

A Rare Complication Indeed

To the Editor:

We read with interest the article "Platypnea-Orthodeoxia Syndrome: A Rare Complication After Right Pneumonectomy" by Bhattacharya and colleagues [1] in which they described a patient with platypnea-orthodeoxia syndrome after right-sided pneumonectomy. They treated the patient with an Amplatzer device to close the patent foramen ovale (PFO). With this device they achieved good symptomatic relief. Figure 1 in their article shows the device, but also an elevated right hemidiaphragm, in our opinion the source of the problem.

Platypnea-orthodeoxia syndrome is indeed rare; however, a PFO can occur in patients with right ventricular compression by an elevated right hemidiaphragm. Autopsy series have shown that the foramen can be opened in up to 30% of patients. The actual reopening of the foramen ovale in patients can be explained as follows: After resection of the right lung, a mediastinal shift occurs. The paralysed diaphragm opposes this shift, resulting in a rotation of the heart around the caval axis and an increased right atrial pressure. In one of our patients, during dynamic magnetic resonance imaging investigation, straining resulted in an increased rotation and increased shunting!

Closure of the PFO with a device stops the shunting, but not the atrial compression. Plication of the diaphragm resolves the interatrial pressure gradient and subsequently stops the blood flow through the PFO, and we have used this technique with good result.

In literature, treatment of interatrial shunting is still done by open heart surgery or, preferably, by percutaneous transcatheter closure [2–4]. We postulate that plication of the diaphragm corrects the cause and obviates the need for a cardiac operation. Spontaneous closure of the PFO after plication of the hemidiaphragm occurred in all patients [5].

Willem N. Welvaart, MD

Department of Surgery
Rivierenland Hospital
Pr. Kennedylaan 1
4002 WP Tiel, The Netherlands
e-mail: w.welvaart@zrt.nl

Koen J. Hartemink, MD
Marinus A. Paul, MD, PhD
Jan W.A. Oosterhuis, MD, PhD

Department of Surgery
VU Medical Center
Amsterdam, The Netherlands

References

1. Bhattacharya K, Birla R, Northridge D, Zamvar V. Platypnea-orthodeoxia syndrome: a rare complication following right pneumonectomy. *Ann Thorac Surg* 2009;88:2018–9.
2. Bellato V, Brusa S, Balazova J, Marescotti S, De Caria D, Bordone G. Platypnea-orthodeoxia syndrome in interatrial right to left shunt postpneumonectomy. *Minerva Anestesiol* 2008;74:271–5.
3. Marini C, Miniati M, Ambrosino N, et al. Dyspnoea and hypoxaemia after lung surgery: the role of interatrial right-to-left shunt. *Eur Respir J* 2006;28:174–81.
4. Godart F, Porte HL, Rey C, Lablanche JM, Wurtz A. Postpneumonectomy interatrial right-to-left shunt: successful percutaneous treatment. *Ann Thorac Surg* 1997;64:834–6.

5. Welvaart WN, Becker A, Marcus JT, Vonk-Noordegraaf A, Oosterhuis JW, Paul MA. Unusual treatment of patent foramen ovale after pneumonectomy. *Eur Respir J* 2010;35:929–31.

Metal Allergy, Atrial Septal Occluder Devices and the Risk of Kounis Syndrome

To the Editor:

In a very important article [1] published in *The Annals of Thoracic Surgery*, Khodaverdian and Jones reported a patient who had severe bronchospasm after implantation of an Amplatzer occluder device (AGA Medical Corp, Golden Valley, MN), in which the removal of the device was necessitated. The authors [1] emphasized that allergy to nickel can be documented as dermatitis or pericardial effusion. However, they did not refer to the "device syndrome" and the Kounis syndrome as a risk of nickel alloy implantation.

