

UCLA

UCLA Previously Published Works

Title

Pulmonary embolus caused by *Suttonella indologenes* prosthetic endocarditis in a pulmonary homograft.

Permalink

<https://escholarship.org/uc/item/9jj7v19v>

Journal

Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography, 24(5)

ISSN

1097-6795

Authors

Yang, Eric H
Poon, Kimble
Pillutla, Priya
et al.

Publication Date

2011-05-15

Peer reviewed



Pulmonary Embolus Caused by *Suttonella indologenes* Prosthetic Endocarditis in a Pulmonary Homograft

Eric H. Yang, MD, Kimble Poon, MD, Priya Pillutla, MD, Matthew J. Budoff, MD, FACC, FAHA, FSCAI, and Jina Chung, MD, FACC, Torrance, California

A 28-year-old Cambodian man with a history of congenital heart disease presented with a 6-month history of increasing fatigue, night sweats, and weight loss. His surgical history included two Blalock-Taussig shunts, ventricular septal defect closure, and placement of a pulmonary valve conduit via a Rastelli procedure. Echocardiographic and cardiac computed tomographic studies revealed a vegetation in the pulmonary homograft. Blood cultures grew gram-negative rods that were eventually identified as *Suttonella indologenes*. The patient underwent a prolonged course of intravenous antibiotics, which was complicated by septic pulmonary embolism that clinically resolved. Bacterial endocarditis caused by aerobic gram-negative organisms is uncommon. The authors report the first case of *S. indologenes* endocarditis in a patient with complex congenital heart disease. (J Am Soc Echocardiogr 2010; ■:■-■.)

Keywords: Echocardiography, Endocarditis, Congenital heart defects, Computed tomography, *Suttonella indologenes*

CASE PRESENTATION

A 28-year-old Cambodian man with complex congenital heart disease presented with a 6-month history of increasing fatigue, night sweats, and an unintentional 30-pound weight loss. The patient reported a history of congenital heart disease, including two Blalock-Taussig shunts placed 9 years of age and a subsequent surgical ventricular septal defect closure and placement of a pulmonary valve conduit via a Rastelli procedure. He denied illicit drug use and was on no medications. The physical exam was significant for a grade IV/VI holosystolic murmur, heard loudest at the right upper sternal border and a grade III/VI early diastolic murmur at the right upper sternal border.

Transthoracic echocardiography (TTE) and subsequent cardiac computed tomographic angiography revealed mesocardia, situs solitus, L-loop ventricle, and a Rastelli-type pulmonary valve conduit (Figure 1). A 1.8 × 1.1 cm mobile echodensity was seen attached to the distal portion of the pulmonary homograft and was associated with increased systolic flow with peak velocity of 3 m/s and severe regurgitation on TTE. Transesophageal echocardiography and cardiac computed tomographic angiography demonstrated the previously seen mobile density as well as additional smaller echodensities attached the wall of the homograft (Figure 2, Videos 1 and 2). Given the patient's symptoms, these findings were concerning for vegetations. The systemic (anatomic right) ventricle was moderately dilated with moderate systolic dysfunction, with an ejection fraction of

38.7%. The pulmonary (anatomic left) ventricle was mildly dilated with normal systolic function (ejection fraction, 55%). Also seen were moderate aortic regurgitation, mild pulmonary atrioventricular valve (mitral) regurgitation, mild to moderate systemic atrioventricular (tricuspid) regurgitation, and the prior ventricular septal defect repair with no residual leak. Four of four blood cultures grew gram-negative rods, later identified as *Suttonella indologenes*, susceptible to ampicillin and ceftriaxone. The patient received a 6-week course of intravenous ceftriaxone 2 g/day through a peripherally inserted central catheter, followed by a 12-week course of oral amoxicillin 875 mg twice a day. The patient was deemed at high risk for surgical intervention because of prior cardiac surgeries and was discharged upon improvement in his constitutional symptoms.

Ten days after discharge, the patient returned to the emergency room complaining of sudden onset of pleuritic right-sided chest pain. Computed tomography of the chest revealed a pulmonary embolism in the right lower lobe (Figure 3). An ultrasound of the peripherally inserted central catheter line site showed no evidence of thrombosis. Repeat TTE demonstrated a marked decrease in the size of the mobile density in the pulmonary homograft, consistent with embolization of homograft vegetation. Repeat blood cultures were negative. The patient's symptoms improved, and he was discharged home on hospital day 3. The patient is doing well 21 months after discharge and remains in New York Heart Association class I.

