Cardiology in the Young (2009), **19**, 627–629
doi:10.1017/S1047951109990333

Brief Report

Duplication of the right coronary artery in a patient with hypertrophic cardiomyopathy and myocardial bridging

Haci Ciftci,[1] Gulten Tacoy,[2] Guliz Yazici[3]

[1]*Veni Vidi Hospital, Cardiology Department, Diyarbakır, Turkey;* [2]*Gazi University Faculty of Medicine, Cardiology Department, Ankara, Turkey;* [3]*Yuksek Ihtisas Hospital, Cardiology Department, Ankara, Turkey*

Abstract Hypertrophic cardiomyopathy is a genetic myocardial disorder. In such patients, myocardial bridging is the most frequent encountered coronary arterial anomaly. Patients may, however, on occasion, present with other much rarer malformations of the coronary arteries. Duplication of the right coronary artery is a very rare anomaly. We have now encountered a patient with hypertrophic cardiomyopathy in whom we found myocardial bridging of the anterior interventricular coronary artery and duplication of the right coronary artery. To the best of our knowledge, this association has not previously been described.

Keywords: Hypertrophic nonobstructive cardiomyopathy; coronary arterial abnormalities; chest pain; dyspnoea

Received: 26 May 2009; Accepted: 19 August 2009; First published online: 14 October 2009

Hypertrophic cardiomyopathy is a myocardial disorder known to be associated with an increased risk of ventricular arrythmias, sudden cardiac death, and myocardial ischaemia. Symptomatic bridging of the coronary arteries by myocardial bundles has a frequency of around 1 to 2%, and can produce ischaemia, acute coronary arterial syndromes, arrhythmias, and be a substrate for sudden death.[1–3] Up to one-third of patients with hypertrophic cardiomyopathy are reported to exhibit such potentially problematic myocardial bridging.[4,5] Duplication of a coronary artery is a very rare anomaly, with the left coronary artery more frequently duplicated than the right.[6] We report here a patient with hypertrophic cardiomyopathy in whom we found duplicated right coronary arteries arising by seperate orifices from the aortic sinus, and a myocardial bridging across the anterior interventricular artery.

Case report

A 31-year-old male patient was admitted to our outpatient cardiology clinic complaining of exertional chest pain and dyspnea. His chest pain appeared during exercise, disappearing with rest, radiated to the back, and had been present for 6 months. His physical examination revealed blood pressure of 100 over 60 mmHg, pulse rate of 80 beats per minute, but no additional abnormal findings. We noted nonspecific changes of the ST-T segments and left axis deviation when we examined his electrocardiogram. Results of laboratory studies were normal. He had no history of hypertension, diabetes mellitus, smoking, or dyslipidaemia, and no family history of cardiovascular disease. Transthoracic echocardiographic examination demonstrated increased thickness of the muscular ventricular septum, at 30 and 20 mm in systole and diastole, and of the posterior ventricular was at 12 and 7 mm, respectively. There was no systolic gradient across in the left ventricular outflow tract. Provacation tests also failed to reveal any gradient. Treadmill exercise testing using the Bruce protocol was stopped because of typical chest pain after 6 METS. Cardiac catheterisation also failed to reveal any gradient across the left ventricular outflow tract with or without provacation, but coronary angiography revealed ectasia in the proximal and distal parts of the anterior interventricular artery, with an extensive muscular bridge extending from the origin of the second septal perforating artery for 25 mm (Fig. 1). The circumflex artery was normal in its appearance, but selective

Correspondence to: Gulten Tacoy, MD, Assist Prof, Gazi University Faculty of Medicine, Cardiology Department, Besevler Ankara Turkiye, 06500. Tel: 0903122025633; Fax: 0903122129012; E-mail: gtacoy@gmail.com

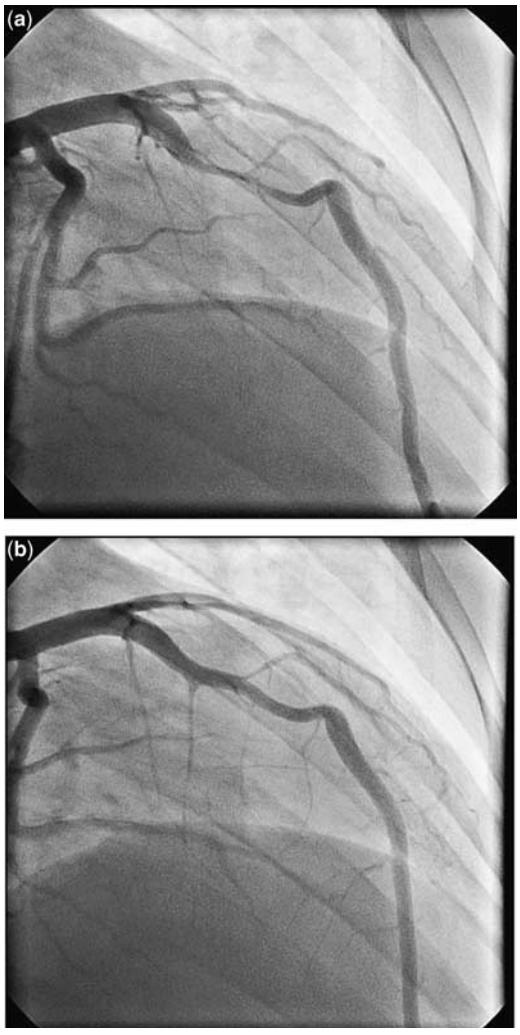

Figure 1.
*The images shown the myocardial bridge across the anterior interventricular artery during (**a**) systole and (**b**) diastole.*

