Case Report
Heart Failure with Transient Left Bundle Branch Block in the Setting of Left Coronary Fistula

Stephen P. Juraschek, Lara C. Kovell, Ryan E. Childers, Grant V. Chow, and Glenn A. Hirsch

Department of Medicine, Johns Hopkins Bayview Medical Center, Johns Hopkins University School of Medicine, 4940 Eastern Avenue, Baltimore, MD 21224, USA

Correspondence should be addressed to Stephen P. Juraschek, spj@jhmi.edu

Received 25 November 2010; Accepted 11 January 2011

Academic Editor: Hendrik T. Tevaearai

Coronary arterial fistulas are rare communications between vessels or chambers of the heart. Although cardiac symptoms associated with fistulas are well described, fistulas are seldom considered in the differential diagnosis of acute myocardial ischemia. We describe the case of a 64-year-old man who presented with left shoulder pain, signs of heart failure, and a new left bundle branch block (LBBB). Cardiac catheterization revealed a small left anterior descending (LAD)-to-pulmonary artery (PA) fistula. Diuresis led to subjective improvement of the patient’s symptoms and within several days the LBBB resolved. We hypothesize that the coronary fistula in this patient contributed to transient ischemia of the LAD territory through a coronary steal mechanism. We elected to observe rather than repair the fistula, as his symptoms and ECG changes resolved with treatment of his heart failure.

1. Introduction
Coronary arterial fistulas are rare cardiac anomalies that create new pathways of blood flow between coronary vessels and thoracic vasculature or chambers of the heart [1]. Although generally asymptomatic, patients can develop complications of thrombosis, congestive heart failure, rupture, endocarditis, and arrhythmias [2]. Here, we describe a patient with acute decompensated heart failure and transient left bundle branch block (LBBB), found to have an underlying left anterior descending (LAD)-to-pulmonary artery (PA) fistula. This case illustrates the potential of coronary fistulas to induce a clinical presentation of cardiac ischemia via a coronary steal mechanism.

2. Case Report
A 64-year-old man with a history of congestive heart failure and stage IV chronic kidney disease secondary to uncontrolled hypertension and diabetes mellitus presented with three days of orthopnea and one night of intermittent left shoulder pain radiating to the back. Physical examination revealed a heart rate of 99 beats per minute and blood pressure of 149/71 mm Hg with an oxygen saturation of 94% on 4 liters of oxygen. Bilateral rales were present in the lower and middle lung fields. Cardiac auscultation revealed distant heart sounds and a regular rate and rhythm without murmur. Jugular venous pressure was elevated to 10 cm H2O. Lower extremity pitting edema (2+) was present. Laboratory analyses were significant for a hematocrit of 24% and creatinine of 3.8 mg/dL. Cardiac enzymes were normal. Twelve-lead electrocardiography (ECG) revealed sinus rhythm at 94 beats per minute and a new LBBB.

An acute myocardial infarction was suspected and the patient was taken for emergent cardiac catheterization, which showed stenosis of up to 40% of the mid-LAD coronary artery and mild disease in other vessels. In addition, a fistula was identified connecting the LAD to the distal main PA (Figures 1(a) and 1(b)). The direction of blood flow was from the coronary vessel into the pulmonary circulation. No acute intervention was performed at the time of cardiac catheterization. Transthoracic echocardiography demonstrated moderate global systolic dysfunction.
with a left ventricular ejection fraction of 40% as well
as a moderate-sized pericardial effusion without echocar-
diographic evidence of increased intrapericardial pressure.
The patient was diuresed with intravenous furosemide with
gradual symptomatic and objective improvement.

A regadenoson nuclear perfusion stress imaging study
performed after 4 days of diuresis and a blood transfusion
was negative for inducible ischemia. Furthermore, an ECG
on the day of the stress imaging study showed resolution
of the LBBB, and one week later the patient’s ECG showed
recovery to near normal left ventricular ejection fraction and
decrease in size of the pericardial effusion.

We hypothesized that the small LAD-PA fistula was
not the sole cause of heart failure symptoms but was a
contributor to the patient’s clinical presentation and ECG
findings. The small fistula in this case “steals” from the LAD-
supplied myocardium, including the left bundle. By itself,
The patient was discharged after 7 days. Since he
was previously asymptomatic without objective evidence of
ischemia in the absence of myocardial oxygen supply and
demand mismatch, we elected to continue close observation
rather than closure of the fistula.

3. Discussion

Coronary arterial fistulas are rare communications between
coronary vasculature and thoracic vessels or cardiac cham-
bers [1]. The most frequent sites of drainage include the
ventricles, pulmonary arteries, the coronary sinus, the supe-
rior vena cava, or the pulmonary veins [1]. Of the different
types of fistulas, 42% originate from the left coronary tree
and 17% drain into the pulmonary artery [3]. Fistulas are
generally congenital in origin, but they can also arise from
cardiac trauma, chest irradiation, cardiac surgery, coronary
angioplasty, and endomyocardial biopsy [1].

