Pseudoaneurysm of the Pulmonary Autograft Following the Ross Procedure

To the Editor,

The Ross procedure has emerged as a good alternative for aortic valve disease, particularly in children and young people in whom repair is not possible.[1] Although the procedure is safe, complications related to the pulmonary autograft have been described.[2] One of these complications has been reported on only two occasions,[3,4] but was potentially severe: pseudoaneurysm of the autograft.

A total of 200 patients have undergone the Ross procedure at our hospital; we present two who were asymptomatic and diagnosed with a false aneurysm of the pulmonary autograft at 2 years of follow-up, with a different cause in each case.

The first was a 27-year-old man who underwent the Ross procedure for severe postendocarditis aortic incompetence. He was admitted one month later for endocarditis on the autograft suture. The patient's progress was favorable following antibiotic therapy, with the pre-discharge echocardiogram showing that the endocardial vegetation had disappeared and both graft valves were functioning normally. The patient was followed as an outpatient, with 6-month echocardiographic controls. At 24 months postoperatively, the patient was asymptomatic; however, follow-up transthoracic echocardiography revealed severe aortic regurgitation due to dilation of the pulmonary autograft root. In addition, a pulsatile retroaortic outpouching with interior flow was seen at the autograft suture, consistent with a pseudoaneurysm (Figure 1). Ultimately, the pulmonary autograft was replaced with an aortic valve (Bono-Bentall) with good results.

The second case was a 37-year-old man who successfully underwent the Ross procedure for severe rheumatic aortic regurgitation. He received regular follow-up, with the two-year transthoracic echocardiogram showing a pulsatile retroaortic outpouching with interior flow, that was not present in the echocardiogram the previous year and was consistent with a pseudoaneurysm of the autograft. In addition, he had mild dilation of the aortic ring that caused mild aortic regurgitation. Transesophageal echocardiography disclosed

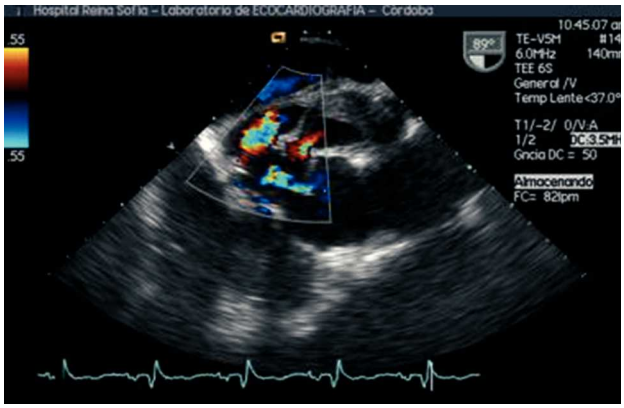

Figure 2. Transesophageal echocardiographic image at 90º, in which color Doppler reveals the shunt of the pseudoaneurysm with the left ventricular outflow tract through two points of entry.

the point of entry in the inferior suture of the autograft in the noncoronary sinus, with extension to the left coronary sinus, compromising the origin of the left coronary artery (Figure 2). Ultimately, the patient underwent surgery with aortic valve placement (Bono-Bentall).

The cause of pseudoaneurysm of the pulmonary autograft has been discussed in recent years. It has been attributed to structural weakness of the pulmonary autograft which, when exposed to systemic pressures, experiences progressive dilation and localized rupture, thereby originating the point of entry of the pseudoaneurysm.[4] In our first case, the pseudoaneurysm could be explained by the presence of postinfection friable tissue, whereas the other could be due to weakness of the pulmonary annulus, despite reinforcement via annuloplasty.

Since both pseudoaneurysms were retroaortic, a relatively inaccessible area for surgical control, we suggest monitoring by intraoperative transesophageal echocardiography. A review of our series showed that these 2 patients had no intraoperative study, and therefore this complication might have been avoided.

