Minimally Invasive Closure of Bronchopleural Fistulas

To the Editor:

We would like to congratulate Fruchter et al1 for their ingenious, effective, and minimally invasive way of treating the very challenging condition of bronchopleural fistulas as reported in a recent issue of CHEST (March 2011). However, because we were cited in their work, we must make a correction. The misunderstanding is that our first publication was a case report of our first patient (53-year-old man with a right upper lobectomy) using this method.2 Later, Scordamaglio et al3 published a review of our experience endoscopically treating airway fistulas (two bronchopleural and one tracheoesophageal), which included this same patient. Since then, five more fistulas in four other patients have been treated, making a total of seven fistulas treated in six patients (one patient with two fistulas), with five of them already closed. Although the technique used is similar to that in Fruchter et al,1 there are small differences we believe are worth noting.

Unlike Fruchter et al1 we used the Occlutech Figulla ASD Occluder N (Occlutech International AB; Helsingborg, Sweden) because this device has a design that makes it more malleable. Fruchter et al1 stated that “bronchography was performed to outline the anatomy of the fistula, in particular its length.” Because we treated only total fistulas, the “fistula length” was not a concern in our cases because we could see the end of the stump with the bronchoscope. In our cases, the diameter of the fistula was the most relevant measure for choosing occluder size. We estimated diameter by insufflating a balloonled catheter or an endobronchial blocker inside the fistula. Consequently, we have not used fluoroscopy or bronchography.

Finally, we agree with the authors’ statement that the procedure can be carried out in a bronchoscopy suite in patients under conscious sedation, which is the most remarkable distinction between the classic surgical procedure of treating this condition and this new minimally invasive method. Once again, we congratulate the authors for their valuable contribution.

Correspondence

826

2011 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-0719

ACKNOWLEDGMENTS

Other contributions: This work was performed at the Thoracic Surgery Department, Heart Institute (InCor) do Hospital das Clinicas da Faculdade de Medicina da Universidade de São Paulo.

REFERENCES


Pulmonary Graft Dysfunction and Elevated Pulmonary Pressures

To the Editor:

In a recent study, Fang and colleagues1 brought up some interesting points concerning elevated pulmonary pressures (i.e., mean pulmonary arterial pressure [mPAP]) and primary graft dysfunction (PGD). In their study, they propose that elevated pulmonary pressures are associated with the development of PGD.

We congratulate the authors on their hard work and success. However, we do have some concerns:

• The authors do not clearly define their cutoff for elevated pulmonary pressure. According to the American College of Chest Physicians,2 most experts generally accept elevated pulmonary pressures to be an mPAP > 25 mm Hg with a pulmonary capillary or left atrial pressure < 15 mm Hg. The authors have a wide range of elevated pulmonary pressures for both the PGD and the non-PGD groups (38.5 ± 16.3 mm Hg and 29.6 ± 11.5 mm Hg, respectively).

The authors have reported to CHEST the following conflicts of interest. Drs Tedde, Scordamaglio, Rodrigues, Minamoto, and Alfinito are conducting a study (clinicaltrials.gov; Identifier: NCT01153074) for which Occlutech International AB has donated 15 Occlutech Figulla ASD Occluder N devices for a clinical protocol.

Correspondence to: Miguel L. Tedde, MD, PhD, R. Itambé, 367 ap 151 A, 01239-001 Higienópolis, São Paulo, Brazil; e-mail: tedde@usp.br

© 2011 American College of Chest Physicians.

DOI: 10.1378/chest.11-0719

ACKNOWLEDGMENTS

Other contributions: This work was performed at the Thoracic Surgery Department, Heart Institute (InCor) do Hospital das Clinicas da Faculdade de Medicina da Universidade de São Paulo.

REFERENCES


Pulmonary Graft Dysfunction and Elevated Pulmonary Pressures

To the Editor:

In a recent study, Fang and colleagues1 brought up some interesting points concerning elevated pulmonary pressures (i.e., mean pulmonary arterial pressure [mPAP]) and primary graft dysfunction (PGD). In their study, they propose that elevated pulmonary pressures are associated with the development of PGD.

We congratulate the authors on their hard work and success. However, we do have some concerns:

• The authors do not clearly define their cutoff for elevated pulmonary pressure. According to the American College of Chest Physicians,2 most experts generally accept elevated pulmonary pressures to be an mPAP > 25 mm Hg with a pulmonary capillary or left atrial pressure < 15 mm Hg. The authors have a wide range of elevated pulmonary pressures for both the PGD and the non-PGD groups (38.5 ± 16.3 mm Hg and 29.6 ± 11.5 mm Hg, respectively).