While fistulas are usually asymptomatic, their natu-
ral history can be variable. Adult patients who develop
symptoms often do so in the 5th or 6th decade [1, 2].
Common clinical presentations include dyspnea, congestive
heart failure, angina, aneurysm, or myocardial infarction
[4]. Whether or not a patient will develop symptoms
is determined by the degree of fistula-induced volume
overload, as well as the severity of left-to-right shunting
secondary to fistula size and location [1].

Coronary angiography is the primary diagnostic tool for
defining fistula anatomy [2]. Transesophageal echocardi-
ogram may be useful in delineating the origin, course, and
drainage of a fistula [5]. Closure rarely occurs spontaneously
in adults. Therefore, in the presence of large shunts or even in
asymptomatic patients, surgical or transcatheter ligation may
be considered to prevent long-term sequelae such as steal,
spontaneous rupture, heart failure, or myocardial ischemia
[6, 7]. Transcatheter closure is first-line therapy in suitable
anatomic cases due to lower cost, shorter recovery time,
and reduced hospital stay. This avoids thoracotomy and
cardiopulmonary bypass, which could accompany a surgical
approach [8].

This case has several limitations. Although coronary
steal may have contributed to ischemia of the left bundle
as evidenced by subsequent ECG findings, we cannot
exclude a rate-related bundle branch block. However, this
is less likely since an ECG tracing 5 months earlier, at the
same heart rate, revealed a normal QRS duration as did

**Figure 1:** Right anterior oblique cranial views during coronary angiography revealed a fistula (arrow, a) arising from the left anterior
descending (LAD) artery, which was then found to empty into the pulmonary artery (PA) via a small branch (arrow, b).
subsequent ECG tracings at similar heart rates. A right heart catheterization was not performed to assess filling pressures or to calculate a shunt fraction. Even without direct measures of these pressures, it was still evident that the patient was in congestive heart failure by history and physical exam findings. He improved significantly with diuresis, suggesting elevated filling pressures also contributed to the supply-demand mismatch. The left ventricular systolic function may have also appeared decreased at presentation because of the septal motion abnormality from the LBBB.

4. Conclusions

Our patient, who presented with orthopnea, shoulder pain, and a new LBBB, illustrates that even a small coronary fistula may “steal” enough blood flow to mimic the symptoms of an acute myocardial infarction in the setting of a transient oxygen supply-demand mismatch. A coronary fistula, therefore, may not be the sole culprit lesion or an innocent bystander, but an accomplice in causing signs and symptoms of acute heart failure.

Disclosures and Financial Support

The authors have no disclosures or financial conflicts of interest relevant to this paper to report. All authors contributed to the writing, editing, and content of this manuscript.

References

[1] S. Balanescu, G. Sangiorgi, S. Castelvecchio, M. Medda, and L. Inglese, “Coronary artery fistulas: clinical consequences and methods of closure. A literature review,” Italian Heart Journal, vol. 2, no. 9, pp. 669–676, 2001.
[2] S. A. Qureshi, “Coronary arterial fistulas,” Orphanet Journal of Rare Diseases, vol. 1, no. 51, pp. 1–6, 2006.
[3] D. C. Levin, K. E. Fellows, and H. L. Abrams, “Hemodynamically significant primary anomalies of the coronary arteries. Angiographic aspects,” Circulation, vol. 58, no. 1, pp. 25–34, 1978.
[4] R. M. Gowda, B. C. Vasavada, and I. A. Khan, “Coronary artery fistulas: clinical and therapeutic considerations,” International Journal of Cardiology, vol. 107, no. 1, pp. 7–10, 2006.
[5] L. Olivotti, S. Moshiro, G. Santoro, A. Nicolino, and F. Chiarella, “Percutaneous closure of a giant coronary arteriovenous fistula using free embolization coils in an adult patient,” Journal of Cardiovascular Medicine, vol. 9, no. 7, pp. 733–736, 2008.
[6] T. Tirilomis, I. Aleksic, T. Busch, D. Zenker, W. Ruschewski, and H. Dalichau, “Congenital coronary artery fistulas in adults: surgical treatment and outcome,” International Journal of Cardiology, vol. 98, no. 1, pp. 57–59, 2005.
[7] Y. Ata, T. Turk, M. Bicer, M. Yalcin, F. Ata, and S. Yavuz, “Coronary arteriovenous fistulas in the adults: natural history and management strategies,” Journal of Cardiothoracic Surgery, vol. 4, no. 1, p. 62, 2009.
[8] X. Y. Zhu, D. Z. Zhang, X. M. Han et al., “Transcatheter closure of congenital coronary artery fistulae: immediate and long-term follow-up results,” Clinical Cardiology, vol. 32, no. 9, pp. 506–512, 2009.