In a recent report, patients allergic to nickel, as evidenced by cutaneous patch skin tests and suffering from interatrial shunts, were implanted with the full nitinol Amplatzer occluder device (AGA Medical Corp) and the low nitinol Premere closure systems (St. Jude Medical Inc GLMT, St. Paul, MN). Eight of 9 patients experienced a number of symptoms, which included chest discomfort, exertional dyspnea, asthenia, palpitations, worsening of migraine headaches, and mild leukocytosis between postoperative days 2 and 3. Interestingly, in the same report, 2 patients with negative skin patch testing who had occluder system implantation had postoperative atrial fibrillation that was resolved with anti-arrhythmic treatment.

The previously described symptoms were similar to those of the Kounis syndrome [2], which was named the "device syndrome" [3]. When patients were treated with prednisone and clopidogrel, the symptoms resolved within 1 week of therapy.

In patients undergoing percutaneous atrial septal defect and patent foramen ovale closure with full nickel Amplatzer occluders (AGA Medical Corp), a nickel allergy has been the cause of systemic effects, such as chest discomfort, palpitation, and migraine headaches with or without aura [4]. The postulation was that a local allergic reaction to the implanted device could result in formation of platelet adhesions, which could then embolize in the heart and brain and cause chest discomfort and headaches.

The Kounis syndrome [5], which is similar to the symptoms of the "device syndrome," is defined as concurrence of acute coronary syndromes with conditions associated with mast cell activation involving interrelated and interacting inflammatory cells, including allergic or hypersensitivity, and anaphylactic or anaphylactoid insults. This is caused by inflammatory mediators such as histamine, neutral proteases, arachidonic acid products, platelet-activating factor and a variety of cytokines and chemokines released during the activation process.

There are two variants of the Kounis syndrome: (1) a type I variant that includes patients with normal coronary arteries without predisposing factors for coronary artery disease, and (2) a type II variant that includes patients with culprit but quiescent pre-existing atheromatous disease.

Metals are used in industrial productions involving jewelry, cosmetics, paints, leather, dyes, household products, personal adornments, pharmaceuticals, and medical devices, such as dental and orthopedic and cardiovascular implants. Metals are allergenic substances and hypersensitivity reactions to nickel occur in as much as 17.2% of the population. The metals are the most frequent cause of allergic contact dermatitis [6]. Allergic reactions to metallic implants have been incriminated for post-

operative complications, such as loosening or formation of new tissue around the metals [7].

The most severe complication of intracoronary stents is an intra-stent thrombosis, and the Kounis syndrome is regarded as its potential cause. Until now the majority of intracoronary stents were made from 316 L stainless steel, which contains nickel, chromium, and molybdenum. However, the new generation stents avoid nickel and use chromium and cobalt, which are less allergenic with fewer complications [8]. We suggest that the same should apply to all intracardiac devices.

Nicholas G. Kounis, MD, PhD
George N. Kounis, MD, MS
Constantinos Koutsojannis, PhD
Grigorios Tsigkas, MD
George Almpanis, MD
Andreas Mazarakis, MD, PhD

Medical Sciences, Patras Highest Institute of Education
and Technology

Department of Cardiology
University of Patras Medical School
Queen Olgas Square
7 Aratou St
Patras 26221, Greece
e-mail: ngkounis@otenet.gr

References

1. Khodaverdian RA, Jones KW. Metal allergy to Amplatzer occluder device presented as severe bronchospasm. *Ann Thorac Surg* 2009;88:2021-2.
2. Almpanis G, Tsigkas G, Kounis G, Kounis N. Nickel allergy, amplatzer atrial septal occluder device, and the risk of Kounis syndrome. *Clin Cardiol* 2010;33:117.
3. Rigatelli G, Cardaioli P, Giordan M, et al. Nickel allergy in interatrial shunt devicebased closure patients. *Congenit Heart Dis* 2007;2:416-20.
4. Wertman B, Azarbal B, Riedl M, Tobis J. Adverse events associated with nickel allergy in patients undergoing percutaneous atrial septal defect or patent foramen ovale closure. *J Am Coll Cardiol* 2006;47:1226-7.
5. Kounis NG. Kounis syndrome (Allergic angina and allergic myocatal infarction): a natural paradigm? *Int J Cardiol* 2006;110:7-14.
6. Oppei T, Schnuch A. The most frequent allergens in allergic contact dermatitis. *Dtsch Med Wochenschr* 2006;131:1584-9.
7. Hillen U, Haude M, Erbel R, Goos M. Evaluation of metal allergies in patients with coronary stents. *Contact Dermat* 2002;47:353-6.
8. Kedhi E, Joesoef KS, McFadden E, et al. Second-generation everolimus-eluting and paclitaxel-eluting stents in real-life practice (COMPARE): a randomised trial. *Lancet* 2010;375:201-9.