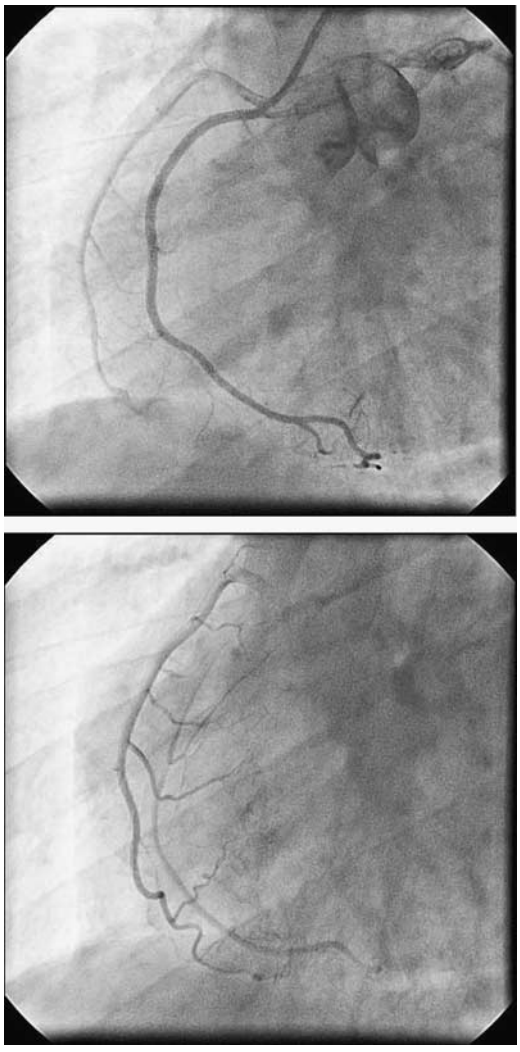

Figure 2.
The images show duplication of the right coronary artery.

injection of the right coronary aortic sinus revealed two arteries arising by separate orifices and taking the anticipated course of the right coronary artery (Fig. 2). Both arteries gave rise to atrial and ventricular branches. We treated him medically, initially prescribing Verapamil thrice daily at a dose of 80 mg. After 3 months medication, his exertional capacity was much improved, and his angina had regressed significantly.

Discussion

Hypertrophic cardiomyopathy is a common genetic disorder, having a prevalence of 1 in 500. Depending on the state of the left ventricular outflow tract, patients can be classified as obstructive and nonobstructive. Important bridging of coronary arteries by myocardial bundles is reported in up to one-third of these patients, and can produce various symptoms. Survival over the long term, nonetheless, is similar with and without myocardial bridging.[7] Other anomalies such as a single coronary artery or coronary arterial fistulas can also occur, but intramyocardial microvessel diseases are the most frequent coronary arterial problems.[1–3] Other anomalies as discovered at coronary angiography are found in only around 1% of individuals.[8,9] Duplication of a coronary artery is very rare, with the left coronary artery more frequently duplicated than the right.[6] No more than 22 patients were collected together with this lesion in one review.[4] Of these 22 patients, however, 17 were turkish, a relationship between coronary arterial anomalies and geographic variations being well established.[10] As far as we are aware, however, such duplication of the right coronary artery has not previously been encountered in a patient with

https://doi.org/10.1017/S1047951109990333 Published online by Cambridge University Press

hypertrophic cardiomyopathy and myocardial bridging. Use of beta blockers, and blockers of the calcium channels, are known to control the symptoms of patients having evidence of ischaemia, and our patient has improved markedly subsequent to treatment with verapamil.

References

1. Atmaca Y, Dandachi R, Oral D. A rare variant of single coronary artery and non-obstructive hypertrophic cariomyopathy. J Invasive Cardiol 2002; 14: 469–470.
2. Kurşaklıoğlu H, Iyisoy A, Çelik T, Barçın C, Köse S, Işık E. An unusual type of single coronary artery anomaly in a patient with hypertrophic obstructive cardiomyopathy-Case report. Anadolu Kardiyol Derg 2005; 5: 232–233.
3. Aylan Ö, Özeke Ö, Gölbaşı Z. Coronary artery-left ventricular fistulae associated with apical hypertrophic cardiomyopathy. Eur J Echocardiography 2006; 7: 326–329.
4. Sarı İ, Kızılkan N, Sucu M, et al. Double right coronary artery: Report of two cases and review of the literature. Int J Cardiol 2008; 130: E74–E77.
5. Kitazume H, Kramer JR, Krauthamer D, El Tobgi S, Proudfit WL, Sones FM. Myocardial bridges in obstructive hypertrophic cardiomyopathy. Am Heart J 1983; 106: 131–135.
6. Erbağcı H, Davutoğlu V, Türkmen S, Kızılkan N, Gümüşburun E. Double right coronary artery:review of literature. Int J Cardiovascular Imaging 2006; 22: 9–11.
7. Sorajja P, Ommen SR, Nishimura RA, Gersh BJ, Tajik AJ, Holmes DR. Myocardial bridging in adult patients with hypertrophic cardiomyopathy. J Am Coll Cardiol 2003; 42: 889–894.
8. Yamanaka O, Hobbs RE. Coronary artery anomalies in 126995 patients undergoing coronary arteriography. Cath Cardiovasc Diagn 1990; 21: 28–40.
9. Garg N, Tewari S, Kapoor A, Gupta DK, Sinha N. Primary congenital anomalies of the coronary arteries: a coronary arteriographic study. Int J Cardiol 2000; 74: 39–46.
10. Topaz O, DeMarchena EJ, Perin E, Sommer LS, Mallon SM, Chahine RA. Anomalous coronary arteries: angiographic findings in 80 patients. Int J Cardiol 1992; 34: 129–138.