In conclusion, the 2 patients were diagnosed 2 years after the surgery, despite being asymptomatic, confirming that close clinical and echocardiographic follow-up is important. We believe that transthoracic echocardiography, because it is a

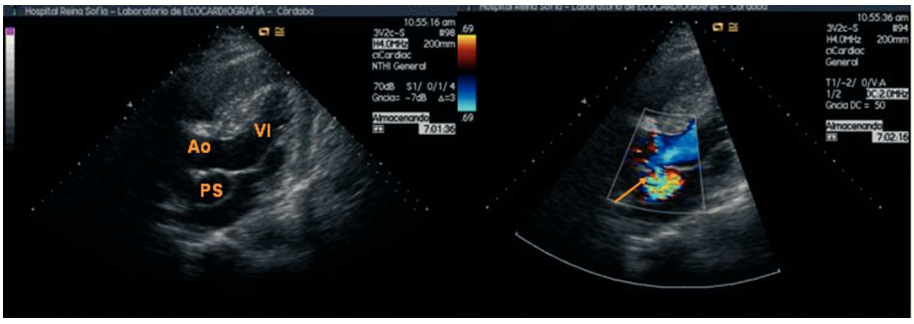

Figure 1. Two transthoracic echocardiography images in a longitudinal parasternal view. The first shows a pulsatile, retroaortic outpouching consistent with pseudoaneurysm; in the second, color Doppler ultrasound reveals flow through the point of entry (arrow). Ao indicates aorta; PS, pseudoaneurysm; LV, left ventricle.

simple, noninvasive technique readily available at most hospitals, would be sufficient for follow-up, whereas other techniques, such as transesophageal echocardiography, would be used to complete the diagnosis.

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Hemolytic Uremic Syndrome Due to Sirolimus in a Heart Transplant Recipient. Case Report

To the Editor,

Hemolytic uremic syndrome (HUS) is characterized by a triad consisting of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure. The exact pathophysiology is unknown. A number of theories have been proposed, and it is known that not all HUS cases are identical, with considerable heterogeneity in the clinical, analytical, and microscopic findings, since the mechanisms that produce it are heterogeneous.[1] It appears that the basic alteration is vascular endothelial injury with the release of factors that promote platelet aggregation. For example, the association of *Escherichia coli* with HUS is mediated by the production of a cytotoxin with a well-described mechanism of endothelial damage; this is not the case with drug-related HUS.

The most frequent cause among recipients of a solid organ transplant is cyclosporine. The second is an episode of acute graft rejection; this precedes the onset of HUS in 30%-50% of cases.[2]

We describe a 61-year-old man who had undergone heart transplant 13 years earlier and came to the hospital for overall poor condition and fever (38.5ºC) with no etiological focus. As a complication of heart transplantation, he had developed multiple epidermoid carcinomas of the skin, treated with brachytherapy and surgery. Because of continuous recurrence of the tumors, the immunosuppression was modified: cyclosporine was switched to sirolimus, mycophenolate mofetil dose was reduced, and corticoids were maintained. Fifteen days after the switch, the patient came to the hospital. The examination showed poor overall condition with hematomas on the trunk, and a normal cardiologic examination. Analytical results were as follows: creatinine level 5 mg/dL (previously, 2.5 mg/dL), thrombocytopenia at 71 000/µL, anemia with hemoglobin levels of 6.2 g/dL, schistocytes, and positive hemolytic markers (LDH, 650 mg/dL), normal coagulation, sirolimus concentration 10 ng/dL, and mycophenolic acid 2 ng/mL. The echocardiography, electrocardiogram, and chest x-ray were normal. The analytical findings led us to suspect HUS. Based on the absence of a previous infectious condition and any signs of rejection in the endocardial biopsy that could be triggers, and given the recent introduction of sirolimus, the latter was considered the etiological factor, with blood levels within the therapeutic range. Sirolimus was switched to cyclosporine, and mycophenolate mofetil and corticoids were maintained. Various sessions of plasmapheresis and packed red blood cell transfusions were performed. Progress was favorable and the patient remained afebrile, in good overall condition, and with improved kidney function (creatinine 2.6 mg/dL, hemoglobin 8.3 g/dL with disappearance of hemolysis markers, and platelet count of 250 000/µL).