with subsequent blood flow obstruction, even if asymptomatic, raises several concerns.

Moderate hypothermic circulatory arrest with selective antegrade cerebral perfusion is becoming the technique of choice for AAD repair. When a carotid or innominate artery is involved, cardiopulmonary bypass using the axillary or femoral artery as a sole arterial cannulation site does not guarantee adequate perfusion of the affected cerebral hemisphere and thus exposes the patient to the risk of nonhomogeneous bilateral cooling of the brain. Second, although the primary aim of the surgical procedure is to replace the ascending aorta and, whenever possible, elimination or repair of the intimal tear, one should be assured that upon completion of the procedure, adequate blood flow to the culprit carotid artery is restored and sustained. Some have advocated the intraoperative deployment of stent devices inside the carotid artery for that purpose [2]. In the author's patient, the use of a preoperative bypass circuit from the femoral artery to the true lumen of the dissected carotid artery not only delays the surgical correction of AAD but also leaves the patient with the risk of subsequent postoperative malperfusion of the corresponding cerebral hemisphere.

Our attitude towards similar cases consists of operating on AAD even when the cerebral blood flow is compromised as soon as possible, although we admit avoiding cases involving comatose patients [3]. The first step of the operative procedure consists of exposing both the femoral and the dissected carotid artery at the same time. An 8-mm Dacron graft (DuPont, Wilmington, DE) is then anastomosed in an end-to-side fashion to the true lumen of the dissected carotid artery, and then cardiopulmonary bypass and systemic cooling is established immediately through the femoral and carotid artery through the Dacron graft. This ensures adequate and homogeneous cerebral perfusion and cooling during cardiopulmonary bypass. Once the aortic replacement is done under circulatory arrest, the Dacron graft connected to the true lumen of the dissected carotid artery is then grafted proximally to ascending aortic graft, thus guaranteeing sustained postoperative adequate perfusion of the brain.

We believe that this technique provides a solution to two major concerns: first, it quickly reestablishes blood flow to the affected cerebral territory, thus guaranteeing adequate cerebral hypothermia during CPB, and second, the Dacron bypass graft to the dissected carotid artery ensures adequate postoperative perfusion of the brain.

Ziad Khabbaz, MD

Service de Chirurgie Cardiovasculaire
Hotel-Dieu de France Hospital
Adib Ishaac St
Ashrafieh, Beirut, Lebanon
e-mail: bzkhazzab@hotmail.com

References

1. Munakata H, Okada K, Kano H, et al. Controlled earlier reperfusion for brain ischemia caused by acute type A aortic dissection. *Ann Thorac Surg* 2009;87:27-8.
2. Lentini S, Tancredi F, Benedetto F, Gaeta R. Type A aortic dissection involving the carotid arteries: carotid stenting during open aortic arch surgery. *Interact Cardiovasc Thorac Surg* 2009;8:157-9.
3. Fann JJ, Sarris GE, Miller DC, et al. Surgical management of acute aortic dissection complicated by stroke. *Circulation* 1989;80:257-63.

Preoperative Carotid Reperfusion in Type A Aortic Dissection: How Safe Is it?

To the Editor:

In the April 2009 issue of the *Annals of Thoracic Surgery*, Munakata and colleagues [1] describe a preoperative technique of controlled reperfusion of the brain suffering from ischemia secondary to acute type A aortic dissection (AAD) extending to the carotid artery. Although the decision to operate on comatose patients with AAD remains controversial, the technical challenge of dealing with AAD involving any of the carotid arteries