follow up results.1–5 Long term results, however, remain uncertain. We report a rare case in which recanalisation of the ductus occurred after coil embolisation of a persistent ductus arteriosus and speculate on the relation between the recanalisation and a minor change in the coil position.

Case report
A 5 year old girl was admitted to our hospital for coil embolisation of a persistent ductus arteriosus. The patient had been doing well until a heart murmur had first been detected at 2 years of age. Her weight was 18 kg. Diagnostic catheterisation and angiography was performed before coil embolisation. At that time, pulmonary arterial pressure was 22/10 mm Hg and Qp/Qs was 2.0. An aortogram revealed a short, conical type of ductus with a narrowest width of 3.2 mm on the lateral projection. The aortic ampulla was 6.5 mm in diameter. A spring steel coil 8 mm in diameter and 10 cm long (Cook Corp, Bloomington, Texas, USA) was selected for occluding the ductus. We used a Jackson detachable system in which the delivery wire and coil were firmly connected by a screw shaped system. This system allows the coil to be detached by turning the delivery wire, as previously described.3,4 After the aortogram, a 5 F delivery catheter (Judkins coronary catheter, right) was advanced from the pulmonary trunk, across the ductus, and into the descending aorta. The coil was pushed out and positioned so that the four distal loops were in the aortic ampulla, and one loop was on the pulmonary side. The aortogram was repeated to confirm residual leakage before releasing the coil. A second aortogram 10 minutes after coil occlusion showed a trivial leak from the ductus (fig 1). The coil was then released and the delivery catheter was withdrawn. After the procedure, the patient was allowed to recover in the paediatric ward. Colour flow imaging performed the morning after
The procedure revealed neither residual leakage nor left pulmonary stenosis (fig 2). The heart murmur completely disappeared after the procedure.

The patient was discharged on the following day. Outpatient follow up examinations were performed one week, one month, three months, six months, and one year after the procedure. At one week, there was no evidence of residual leakage, left pulmonary stenosis, haemolysis or infection. The colour flow imaging study revealed no turbulent flow at the pulmonary end of the ductus. At one month, however, a trivial amount of turbulent flow was detected at the pulmonary end of the coil. This turbulent flow gradually increased during the follow up period. At one year, the flow peaked and was detectable from the pulmonary end of the coil to the pulmonary valve (fig 2C). Another coil embolisation was planned for the near future.

Quantitative measurement of the spring coil was performed on a lateral chest x ray to assess the relation between recanalisation and the coil position and configuration. The distance between the edge of the pulmonary loop and the aortic loops was measured, with reference to the angle between the long axis of the trachea and the coil. The coil had shrunk to approximately 75–80% of its original size during follow up, and the coil loops in the aortic ampulla were deviated anteriorly and superiorly.

Discussion
There is little information regarding recanalisation after coil embolisation of persistent ductus arteriosus, although spontaneous closure of residual leaks is common. Shim et al recently reported excellent results after coil embolisation using a Jackson detachable system; in their study only two of the 75 patients developed recanalisation after coil occlusion. To our knowledge, this phenomenon has not been reported in another patient until now although there was a report of recanalisation after coil embolisation of a cerebral artery. In our patient, colour flow imaging, which is more sensitive in detecting residual leakage than angiography, showed no residual shunt the next morning or one week after the procedure. At one month, however, trivial shunt flow was detected by routine colour flow imaging. We cannot definitely explain the cause of this recanalisation, but speculate that coil shrinkage, which is common in coil embolisation of persistent ductus arteriosus in our experience, may be involved. Experimental studies have previously demonstrated that coverage by neoendothelial cells usually starts a few days after coil implantation and is completed by three months. From this histological point of view, a minor change in the coil position and shrinkage of the coil might first occur because of pressure parallel to the long axis of the coil. Recanalisation might then develop at the site of thrombus formation without neoendothelial coverage, before neoendothelial coverage can be completed. In addition, the ductal shape may be related to the mechanism of recanalisation. The ductus in this patient was relatively short and was similar to the window type of ductus. In this setting, the coil can easily move, and the length of thrombus formed would be short and could be easily recanalised by blood flow.

In conclusion, we report a case of a 5 year old girl in whom recanalisation of a ductus developed one month after coil embolisation of persistent ductus arteriosus and became more evident after one year. This recanalisation is uncommon after coil embolisation and may be.

Figure 2  Colour flow imaging revealed a turbulent flow from the ductus to pulmonary trunk before coil occlusion (A), no evidence of residual leakage at one week (B) but clear evidence of recanalisation one year after embolisation (C).
related to shrinkage of the coil, a change in its positions and ductal shape.


### IMAGES IN CARDIOLOGY

**Pseudoaneurysm of the thoracic aorta presenting with angina: a late complication of aortic valve replacement**

A 52 year old man with aortic regurgitation caused by *Streptococcus mutans* endocarditis underwent aortic valve replacement with a 23 mm Medtronic Hall aortic valve prosthesis. Six years later he presented with exertional chest pain associated with a positive exercise test. A posteroanterior chest x-ray showed a soft tissue bulge at the right mediastinum.

Computed tomography (A) demonstrated a 13 cm pseudoaneurysm of the thoracic aorta. The lumen contains contrast medium but is largely obliterated by thrombus, and there is erosion of the sternum. At aortography, contrast was injected into the pseudoaneurysm demonstrating a narrow neck arising from the aorta in close proximity to the aortic valve prosthesis (B). Coronary angiography showed no evidence of intrinsic coronary artery disease; however, the proximal portion of the right coronary artery was compressed extrinsically by the pseudoaneurysm.

The patient underwent surgical repair of the pseudoaneurysm using extrathoracic bypass. The cause of the aneurysm appeared to be failure of the aortotomy suture line. Having mobilised the aneurysm sac the defect in the aortotomy suture line was repaired with buttressed sutures. Postoperative progress was uneventful and all antianginal treatment was discontinued. Four years later the patient remains well with normal effort tolerance.