

avoided. One lead was implanted in the right atrium, 1 in the left ventricle and 1 for defibrillation in the right ventricular apex. The permanent pacemaker lead was left *in situ* with a silicone end-cap. No further incidents have been documented during patient follow-up.

In the cases presented, the mechanism underlying the induction of ventricular fibrillation could have been due to the continuous transmission—through the ventricular electrode to the interface with the myocardium—of the electrocautery radiofrequency pulses applied to the surrounding tissue or the pacemaker generator itself,² although long pulses were not applied and it was programmed in bipolar mode in both cases. The cases presented raise the issue of possible complications with the use of electrocautery, especially in patients with cardiovascular implantable electronic devices. Although the frequency of complications is low,² they are potentially fatal, and therefore a number of precautionary measures should be applied, including placing the dispersive pad as far from the generator as possible, reducing the energy pulses to less than 5 s' duration and, as a matter of course and in all cases, having continuous monitoring and advanced resuscitation equipment available during the procedure.³ Nevertheless, as long as the risk of bleeding is not especially high, a cold scalpel, with careful dissection followed by complete homeostasis, may be sufficient, thus completely avoiding the use of the electric scalpel in many of these interventions.

Marta Pachón, Miguel A. Arias,* Alberto Puchol, Miguel Jerez-Valero, and Luis Rodríguez-Padial

Unidad de Arritmias y Electrofisiología Cardíaca, Servicio de Cardiología, Hospital Virgen de la Salud, Toledo, Spain

*Corresponding author:

E-mail address: maapalomares@secardiologia.es (M.A. Arias).

Available online 17 July 2012

REFERENCES

1. Levine PA, Balady GJ, Lazar HL, Belott PH, Roberts AJ. Electrocautery and pacemakers: management of the paced patient subject to electrocautery. *Ann Thorac Surg.* 1986;41:313–7.
2. Lo R, Mittrache A, Quan W, Cohen TJ. Electrocautery-induced ventricular tachycardia and fibrillation during device implantation and explantation. *J Invasive Cardiol.* 2007;19:12–5.
3. Crossley GH, Poole JE, Rozner MA, Asirvatham SJ, Cheng A, Chung MK, et al. The Heart Rhythm Society (HRS)/American Society of Anesthesiologists (ASA) Expert Consensus Statement on the perioperative management of patients with implantable defibrillators, pacemakers and arrhythmia monitors: facilities and patient management. *Heart Rhythm.* 2011;8:1114–54.

<http://dx.doi.org/10.1016/j.rec.2012.04.008>

Cardiogenic Shock Secondary to Metamizole-induced Type II Kounis Syndrome

Shock cardiogénico secundario a síndrome de Kounis tipo II inducido por metamizol

To the Editor,

Although the first case was described more than 20 years ago, recent years have seen a marked increase in reports of cases of acute coronary syndrome in the context of allergic reactions, known as Kounis syndrome. Traditionally, 2 variants of the syndrome were reported: type I (due to coronary vasospasm), which occurs in patients with normal coronary arteries, and type II (due to coronary thrombosis) in patients with atherosclerosis.

This syndrome is triggered by the release of inflammatory mediators during mast cell degranulation, although these mediators are also increased in patients with acute coronary syndrome due to non-allergic causes.¹

Among the possible triggering factors of Kounis syndrome are hymenoptera stings, drugs, food, environmental exposure, and diverse conditions such as bronchial asthma, mastocytosis, etc. Any medication can cause this syndrome, but most cases have been reported in relation to beta-lactam antibiotics and non-steroidal anti-inflammatory drugs.