Renal transplant recipients are at significant risk for recurrent HUS. Likewise, its association with cyclosporine is well-documented, and sirolimus has been used as rescue therapy following cyclosporine-induced HUS.[3] However, in recent years there have been reports of HUS cases in renal transplant recipients in relation to sirolimus alone or, even more frequently, to sirolimus plus cyclosporine.[3,4] There is little experience in cardiac transplant recipients, and the published cases are related to cyclosporine or tacrolimus.[2,5]

The diagnosis of a causal relationship between sirolimus and HUS is made by excluding other causes and by observing a proximity in time. In our patient, sirolimus concentrations were within the therapeutic range, as in the other reviewed cases,[3] and therefore, the damage does not appear attributable to overdose.

Sirolimus, a natural macrolide of *Actinomyces*, is an immunosuppressant with a potent antiproliferative effect that appears to decrease the development of secondary tumors,[6] which is the reason we used it in a patient with recurrent skin cancer.

In several published case studies of renal transplant recipients with sirolimus-related HUS, considerable clinical and analytical improvement is described, although without full recovery of kidney function; this is attributed to late diagnosis and treatment.[5] Our patient's kidney function did improve. However, in light of the experience in patients who have received other organs, we consider that the hemolysis markers should be carefully controlled when modifying the immunosuppression therapy, eg, when switching to sirolimus, in order to facilitate early diagnosis and treatment of HUS.

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Coronary Artery Disease and Percutaneous Coronary Intervention in a Set of Twins

To the Editor,

Both genetic and environmental factors are involved in the development of coronary disease (CD). The degree to which each particular risk factor contributes to the development of CD is not precisely known; in particular, the role of genetics, beyond its indirect role through the hereditary component of hypercholesterolemia, is still not clear. Twin studies could be helpful in researching the role of genetics in the development of CD.[1]

We describe 2 identical twins diagnosed with unstable angina, in both cases with right coronary artery disease, within an interval of 6 months, and both treated percutaneously with placement of a coronary stent.

The first case is a 49-year-old man who presented hypertension, smoking, and dyslipidemia as cardiovascular risk factors. The patient, who had no history of heart disease, was hospitalized with clinical symptoms of unstable angina, associated with T-wave negativity on the inferior aspect. Cardiac catheterization disclosed severe stenosis in the proximal segment of the right coronary artery (Figure 1A), with no significant angiographic stenosis in the remaining arteries. An ad hoc percutaneous coronary intervention was performed, with direct implantation of a 4×28-mm Taxus stent (Boston Sci), with good angiographic results and no complications (Figure 1B). At 6 months the patient was clinically asymptomatic and angiographic follow-up showed no restenosis (Figure 1C).

Six months after this patient was admitted, his identical twin brother (age 49) was hospitalized with symptoms of progressive angina. The patient presented the same cardiovascular risk factors (hypertension, smoking, and hyperlipidemia). The baseline electrocardiogram showed no specific repolarization abnormalities. Coronary angiography showed a severe lesion in the middle right coronary artery, but no significant lesions