The authors have reported to CHEST the following conflicts of interest. Drs Tedde, Scordamaglio, Rodrigues, Minamoto, and Alfinito are conducting a study (clinicaltrials.gov; Identifier: NCT01153074) for which Occlutech International AB has donated 15 Occlutech Figulla ASD Occluder N devices for a clinical protocol.

Correspondence to: Miguel L. Tedde, MD, PhD, R. Itambé, 367 ap 151 A, 01239-001 Higienópolis, São Paulo, Brazil; e-mail: tedde@usp.br

© 2011 American College of Chest Physicians.

DOI: 10.1378/chest.11-0719

ACKNOWLEDGMENTS

Other contributions: This work was performed at the Thoracic Surgery Department, Heart Institute (InCor) do Hospital das Clinicas da Faculdade de Medicina da Universidade de São Paulo.

REFERENCES


standard of elevated pulmonary pressures, some patients might not even technically meet the criteria for pulmonary hypertension.

- The authors fail to definitively demonstrate the type of pulmonary hypertension. Although they report that pulmonary hypertension was due to idiopathic pulmonary fibrosis, which would generally place the patients into class 3, the readers are forced to assume this information.
- There is possible misclassification of pulmonary hypertension. In an elegant study, Halpern and Taichman2 demonstrated that many patients with a diagnosis of pulmonary arterial hypertension were misclassified and should have been classified with pulmonary venous hypertension (about one-half the patients in their study were misclassified).
- There is no mention as to whether the patients were treated for pulmonary hypertension.
- There is no mention as to whether the patients had a history of controlled or uncontrolled hypertension. Pulmonary hypertension has many causes, and suboptimal control of hypertension can cause elevated pulmonary pressures (class 2 pulmonary hypertension).
- There were differences in pulmonary pressures during surgery. The patients did not necessarily have a preoperative placement of the pulmonary arterial catheter. Pulmonary pressures can vary with volume resuscitation, intubation, type of mechanical ventilation, sedation, and so forth. The authors do acknowledge this limitation but still go on to state definitively that the lack of these data did not confound the relationship of the mPAP with PGD.

A reproduction of this study, correcting for the above concerns, would add significant value to the current literature. This would then allow clinicians to accurately assess risk factors for PGD in this instance and to counsel patients on the potential complications.

Christopher Jenks, MD
Askin Uysal, MD
Shreveport, LA

Affiliations: From the Department of Medicine and Pediatrics (Dr Jenks) and the Department of Pulmonary and Critical Care (Dr Uysal), Louisiana State University.

Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Christopher Jenks, MD, 1501 Kings Highway, Shreveport, LA 71103; e-mail: cjenks@lsuhsc.edu

© 2011 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-0682

REFERENCES


Response

To the Editor:

We appreciate the interest of Drs Jenks and Uysal in our article.1 We agree that not all the subjects with primary graft dysfunction (PGD) had a mean pulmonary arterial pressure (mPAP) > 25 mm Hg and that not all the subjects without PGD had an mPAP < 25 mm Hg. It was precisely for this reason that we presented our finding of a higher mPAP in patients with PGD than in those without as a continuous variable. The study was limited to patients with idiopathic pulmonary fibrosis, but not all patients with higher mPAP met the World Health Organization criteria for pulmonary hypertension, leading us to avoid the application of potentially inaccurate phenotypes. We do not have records of whether the subjects were actively being treated for pulmonary hypertension with pharmacologic agents.

Further, we agree that many factors may influence the first recorded pulmonary arterial pressure during the transplant procedure in patients with idiopathic pulmonary fibrosis. In order for intravascular volume or mechanical ventilation to account for our findings, patients at greater underlying risk for PGD would have to have more intravascular volume administered and higher levels of ventilator support before the diagnosis of PGD is made. Although this is unlikely, we see this as an opportunity for future research.

Jason D. Christie, MD, FCCP
Steven M. Kawut, MD, FCCP
Philadelphia, PA

Affiliations: From the Division of Pulmonary and Critical Care Medicine, University of Pennsylvania.

Financial/nonfinancial disclosures: The authors have reported to CHEST the following conflicts of interest: Dr Kawut has received funding for consulting, speaking fees, conferences, travel to conferences, research, and serving on steering committees from Gilead, Actelion, United Therapeutics, Pfizer, Gerson Lehrman, and Clinical Advisors. Dr Christie has reported that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Jason D. Christie, MD, FCCP, Division of Pulmonary and Critical Care Medicine, University of Pennsylvania, 719 Blockley Hall 423, Guardian Dr, Philadelphia, PA 19104-6021; e-mail: jchristi@mail.med.upenn.edu

© 2011 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-1228

REFERENCES


Flavocoxid and Hypersensitivity Pneumonitis

To the Editor:

I have just become aware of the report in CHEST (October 2010) by Youssef and Tomic1 of a case of hypersensitivity pneumonitis