Although the pathophysiology of thrombosis of drug-eluting stents is multifactorial, a recent review by Chen et al.² states that Kounis syndrome can be one of the potential causes. The simultaneous use of drugs such as clopidogrel and acetylsalicylic acid in these patients may also act as a potential antigen. These findings have led to the recent proposal to create a new classification for Kounis syndrome that includes type III in relation to thrombosis of drug-eluting stents.³ There have also been reported cases of *tako-tsubo* cardiomyopathy associated with Kounis syndrome through the release of inflammatory mediators.⁴

We present the case of a 53-year-old woman who was receiving vildagliptin/metformin for type 2 diabetes mellitus and atorvastatin for hypercholesterolemia. She smoked 20 cigarettes a day, and had had an osteoporotic D12 vertebral fracture after physical effort 10 days previously, which was treated conservatively by fitting a corset brace and analgesia with paracetamol and ibuprofen. She went to the emergency department for intense back pain despite analgesic and anti-inflammatory treatment. Five minutes after receiving a vial of intravenous metamizole, she started to show generalized urticaria, chest pain, and intense dyspnea, accompanied by severe hypotension (systolic blood pressure: 70 mmHg). The initial electrocardiogram showed sinus tachycardia at 120 bpm with ST elevation in the anterior leads. Treatment began with volume replacement, steroids, and intravenous antihistamines. The patient was also administered 1 vial of subcutaneous adrenaline. Despite these measures, the patient remained in refractory shock, with worsening of the respiratory problems with progressive desaturation, requiring orotracheal intubation and mechanical ventilation. Noradrenaline and dobutamine perfusion was started and an intra-aortic balloon counterpulsation pump was fitted. The hemodynamics department was notified to perform an emergency cardiac catheterization, and the coronary angiography found thrombotic occlusion in the proximal portion of the anterior descending artery. A drug-eluting stent was implanted. An emergency echocardiogram showed antero-septo-apical hypokinesia, with a left ventricle ejection fraction of 35%. A Swan-Ganz catheter was inserted, which confirmed a low cardiac index and high peripheral vascular resistance, data that were compatible with cardiogenic shock. The serum creatine kinase and troponin T peak values were 2426 IU/L and 7.36 ng/mL, respectively. The patient's subsequent clinical course was satisfactory. Treatment with inotropic and vasopressor agents was gradually reduced until its withdrawal, while the intra-aortic balloon counterpulsation pump was removed after 48 h and the endotracheal tube after 3 days. Upon discharge, the patient received treatment

with beta-blockers, angiotensin-converting-enzyme inhibitors, aldosterone antagonists, dual antiplatelet therapy with acetylsalicylic acid and clopidogrel, and statins. An echocardiograph prior to discharge showed a left ventricle ejection fraction of 45%. In this syndrome, ventricular function typically returns to normal levels after several weeks. Our patient lived in another autonomous region and consequently we do not have this datum.

The prognosis of Kounis syndrome is generally good, although complications such as pulmonary edema, arrhythmia and thrombus have been reported during the acute phase. Presentation as cardiogenic shock is extremely rare and only 1 similar case report has been published previously.⁵

There are no clinical guidelines for treating Kounis syndrome at present. There are too few cases to be able to reach any definitive conclusions on the treatment of this syndrome but, in general, these patients require treatment with corticosteroids, antihistamines, and antithrombotic drugs. Treatment with adrenaline is controversial, as it can worsen ischemia, prolong the QT interval, and induce coronary vasospasm and arrhythmia, but in general it must be administered in the event of severe hypotension or cardiac arrest. Vasodilator agents, including nitrates and calcium channel blockers, must be considered as first-line therapy for previously healthy young patients. The acute coronary syndrome protocol should be followed in patients with the type II variant.⁶

Kounis syndrome encompasses a group of symptoms that is probably under-diagnosed and should be included in the differential diagnosis of cardiogenic shock.

Francisco J. Garcipérez de Vargas,* Javier Mendoza, Pablo Sánchez-Calderón, Carolina Ortiz, Rosa Porro, and María V. Mogollón-Jiménez

Servicio de Cardiología, Hospital San Pedro de Alcántara, Cáceres, Spain

*Corresponding author:

E-mail address: fj.garci@hotmail.com (F.J. Garcipérez de Vargas).