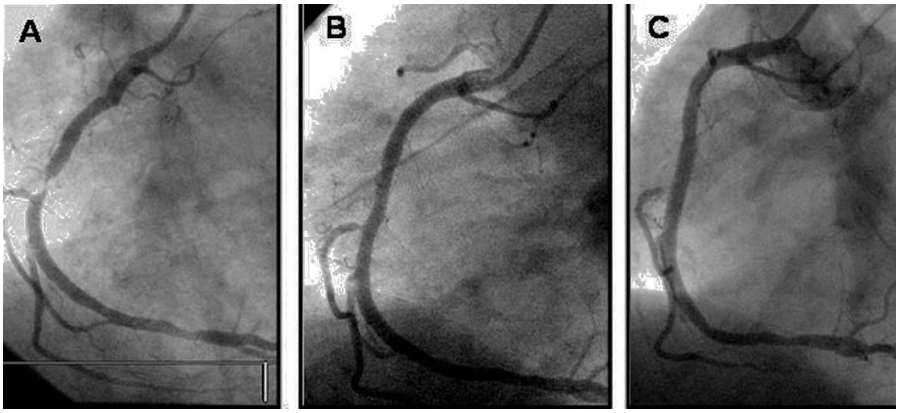

Figure 1. A: severe stenosis in the proximal segment of the right coronary artery. B: immediate direct postimplantation of a 4×28-mm Taxus *stent*. C: angiographic control at 6 months' follow-up.

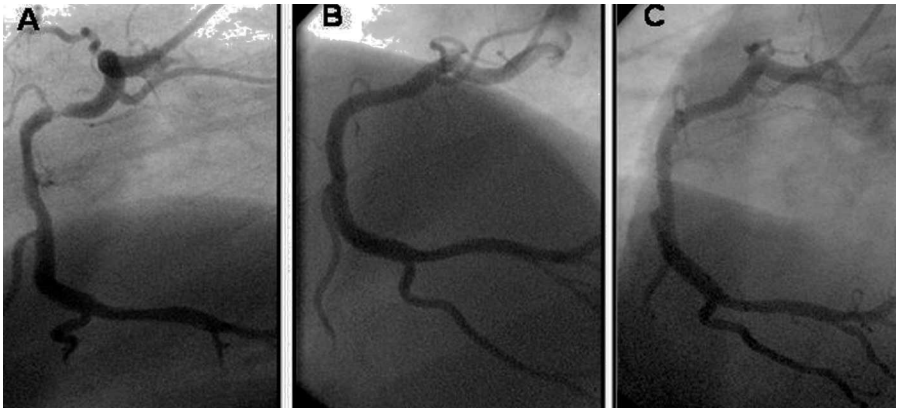

Figure 2. A: severe lesion in the middle right coronary artery. B: immediate direct postimplantation of a 4×15-mm Driver stent. C: angiographic control at 6 months' follow-up.

in the remaining coronary vasculature (Figure 2A). As occurred with the patient's brother, the right coronary artery was treated, in this case with a 4×15-mm bare metal stent (Driver, Medtronic Inc.) (Figure 2B). After six months' follow-up, this patient remained asymptomatic, with no restenosis in follow-up angiography (Figure 2C).

The earliest cardiologic studies in twins consisted of electrocardiogram analysis in 1925. By the late 20th century, studies have focused on CD, with considerable attention and debate regarding the genetic versus environmental factors in the pathogenesis of this condition.

It has been shown that twins are closely matched in terms of CD, although this effect decreases with age. It is estimated, for example, that the probability of dying due to CD in the next 10 years is 50% for a 55-year-old man if his twin has had a myocardial infarction.[2] In 1958, Benedict[3] described a set of twins with CD. His report mentioned several points of interest: young age (40 years), similar electrocardiogram changes, and development of coronary events with a small difference in time (6 months). At that time, this report was considered an argument in favor of the role of hereditary factors on the development of CD.[2,3] For example, a later study reported on a set of 50-year-old twins who presented with an acute coronary syndrome two years apart; one had an infarction and the other angina, but the location and characteristics of the coronary lesions were similar. It was considered that a high index of suspicion of occult CD should be maintained in the asymptomatic twin if a coronary event has occurred in the other twin.[2]

In terms of coronary angiography, little is known about angiographic expression of CD in twins.[4,5] In some cases, the location of the CD was similar in both twins. In contrast, other authors mention that, despite the similarity in risk factors, there may be important differences in both the anatomy and the degree of CD in identical twins.[2,4] The 2 patients we present had lesions in the same location (right coronary artery) and, curiously, the procedures were performed by the same professional (RM).

