Indian Pacing and Electrophysiology Journal 16 (2016) 187–191

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

The effect of mitral valve surgery on ventricular arrhythmia in patients with bileaflet mitral valve prolapse

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ARTICLE INFO

Article history:
Received 1 July 2016
Accepted 21 October 2016
Available online 24 October 2016

Keywords:
Bileaflet mitral valve prolapse
Mitral valve surgery
Papillary ventricular arrhythmias
Sudden cardiac death
Ventricular arrhythmias
Ventricular ectopy

ABSTRACT

Background: Bileaflet mitral valve prolapse (biMVP) is associated with frequent ventricular ectopy (VE) and malignant ventricular arrhythmia. We examined the effect of mitral valve (MV) surgery on VE burden in biMVP patients.

Methods: We included 32 consecutive patients undergoing MV surgery for mitral regurgitation secondary to biMVP between 1993 and 2012 at Mayo Clinic who had available pre- and post-operative Holter monitoring data. Characteristics of patients with a significant reduction in postoperative VE (group A, defined as >10% reduction in VE burden compared to baseline) were compared with the rest of study patients (group B).

Results: In the overall cohort, VE burden was unchanged after the surgery (41 interquartile range [16, 196] pre-surgery vs. 40 interquartile range [5186] beats/hour [bph] post-surgery; $P = 0.34$). However, in 17 patients (53.1%), VE burden decreased by at least 10% after the surgery. These patients (group A) were younger than the group B (59 ± 15 vs. 68 ± 7 years; $P = 0.04$). Other characteristics including pre- and postoperative left ventricular function and size were similar in both groups. Age <60 years was associated with a reduction in postoperative VE (odds ratio 5.8; 95% confidence interval, 1.1–44.7; $P = 0.03$). Furthermore, there was a graded relationship between age and odds of VE reduction with surgery (odds ratio 1.9; 95% confidence interval 1.04–4.3 per 10-year; $P = 0.04$).

Conclusions: MV surgery does not uniformly reduce VE burden in patients with biMVP. However, those patients who do have a reduction in VE burden are younger, perhaps suggesting that early surgical intervention could modify the underlying electrophysiologic substrate.

1. Introduction

Two recent studies have implicated that mitral valve prolapse (MVP), particularly bileaflet prolapse, as a cause of sudden cardiac death (SCD) in the setting of an otherwise structurally normal heart [1,2]. Basso et al. found that 7% of young SCD victims had MVP as the only identifiable abnormality on careful clinical evaluation and autopsy [1]. Further, Sriram and colleagues described a novel syndrome marked by the tetrad of bileaflet MVP, frequent ventricular ectopy (VE), female preponderance, and repolarization abnormalities within a cohort of survivors after unexplained out-of-hospital

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Peer review under responsibility of Indian Heart Rhythm Society.

http://dx.doi.org/10.1016/j.ipej.2016.10.009

cardiac arrest [2].

Although implantable cardioverter defibrillator (ICD) therapy could reduce the risk of SCD in some high risk patients, there are no known therapies to reduce the burden of ventricular ectopy or risk of sustained ventricular arrhythmia. In isolated case reports, mitral valve surgery has been shown to reduce the burden of refractory ventricular arrhythmia [3–5]; however, not all MVP patients have a uniform reduction in arrhythmia burden after the surgery [6]. Moreover, MV surgery could potentially be pro-arrhythmic in some patients. To clarify the relationship between mitral valve surgery and VE burden, we sought to 1) determine whether MV surgery alters the burden of VE in bileaflet MVP patients, and 2) identify factors associated with a reduction in postoperative VE burden.

2. Materials and methods

This study was approved by the Mayo Clinic Institutional Review Board waiving the informed consent. The study utilized data from the Mayo Clinic Surgical Database including consecutive patients who had undergone MV surgery at Mayo Clinic — Rochester from October 1, 2007 through February 28, 2013. Eligibility criteria were as follows:

1) Patients underwent MV surgery primarily due to bileaflet MVP.
2) Patients had MV surgery after October 1, 2007 when the Holter database at our institution became available electronically.
3) Patients had both pre- and post-operative Holter monitor data for review.
4) Patients were ≥18 years at the time of surgery.

Patients without research authorization were excluded. The final cohort comprised 32 consecutive bileaflet MVP patients who had undergone MV surgery at the Mayo Clinic — Rochester with available pre- and post-operative Holter monitors.

2.1. Data collection

Medical records were reviewed to define the clinical, echocardiographic, and surgical data. All study patients underwent surgery for symptomatic severe mitral regurgitation secondary to bileaflet MVP confirmed by preoperative echocardiographic imaging and at surgical inspection [7]. Patients with ICDs were identified through a review of ICD interrogation reports for anti-tachycardia therapies or ICD shock. Follow-up was until February 30, 2014.