Available online 19 July 2012

REFERENCES

1. Biteker M. Current understanding of Kounis syndrome. *Expert Rev Clin Immunol*. 2010;6:777–88.
2. Chen JP, Hou D, Pendyala L, Goudevenos JA, Kounis NG. Drug-eluting stent thrombosis: the Kounis hypersensitivity-associated acute coronary syndrome revisited. *JACC Cardiovasc Interv*. 2009;2:583–93.
3. Biteker M. A new classification of Kounis Syndrome. *Int J Cardiol*. 2010;145:553.
4. Yanagawa Y, Nishi K, Tomiharu N, Kawaguchi T. A case of takotsubo cardiomyopathy associated with Kounis syndrome. *Int J Cardiol*. 2009;132:e65–7.
5. Tanboga IH, Karabay CY, Can MM, Akgün T, Güler A, Turkyılmaz E, et al. Kounis syndrome presenting with cardiogenic shock. *J Cardiovasc Med*. 2011;12:833–6.
6. Cevik C, Nugent K, Shome GP, Kounis NG. Treatment of Kounis syndrome. *Int J Cardiol*. 2010;143:223–6.

<http://dx.doi.org/10.1016/j.rec.2012.04.011>

Late Arterial Switch, Without Previous Preparation or Extracorporeal Membrane Oxygenation Back-up

Switch arterial tardío, sin preparación previa ni oxigenador de membrana extracorpórea de soporte

To the Editor,

Arterial switch is considered the treatment of choice for transposition of the great arteries (TGA). There is consensus that this procedure should be performed before 2 weeks of life, to prevent regression of the left ventricle (LV) in a subpulmonary position.¹ We report 3 cases of single TGA in which the intervention was performed beyond 14 days without prior preparation or extracorporeal membrane oxygenation (ECMO).

Case 1 was a child of 18 days admitted with cardiac arrest. TGA and *situs inversus* were diagnosed after cardiopulmonary resuscitation. An urgent Rashkind procedure was performed and, once stabilized, the child underwent surgery the following day.

Case 2 was a cyanotic boy referred from another hospital at 12 h of life and with prostaglandins. Hemodynamic status was poor, with severe ventricular dysfunction and 50% saturation. An urgent Rashkind procedure was performed after diagnosis of TGA. The child subsequently suffered *Klebsiella* pneumonia and sepsis as well as acute renal failure, forcing a delay in surgery until day 27 of life.

Case 3 was a 6-day-old boy who was referred from another hospital with low output and cyanosis. An urgent Rashkind procedure was performed. The child then suffered renal failure, which required peritoneal dialysis, and *Klebsiella* sepsis. On resolution of these problems, the patient underwent surgery at 28 days of life.

Most groups opt to correct TGA using the arterial switch before 2 weeks of life. There are anecdotal reports of the procedure being performed beyond 14 days, with the risk of left ventricular failure in the new systemic position.^{1,2} The two most common causes of this situation are late referral and contraindications for surgery.

Ultrasound criteria for subpulmonary LV exist³ and include paradoxical septal motion, reverse ventricular morphology (spherical right and left semilunar valves), and left free wall thinning, among others. Surgically, a simple measurement of pressures in both ventricles provides a right/left (LV/RV) relationship, with an arterial switch being inadvisable at a ratio <0.6.

Currently, the 3 main options proposed in this situation are: a) preparatory surgery using pulmonary cerclage and systemic-pulmonary fistula, followed by arterial switch in a second stage^{2,4}; b) arterial switch with ECMO support until ventricular remodeling occurs^{5,6}; c) discharge of the patient and atrial correction (Senning, Mustard) after a few months.⁶ *A priori*, the option of using ECMO as support (if necessary) seems the most appropriate.

A German team⁴ proposed a simple method based on the pressure ratio between the 2 ventricles after a 15–30 min trial period of pulmonary cerclage. After the trial period, the arterial switch is performed if the ratio is adequate (LV/RV > 0.6), or preparation via pulmonary cerclage and fistula is carried out followed by correction in a second stage. The work published by the Great Ormond Street Hospital almost 10 years ago and updated in 2004^{1,5} demonstrated similar survival rates in patients undergoing surgery between 2 weeks and 2 months, and indicated a greater need for inotropics and mechanical ventilation. Recently, another group published results in patients receiving surgery at 6 months,⁶ and showed higher than usual mortality, despite the use of ECMO. In the