Unlike Mendelian genetics, cardiac diseases may represent a more complex association of genes that modify the course of the disease, with environmental influences as risk factors. In any case, the patients we describe illustrate the genetic component in the pathogenesis of CD and, at least, confirm the value of establishing a high degree of suspicion of CD in a twin once CD has been documented in the other.

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Arrhythmogenic Right Ventricular Dysplasia in the Elderly

To the Editor,

The prevalence of arrhythmogenic right ventricular dysplasia (ARVD) varies widely according to the series and the geographical area, and accounts for 5% of all sudden deaths in patients <65 years old.[1] This condition is an autosomal dominant disease with variable penetrance and incomplete expression. A higher prevalence is observed in men, and a family history is found in about 30% of patients.[2] The mean age at diagnosis is around 30 years, whereas presentation in patients >65 years old is rare.

We describe the case of a 76-year-old man with a history of benign prostatic hyperplasia, but no cardiovascular risk factors. The family history included death of 2 brothers at age 50 and 60 due to uncertain causes, as well as death of a sister at age 11, also for unknown reasons; two of the patient's sons had died accidentally. The patient consulted for mid-chest discomfort, dizziness, and palpitations; the electrocardiogram showed regular, monomonphic wide QRS complex tachycardia and a left bundle-banch block morphology. Tachycardia persisted following administration of intravenous amiodarone and electrical cardioversion was performed; sinus rhythm was restored with left anterior bundle-branch block and negative T-waves in V_2 and V_3.

The echocardiogram showed good function of the left ventricle (LV) and dysfunction of the right ventricle (RV) with dilation (43 mm), but no valve abnormalities or evidence of pulmonary hypertension (Figure 1A). Catheterization revealed RV dilation and considerable trabeculation with a "stack of coins" image, as well as normal pulmonary pressure; the LV and coronary arteries were normal (Figure 1B). Magnetic resonance imaging (Figure 1C) showed RV dilation with an area of apical dyskinesia and wall thinning. Holter monitoring disclosed 352 premature ventricular beats, with no episodes of ventricular tachycardia (VT). An electrophysiological study performed after intravenous amiodarone for several days showed ventricular electric stability.

Based on the presence of diagnostic criteria for ARVD,[2] the patient was diagnosed with this condition, and because he had experienced VT while under amiodarone therapy, a single-chamber defibrillator was implanted.[3] He subsequently had episodes of paroxysmal atrial fibrillation that triggered inappropriate discharges and in the following months, presented VT that was controlled with sotalol 160 mg/day.

A search of the Medline database (National Library of Medicine, Bethesda, MD) found 5 cases of ARVD among the elderly[4-7]; all of these patients had died and were diagnosed by histology (Table). One patient was diagnosed at 72 years,

