

# Early neurological complications after transcatheter closure of atrial septal defect with nitinol wire mesh occluder

Wczesne powikłania neurologiczne po przezcewnikowym zamknięciu ubytku w przegrodzie międzyprzedsionkowej za pomocą okludera z siatki z drutu nitinolowego

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## Abstract

Atrial septal defect (ASD) was closed percutaneously in an 18-year-old boy with a 27 mm nitinol wire mesh occluder according to standard procedures. Three hours after the procedure, he presented anxiety attacks, aggression, a vacant stare and a verbal/speaking disorder. Small ischaemic stroke localised in the right temporal/parietal region of the central nervous system was confirmed by computed tomography examination. Activated partial thromboplastin time was then 54 s despite continuous heparin infusion. Heparin dose was increased and symptomatic treatment was introduced (mannitol, furosemide, propofol, haloperidol). The next day, all symptoms disappeared. He remained in good clinical condition, without neurological disorders 1.5 months after the procedure. Complications related to transcatheter ASD closure still exist despite the fulfillment of standard procedural criteria. Frequent coagulological examinations during and after the procedure are necessary. Close observation and follow-up of treated patients is mandatory.

**Key words:** transcatheter atrial septal defect closure, complications

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## INTRODUCTION

Atrial septal defect type II (ASD) is one of the commonest congenital heart defects [1]. Since the late 1990s, ASD closure with transcatheter techniques has progressively increased, becoming the treatment of choice. Many studies have demonstrated their efficacy and safety over the past 15 years, mostly with nitinol wire mesh Amplatzer Atrial Septal Occluder (ASO) [2–4]. Nevertheless there are still complications, although not frequent, such as thrombi formation and stroke, that deserve special attention [1, 5, 6]. We present the case of an adolescent male who presented ischaemic stroke early after ASD percutaneous closure with a nitinol wire mesh occluder.

## CASE REPORT

An 18-year-old male, weighing 86 kg, with ASD was admitted to our centre for transcatheter closure of the defect. On admission, he was in good general condition. Physical examination

revealed a 2/6 systolic murmur over 2–3 left sternal border and a fixed, split second heart sound. In ECG, we found sinus rhythm, right axis deviation and partial right bundle branch block. In transthoracic echocardiography (TTE), there was enlargement of the right atrium and ventricle, paradoxical movement of interventricular septum and ASD II 10 mm in diameter. The procedure in cathlab was performed under intravenous sedation (fentanyl i.v. 50 mg), through puncture of the right femoral vein. After placement of a 6 F sheath, 8,000 U of heparin were administered intravenously. The procedure was performed under fluoroscopic and transoesophageal echocardiography (TEE) guidance. TEE confirmed an 8 × 13 mm ASD. During the sizing procedure, using the stop flow technique with a 34 mm calibrating balloon (AGA Med., Plymouth, MN, USA), the stretched diameter was assessed as 25 mm, however another shunt through the second small defect (8 mm) was diagnosed. The distance between

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both defects was 7 mm. With the use of a long extra stiff wire, a calibrating balloon was exchanged on a 14 F transeptal Mullins sheath with its end placed in the left atrium. After backward flow through the side arm of the sheath (cleaning the inner lumen of possible air bubbles or clots), a 27 mm Figulla Flex ASD (Occlutech AB, Helsingborg, Sweden) nitinol wire mesh occluder was implanted. It is very similar to ASO. In implants from 24 to 33 mm sizes, the left atrial disc is 15 mm wider than the waist (in ASO: 14 mm). Because of the presence of the second defect, we decided to use an oversized device, to cover second fenestration. Implantation needed one reposition because of an initially oblique position of the device. Eventually, proper device position was confirmed by TEE before and after releasing of the device. A small residual shunt in the upper border of the device was however encountered. Fluoroscopy time was 4.1 min. Immediately after the procedure, constant infusion of heparin was administered: 300  $\mu$ /kg/24 h, which means 50  $\mu$ /kg/4 h. The patient remained conscious with logical communication. His activated partial thromboplastin time (APTT) result immediately after the procedure was 140 s. After 3 h, already in the cardiological ward, he presented anxiety attacks, aggression, a vacant stare and a verbal/speaking disorder. At that time a blood sample for APTT was taken (result 54 s) and additionally he received 4,000 U of heparin. APTT increased to 130 s. Because of the strong anxiety, propofol i.v. and haloperidol were administered. The brain computed tomography (CT) pictures suggested early ischaemic lesions in the right temporal/parietal region of the central nervous system. Neurological examination was difficult because of sedation, but generally no motoric changes were diagnosed, only trivial symptoms of central damage of VII scullar nerve. After consultation with the neurologist, heparin infusion was stopped, and in its place 0.4 clexan and 150 mg acetylic acid were administered as well as mannitol and furosemide. Sedation with propofol was continued overnight. On the next day, sedation was stopped. The patient was completely conscious, quiet, in a good general condition, without neurologic disorders. After 72 h of therapy with mannitol and furosemide, angio-CT was repeated. It revealed a tiny iso-hypodensive focus in the temporal region, suggesting ischaemic lesion. Repeated TEE examination was normal. No neurological disorders were found during physical examination. At that point, aspirin 150 mg and clopidogrel 75 mg were introduced. Despite platelet function, a blood coagulogram was within normal ranges. The patient was discharged home after eight days. He remained in good clinical condition, without neurological disorders, one and half months after the procedure.

