Letters to the Editor

The Editor welcomes submissions for possible publication in the Letters to the Editor section that consist of commentary on an article published in the Journal or other relevant issues. Authors should:

- Include no more than 500 words of text, three authors, and five references
- Type with double-spacing
- See http://jtcs.ctsnetjournals.org/misc/ifora.shtml for detailed submission instructions.
- Submit the letter electronically via jtcvs.editorialmanager.com.

Letters commenting on an article published in the JTCVS will be considered if they are received within 6 weeks of the time the article was published. Authors of the article being commented on will be given an opportunity to offer a timely response (2 weeks) to the letter. Authors of letters will be notified that the letter has been received. Unpublished letters cannot be returned.

Severe mitral regurgitation after percutaneous transmitral commissurotomy: Underestimated subvalvular disease

To the Editor:

We read with interest the article by Varma and associates[1] in which the authors describe 23 cases of severe acute mitral regurgitation (MR) after percutaneous transmitral commissurotomy (PTMC). Tearing of either the anterior (n = 20) or posterior (n = 3) mitral leaflet was responsible for production of acute MR. The authors also found that the majority of these patients had significant subvalvular disease, which was underestimated on transthoracic echocardiography.

We had a similar experience, as reported earlier.[2] Acute, severe MR developed in 25 of 752 patients undergoing PTMC and necessitated emergency mitral valve replacement or repair. The most common lesion responsible for acute MR was a radial tear in either the anterior (n = 16) or posterior (n = 2) mitral leaflet. Three patients had rupture of one or more chordae. In the remaining 4 patients, creation of a pseudo-orifice in the mitral apparatus was responsible for acute MR. Moderate-to-severe commissural fusion was present in all cases, and commissural fracture was seen in only 1 case, suggesting very serious commissural fusion. The most interesting finding was the presence of moderate (n = 14) or severe (n = 5) subvalvular fusion as against pre-PTMC transthoracic echocardiographic findings of no subvalvular disease in 13 patients, mild disease in 7 patients, and moderate disease in 5 patients.

We believe the presence of undiagnosed, significant subvalvular disease may lead to either faulty transmission of balloon pressure forces leading to leaflet tear or improper engagement of the balloon leading to chordal rupture or pseudo-orifice creation. Thus, the presence of subvalvular disease plays a major role in the production of these lesions. Unfortunately, transthoracic echocardiography may not be a sensitive tool to evaluate subvalvular disease, as it was missed in most of our patients.

Shiv Kumar Choudhary, MCh
Sachin Talwar, MCh
Panangipalli Venugopal, MCh
Cardiothoracic Centre
All India Institute of Medical Sciences
New Delhi, India

References

1. Varma PK, Theodore S, Neema PK, Ramachandran P, Sivadasaanpillai H, Nair KK et al. Emergency surgery after percutaneous transmitral comissurotomy: operative versus echocardiographic findings, mechanisms of complications, and outcomes. *J Thorac Cardiovasc Surg.* 2005;130:772-6.
2. Choudhary SK, Bhan A, Sharma R, Airan B, Das B, Kumar AS, et al. Pathology of severe mitral regurgitation following balloon valvuloplasty. *Asian Cardiovasc Thorac Ann.* 1997; 5:25-30.

doi:10.1016/j.jtcvs.2005.10.044

Reply to the Editor:

We thank Choudhary and his colleagues for their interest in our article.[1] Experience in our center and also reported by others[2] shows that the majority of patients with severe subvalvular disease achieve a satisfactory outcome with percutaneous transmitral commissurotomy (PTMC). Hence we believe that we have to look beyond the role of undiagnosed subvalvular disease as the sole mechanism in producing mitral regurgitation (MR) after PTMC. This allows us to further clarify our hypothesis.

In 14 cases of MR that we analyzed, only 10 involved severe subvalvular disease. However, all had severe commissural fusion. In PTMC, commissural splitting is the mechanism for relieving mitral stenosis. Normally, commissures split at the point of least resistance to balloon pressure. However, in the presence of such rigid commissures, areas with lesser resistance can "give way." The posterior leaflet is often rolled up and thickened, whereas a more pliable anterior leaflet becomes vulnerable. In the series described by Choudhary

and colleagues, a split could be not be achieved in most cases because of severe commissural fusion. Tearing of the anterior leaflet (16/25) was the commonest cause of MR necessitation surgery in their study and in most other large series.[1,3] These data further support our findings that severity of commissural disease may be the most important determinant of production of MR after PTMC.

Failure to assess adequately the commissural disease might be the reason why many scoring systems for assessment of valves for PTMC have poor predictive value regarding the outcome and complications.[4] However, echocardiographic assessment of commissural morphology has excellent correlation with outcome.[4,5] These researchers[4] observed that there was more frequent need for oversizing the balloon in patients with single or no commissural splitting. Furthermore, in patients in whom commissural splitting could not be achieved (n = 4), there was a tear of the anterior mitral leaflet. This corroborates our findings that, in the presence of severe fusion, more pressure is required to split the commissures and then areas that offer lesser resistance can "give way." Hence, in the presence of severe commissural fibrosis detected by echocardiography, especially in the presence of calcified leaflets and thin anterior leaflets, and given the fact that valvular disease tends to be underestimated by echocardiography, PTMC carries an increased risk of leaflet tear.