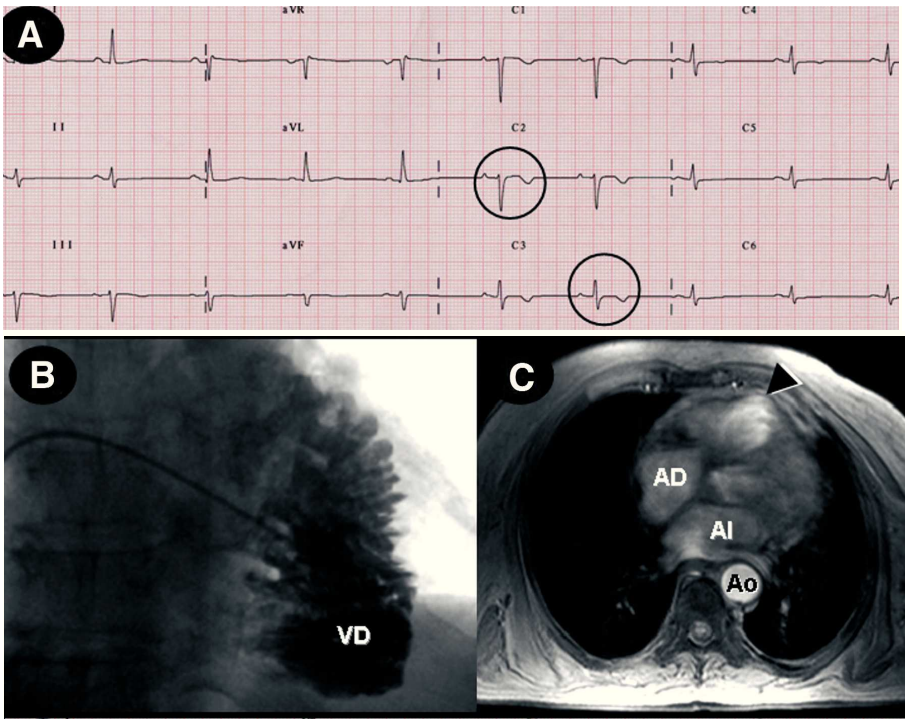

Figure 1. A: baseline electrocardiogram shows negative T waves in V_2 and V_3 (circles). B: right ventriculography discloses dilation of the right ventricle (RV) and "stack of coins" image. C: magnetic resonance image showing apical aneurysm (arrow).

TABLE. **Cases of Arrhythmogenic Right Ventricular Dysplasia in Elderly Patients Reported in the Literature***

Case	Age, Y	Sex	VT	ECG	Autopsy	Author and Literature Reference
1	76	Male	Yes	T(–) V2, V3	No	Present case
2	72†	Male	Yes	AF, T (–) V2, V3	Yes	Kamide K et al[4]
3	76	Female	Yes‡	T (–) V2, V3, ? QTc	Yes	Barriales et al[5]
4	74	Male	Yes	R V2 through V6 and e waves	Yes	More D et al[6]
5	73	Male	No	R V2 through V6 and e waves	Yes	
6	84	Female	No	CAVB, T (–) V2	Yes	Ferreira AC et al[7]

*CAVB indicates complete atrioventricular block; AF, atrial fibrillation.
†This patient presented clinical symptoms of recurrent ventricular tachycardia from age 45, although the diagnosis was made at age 72.
‡This patient presented polymorphic ventricular tachycardia.

although he had presented a history of VT from age 45.[4] The mean patient age was 75.8 years; 4 were men and 2, women. None of the cases mentioned a family history, except for the present case. Four had presented VT, one of them polymorphic VT,[5] which, even though it is not the characteristic type, has been described in some cases of ARVD. The most frequent electrocardiographic findings were negative T-waves in the right precordials (V_2 and V_3), and in two cases, epsilon waves.[7]

New pharmacological therapies, radio frequency ablation, and defibrillators[3] will probably have a favorable impact on the survival of these patients and will make it not unlikely to see elderly patients with ARVD. The possibility of a late onset of the disease should also be suspected. A two-chamber defibrillator in these patients could improve the diagnosis of supraventricular arrhythmias, because they are elderly patients in whom the prevalence of atrial fibrillation is increased and because inappropriate therapies for supraventricular tachyarrhythmia may be more common among patients with ARVD.[8]

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Kearns-Sayre Syndrome: Recurrent Syncope and Atrial Flutter

To the Editor,

Kearns-Sayre syndrome is a rare disease linked to mitochondrial inheritance. The characteristic diagnostic triad consists of progressive external ophthalmoplegia, pigmentary degeneration of the retina, and atrioventricular block.[1] It may also be associated with mental retardation, ataxia, deafness, muscle weakness, and endocrine disorders, such as diabetes mellitus or hypothyroidism. We present the case of a 37-year-old man with a noticeable phenotypic manifestation (Figure 1). From childhood, he had progressively presented photophobia, progressive loss of vision, external ophthalmoplegia, neurosensory deafness, Addison's syndrome, muscle weakness, and ataxia. His present clinical symptoms consisted of recurrent syncope and atrial flutter. The association of Kearns-Sayre syndrome with atrial flutter has not been described.