## DISCUSSION

Closure of ASD with transcatheter technique devices is a safe and efficient treatment. Despite its widespread acceptance, there are associated risks and complications such as device migration or malposition, erosion, cardiac tamponade, atrio-

ventricular block, and embolism deriving from neurological disorders, that demand our attention [1, 4–8]. Most of the papers describing complications after percutaneous ASD have been related to the application of ASO, because this device has been used more frequently. The commonest presentation of ASD is as a single defect that can vary in size, but double secundum atrial defects are also encountered [1]. In such cases, different strategies can be used. Based on our experience in this matter, we chose the application of one device to close both fenestrations [9]. In our patient, the distance between ASD was 7 mm. Because of this, we applied a slightly overestimated device (namely a 27 mm Figulla ASD occluder). Its left atrium retention disc is of 7.5 mm. Small residual shunt persisted through second ASD immediately after the procedure. According to our previous observations, such shunts tend to close during one year follow-up [9]. The explanation of this phenomenon is not clear. It is probably related extending with time of the nitinol wire device waist or covering of the fenestration by endothelium.

There have not been many publications about neurological disorders following ASD transcatheter closure; most of them were transient and poorly documented. The procedure of our patient was realised in standard fashion [10] and the procedural coagulation blood tests were correct. Our surprise was caused by the lowered value of APTT observed 3 h after the closure (despite standard therapy with heparin), when the appearance of neurological symptoms started. Our procedural anticoagulation protocol is even more aggressive than that presented by Majunke et al. [10]. Described neurological disorders after device implantation have included migraine, visual disturbances or transient ischaemic attack [1, 4–8]. Our data documents stroke occurrence one month after the procedure of patent foramen ovale (PFO) closure with an Amplatzer PFO device [11]. It is possible that it occurred before complete endothelisation and could therefore have been secondary to an embolism from a device despite antiplatelet therapy. Thrombus formation during percutaneous ASD closure with Amplatzer occluder was documented in two patients despite periprocedural anticoagulation [12]. In the study by Du et al. [13], the risk of major complications with transcatheter device closure of ASD with an Ampaltzer device was 1.6% (7/442 cases) — including cerebral embolism with extremity numbness in one patient seven days after the procedure. In the study by Amin et al. [14], the risk of embolisation of ASO from 978 devices implanted was about 1% and the incidence of device erosion was 0.1%. They described three deaths — two related to heart perforation during catheterisation and the third (two days after the procedure) probably to do with arrhythmia.

There exist some small differences between nitinol wire mesh occluders such as Amplatzer and Occlutech Figulla ASD devices [15, 16]. On the other hand, Pac et al. [17] had very similar, positive results comparing percutaneous ASD closure

to ASO and Figulla Occluders. Some investigators have suggested that Occlutech Figulla occluders, although they have a low complication rate, do have a relatively high percentage of small residual shunts six months after percutaneous ASD and PFO closure [18].

In the case presented here, we were able to document in the CT a small ischaemic lesion a short time after percutaneous closure of ASD with no clinical sequels in follow-up. The echocardiograms performed during and after the procedure did not show additional complications such as thrombus formation, cardiac erosion or pericardial effusion.

### CONCLUSIONS

Complications related to transcatheter ASD closure still exist despite the fulfillment of standard procedural criteria. Frequent coagulological examinations during and after the procedure are necessary. Close observation and follow-up of treated patients is mandatory.

**Conflict of interest:** none declared

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