2.2. Holter monitoring data

Holter monitors were not routinely preformed in patients undergoing MV surgery at our institution and those without pre- and post-operative Holter monitors were excluded. In this study cohort, the indications for Holter monitors were palpitations (53.1%), atrial fibrillation (21.9%), known premature ventricular contraction (PVC)/non-sustained ventricular tachycardia (NSVT) (9.3%), syncope (3.1%), VT (3.1%), dyspnea (3.1%) and dizziness (3.1%), respectively. The Holter monitors data were reviewed for VE frequency, or number of ectopic, beats/hour (bph) in a 24-h period. The morphology data of pre- and post-operative VE was gathered from Holter monitors and 12-lead electrocardiographic tracings when available. The site of origin was grouped into outflow tract including right ventricular outflow tract (RVOT), left ventricular outflow tract (LVOT), papillary muscles (anterolateral or posteromedial muscles), or other, according to published criteria by two observers (FFS and EE) [8–10] (See Fig. 1). Discrepancies were resolved by consensus. Abnormalities in the T-wave morphology, i.e. flattening or inversion, were noted.

The formula for percent changes in postoperative VE burden was as follows: (Postoperative VE frequency/Preoperative VE frequency - 1) x 100. More than 10% reduction in VE frequency was defined as a significant reduction.

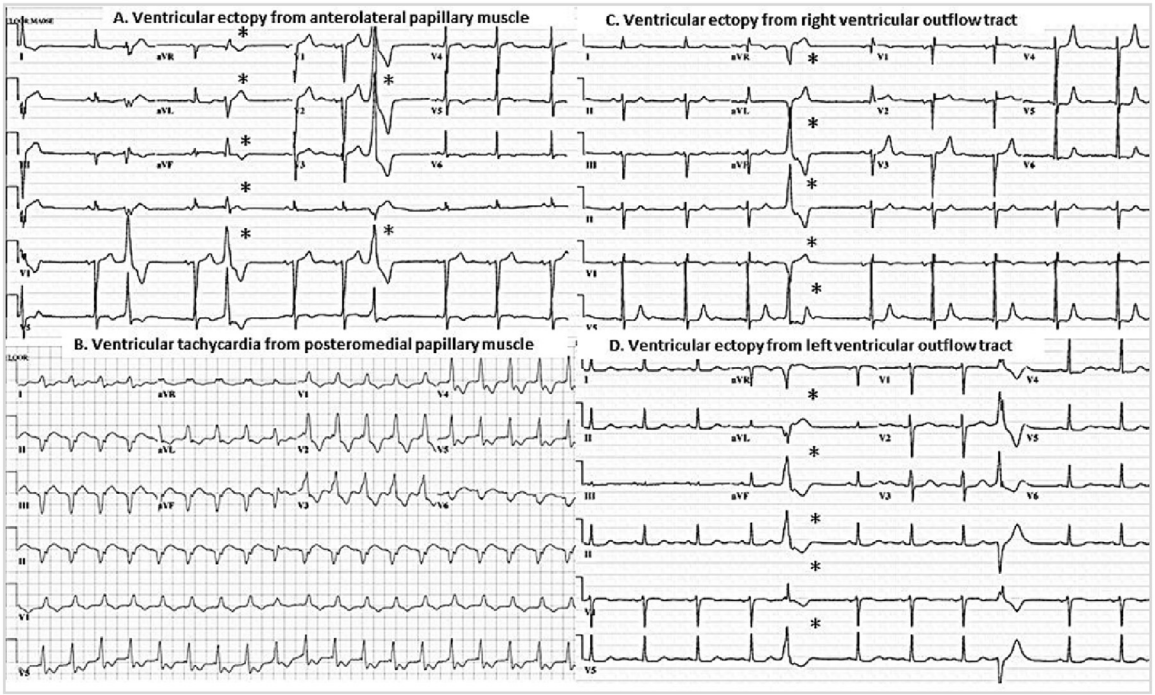

Fig. 1. Electrocardiographic tracings identify site of origin of ventricular ectopy. (A) demonstrates ventricular ectopy from anterolateral papillary muscle with wide QRS complex and atypical right bundle branch block morphology (V1 lead) and inferior right axis. (B) presents posteromedial papillary muscle ventricular tachycardia with similar atypical right bundle branch block morphology but superior and left axis. (C) Right ventricular outflow tract ectopy with negative in V1 lead (left bundle branch bock morphology) and positive deflection in leads II, III, and aVF. In contrast to ectopy from left ventricular outflow tract, (D) which has positive reflection in V1 lead.