Twelve years before the current episode, he had been cardiologically assessed and diagnosed with Kearns-Sayre syndrome. At that time, the patient was asymptomatic, but the electrocardiogram showed right bundle-branch block and left anterior fascicular block. Periodic follow-up was prescribed. In the successive follow-ups, the patient remained asymptomatic with no changes in the electrocardiogram.

The onset of the current clinical symptoms started during the postoperative of a cholecystostomy, when the patient presented several episodes of syncope. The electrocardiogram disclosed atypical flutter rhythm and regular conduction with an approximate ventricular frequency of 106 bpm, right bundle-branch block, and left anterior fascicular block (Figure 2). The echocardiogram was normal. An electrophysiological study established the diagnosis of cavotricuspid isthmus-dependent flutter and counterclockwise rotation around the tricuspid valve. Isthmus ablation was performed successfully. After recovery of sinus rhythm, the atrioventricular conduction study showed prolongation of the HV interval and a short

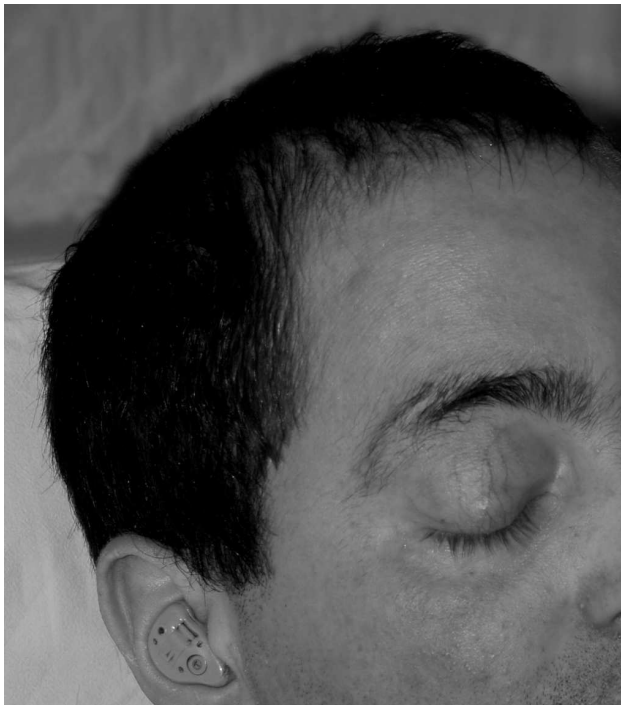

Figure 1. Patient phenotype. The presence of exophthalmos and bilateral ptosis is noticeable.

AH interval. Increased atrial stimulation disclosed worsening of infra-His conduction. These findings were considered to explain the recurrent syncope symptoms and therefore, a dual-chamber pacemaker was implanted. The patient subsequently remained asymptomatic, with no signs of further rhythm disorders. The most frequent and characteristic cardiac manifestation in this syndrome is conduction alteration, which progresses in an accelerated and unpredictable manner to complete block.[2] This is considered the main mechanism behind an elevated incidence of sudden death in Kearns-Sayre syndrome.

The practice guidelines recommend pacemaker implantation in patients with Kearns-Sayre syndrome, Class I indication, and level of evidence C in the cases of third-degree and advanced second-degree (but still asymptomatic) atrioventricular block, and with Class IIb indication in any degree of block.[3]

We are not aware of any previous report of Kearns-Sayre syndrome associated with atrial flutter. Localized slow conduction is a necessary condition for a reentrant arrhythmia such as atrial flutter to occur. In Kearns-Sayre syndrome, only infra-His conduction defects have been described. It is unknown whether there may be other areas of slow conduction that would promote reentrant arrhythmias.

In this syndrome, the atrioventricular conduction abnormality is characterized by prolongation of His-ventricular associated with atrial-His shortening and therefore, the finding of a normal PR interval may be an error. We question the value of serial observation of the surface electrocardiogram to predict the occurrence of syncope due to the particularity of the conduction disorder and unpredictable progress.

In this case, it is possible that atrial flutter acted as a trigger of syncope, although the electrophysiological study after flutter

BOOKS RECEIVED

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