Praveen Kerala Varma, MCh[a]
Praveen Kumar Neema, MD[b]
Division of Cardio-thoracic and Vascular Surgery[a]
Division of Anesthesiology[b]
Sree Chitra Tirunal Institute for Medical Sciences & Technology, Trivandrum, India 695 011

References

1. Varma PK, Theodore S, Neema PK, Ramachandran P, Sivadasanpillai H, Nair KK et al. Emergency surgery after percutaneous transmitral commissurotomy: operative versus echocardiographic findings, mechanism of complications, and outcomes. *J Thorac Cardiovasc Surg.* 2005;130:772-6.
2. Bahl VK, Chandra S, Takwar KK, Kaul U, Manchanda SC, Sharma S, et al. Influence of subvalvular fibrosis on results and complications of percutaneous mitral commissurotomy with use of the Inoue balloon. *Am Heart J.* 1994;127:1554-8.
3. Kaul UA, Singh S, Kalra GS, Nair M, Mohan JC, Nigam M, et al. Mitral regurgitation following percutaneous transvenous mitral commissurotomy. *J Heart Valve Dis.* 2000;9:262-6.
4. Agarwal BL, Kapoor A, Singh R, Tewary S, Radhakrishnan S, Shrivastava S, et al. Predictive accuracy of commissural morphology and its role in determining the outcome following Inoue balloon mitral valvotomy. *Indian Heart J.* 2002;54:39-45.
5. Fatkin D, Roy P, Morgan JJ, Feneley MP. Percutaneous balloon mitral valvotomy with the Inoue single-balloon catheter: commissural morphology as a determinant of outcome. *J Am Coll Cardiol.* 1993;21:390-7.

doi:10.1016/j.jtcvs.2005.10.043

FloWatch and pseudoaneurysm: Complication versus coincidence

To the Editor:

Michel-Behnke and colleagues[1] reported the development of pseudoaneurysm of the main pulmonary artery after implantation of an adjustable FloWatch-PAB for pulmonary artery banding.

The observed complication has been reported as subsequent to the device implantation, but no data were available to consider it as consequent to the use of the device itself. The authors carefully specified that "the mechanism of rupture of the pulmonary artery with pseudoaneurysm formation is unclear but might have been due to a lesion set during preparation."[1] Their conclusion was that "careful preparation of the pulmonary trunk and smooth placement of the device around it seem essential to prevent aneurysm formation."[1]

Pseudoaneurysm formation is a well-known complication of conventional pulmonary artery banding.[2] Although we appreciate the suggestions proposed by Michel-Behnke and colleagues,[1] and we could not agree more on the importance of a very careful dissection of the pulmonary artery to avoid a "via falsa" for the placement of the band, we respectfully disagree with the observation that the FloWatch-PAB could be implicated with the mechanism of pseudoaneurysm formation.

Looking carefully at the available intraoperative images, the main pulmonary artery appears with a visible mark like a previous conventional banding. If this was the case, the surgical dissection of the adhesions from the previous operation could have contributed to the observed complication, independently from the use of FloWatch-PAB. Also, the time frame of the formation of the pseudoaneurysm (7 days) is quite unusual for such a complication.

Furthermore, among the several advantages of the FloWatch-PAB,[3,4] 3 in particular are useful to reduce the incidence of pseudoaneurysm formation in comparison with the conventional banding. First is the capability of performing a progressive narrowing of the pulmonary artery with a gradual increase of the pressure gradient through the banding, avoiding the traumatism of the sudden constriction of a dilated, hypertensive, fragile pulmonary artery.[3,4] Second is the shape of the device (banana-like), allowing a reduction of the cross-sectional area of the pulmonary artery without modification of the length of the circumference of the pulmonary artery itself. This is a major advantage on conventional banding, in which reduction of the cross-sectional area was obtained with significant circumferential reduction of the length of the pulmonary arterial wall.[5] Because of the shape of the device, there is not need for additional sutures to fix the device to the wall of the pulmonary artery to prevent migration, as for the conventional banding. Finally, because of the 2 above advantages, we were able to show in our animal experiments normal macroscopic and histologic aspects of the pulmonary artery in correspondence of the placement of the device up to 14 months with the device in situ.[3]

The experimental data have been confirmed in the clinical experience, extended to 26 patients in the combined series of Lausanne and Liverpool, and extended to a total number of 67 devices implanted to our knowledge, with the only pseudoaneurysm in the case reported by Michel-Behnke and colleagues.[1]

Furthermore, the absence of lesions of the wall of the pulmonary artery has been substantiated by the observation that, as previously observed in all animals at the moment of FloWatch-PAB removal[3] and in all 12 in the combined series of Lausanne and Liverpool who underwent FloWatch-PAB removal at the time of intracardiac repair, there was no need for pulmonary artery reconstruction because the pulmonary artery expanded to the normal size with the simple FloWatch-PAB removal.[5]

Antonio F. Corno, MD, FRCS, FACC, FETCS
Marco Pozzi, MD, FRCS, FETCS
Liverpool, United Kingdom

Ludwig K. von Segesser, MD, FACS, FETCS
Lausanne, Switzerland