

Dizygotic twin with congenital AV block

To the Editor,

The incidence of congenital atrioventricular (AV) block is estimated to be about 1 in 15,000-20,000 births. Although it is a rare disease, it increases fetal, neonatal, and childhood morbidity and mortality, and 60%-65% of all cases are reported to require pacemaker implantation before reaching adulthood (1). This disease can be idiopathic, but it is mostly caused by transmission of auto-antibodies, such as anti-Ro/SS-A and anti-La/SS-B antibodies, from mothers affected by an autoimmune disease, which damages the cardiac conduction system. Half of these mothers are asymptomatic or are not diagnosed until after their delivery.

Dizygotic twin sisters, aged 22 years with congenital AV block and a permanent pacemaker implanted in their adolescence, presented to our hospital for a routine check-up. A two-chamber permanent pacemaker was implanted in twin A and was left in DDD mode. A two-chamber permanent pacemaker was also implanted in twin B, but it was left in VDD mode. The electrocardiograms showed that the pacemaker rhythm (atrial sense, ventricular pacing) and transthoracic echocardiograms of both twins were normal. Twin A had a history of removal of the pacemaker due to infection, implantation of a new pacemaker 4 years ago, and coil embolization of patent ductus arteriosus (PDA) 3 years ago; otherwise, both twins were healthy.

Complete heart block can occur as an isolated entity, or it can accompany other congenital heart defects, like transposition of great arteries, PDA, or atrial septal defect (2). The history of PDA in one of the twins may suggest maternal infection or drug abuse during pregnancy, but there was no evidence to support this hypothesis. In addition, the mother of the twins did not have any autoimmune disease. Her anti-Ro/SS-A and anti-La/SS-B antibodies were negative. Herein, we present the first dizygotic twins in the literature with congenital AV block. Killen et al. (3) reported a similar case in chorionic diamniotic twins exposed to maternal anti-Ro/SS-A and anti-La/SS-B antibodies. However, in that report, one twin had sinus rhythm and the other had Mobitz type 1 second-degree AV block (Wenckebach). The treatment of congenital heart block includes intrauterine steroid (4) and intravenous immunoglobulin (IVIG) (5) and implantation of a permanent pacemaker after birth.

Uğur Nadir Karakulak, M. Kandemir Cengaver, Elifcan Aladağ*, Naresh Maharjan
Departments of Cardiology and *Internal Medicine, Faculty of Medicine, Hacettepe University; Ankara-Turkey

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Address for Correspondence: Dr. Uğur Nadir Karakulak, Hacettepe Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, P.O. 06100 Sıhhiye, Ankara-Türkiye
 Phone: +90 312 305 17 81
 Fax: +90 312 311 40 58
 E-mail: ukarakulak@gmail.com
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Primum non nocere

To the Editor,

Several drugs, such as dopamine antagonist antipsychotics, metoprolol, cisapride, and domperidone, cause Parkinsonian symptoms, such as akinesia, rigidity, and rest tremor. These drugs produce these side effects via blockage of D2 dopamine receptors in basal ganglia. Trimetazidine, as an anti-ischemic agent may also lead to Parkinsonian symptoms or cause deterioration of clinical status of patients with Parkinson's disease.

An 86-year-old female patient with Parkinson's disease was admitted to a cardiology outpatient clinic for routine check-up. She had coronary artery bypass grafting 12 years ago. The patient was taking dabigatran 110 mg twice a day due to atrial fibrillation, metoprolol succinate 100 mg, perindopril 10 mg, atorvastatin 20 mg, and trimetazidine 35 mg twice a day. She was prescribed trimetazidine on account of chest pain unrelated to exertion 9 months ago. Electrocardiography revealed atrial fibrillation, with a heart rate of 74 per minute. Ejection fraction of 42% and moderate mitral regurgitation were detected on the echocardiography. Her effort capacity was too limited owing to Parkinsonism. Therefore, it could not be assessed whether exertional angina or dyspnea was present. A neurology consultation was recommended due to severe bradykinesia and postural instability during walking. However, it was ascertained that she had been on close follow-up by the neurology department for 7 months, and no significant clinical improvement was provided, even with dose increments of levodopa and, thereafter, addition of carbidopa and benserazide, respectively.

The patient's physical performance deteriorated in the last 7 months by virtue of accelerated progression of Parkinsonism. There was something bizarre in the patient's clinical status. She was doing well with only a moderate dose of levodopa, and it is questionable what happened and why she got worse rapidly. The physician was remembering an adverse effect of trimetazidine, which leads to extrapyramidal side effects. However, he was not quite sure whether trimetazidine could possibly cause it. After searching PubMed for adverse drug reactions of trimetazidine, case reports with Parkinsonism after trimetazidine use were detected (1). Trimetazidine was discontinued. After 3 months, the patient was taking only levodopa again, and the outcome was quite favorable after discontinuation of trimetazidine, with an almost full recovery to her past physical performance.

Trimetazidine is quite frequently used in cardiology practice as an anti-ischemic agent, albeit it might cause heartburn, nausea, and vomiting, as well as extrapyramidal side effects. The 2013 ESC guidelines on

the management of stable coronary artery disease revealed a contraindication of trimetazidine in patients with Parkinson's disease.

As a cardiologist, we should learn all adverse effects of drugs that we are using in our daily cardiology practice, as well as pharmaceutical effects we already know well. This case teaches cardiologists to prescribe trimetazidine exactly when it is needed, and it may lead to Parkinson-like side effects, even in patients without Parkinson's disease. There is a main principal precept of bioethics that all medical students are taught in school: primum non nocere.

Çağın Mustafa Üreyen, Şakir Arslan, Yunus Cem Baş
Department of Cardiology, Antalya Education and Research
Hospital; Antalya-Turkey

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Address for Correspondence: Dr. Çağın Mustafa Üreyen,
Antalya Eğitim ve Araştırma Hastanesi, Varlık Mah.
Kazımkarabekir Caddesi Soğuksu, Muratpaşa, Antalya-Türkiye
Phone: +90 505 535 14 69
Fax: +90 242 249 44 62
E-mail: drcaginureyen@gmail.com

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