DISCUSSION

This is the first known case of echocardiographically documented prosthetic endocarditis caused by *S. indologenes*. The same bacterium under prior classification, *Kingella indologenes*,¹ was reported in a patient with a history of mitral and aortic valve replacements and positive blood cultures, but without vegetations seen on TTE and M-mode imaging.² Although *S. indologenes* is not officially part of the HACEK group (*Haemophilus parainfluenzae*, *H. aphrophilus*,

From the Division of Cardiology, Department of Medicine, Harbor-UCLA Medical Center, Torrance, California.

Reprint requests: Eric H. Yang, MD, Harbor-UCLA Medical Center, Division of Cardiology, 1000 West Carson Street, Box 405, Torrance, CA 90509 (E-mail: datsunian@gmail.com).

0894-7317/\$36.00

Copyright 2010 by the American Society of Echocardiography.

doi:10.1016/j.echo.2010.08.005

Abbreviations

IE = Infective endocarditis

TTE = Transthoracic
echocardiography

H. paraphrophilus, *H. influenzae*, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, *Kingella kingae*, and *K. denitrificans*, it is considered to be part of the

Cardiobacteriaceae family, with similar 16S ribosomal ribonucleic acid sequences as *C. hominis*.¹

Infective endocarditis (IE) caused by fastidious gram-negative bacilli of the HACEK group is responsible for about 5% to 10% of native valve community-acquired endocarditis in nonusers of intravenous drugs. In the congenital heart disease population, the incidence of

of cases.⁴ An Oregon registry of patients with surgically corrected congenital heart defects classified corrected pulmonary atresia as “high” annual risk for IE, with 6.4 cases per 1,000 patient-years. Overall, there was a 10-year cumulative incidence of 5.3% in patients with histories of pulmonary atresia combined with an intact ventricular septum and 6.4% combined with a ventricular septal defect.⁵ A retrospective review done by Di Filippo *et al.*⁶ at their institution comparing characteristics of 153 cases of IE in patients with congenital heart disease before and after 1990 saw an increase in IE in Rastelli correction cases after 1990 (27.8% vs 4.8%).

The American Heart Association guidelines currently recommend treatment with ceftriaxone, ampicillin-sulbactam, or ciprofloxacin

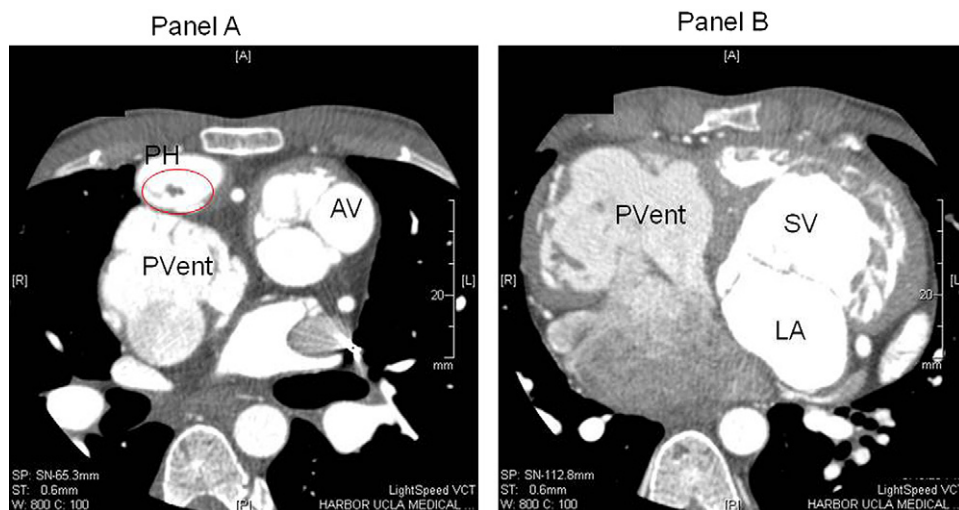


Figure 1 Sixty-four-slice cardiac computed tomographic angiographic axial views of the patient. **(A)** Transverse image at the aortic valve (AV) level. Note the filling defect in the proximal aspect of the pulmonary homograft (PH), which represents part of the vegetation (circle). **(B)** Transverse image at the atrioventricular valve level. Note the marked dilation and hypertrophied systemic ventricle (SV). LA, Left atrium; PVent, pulmonary ventricle.

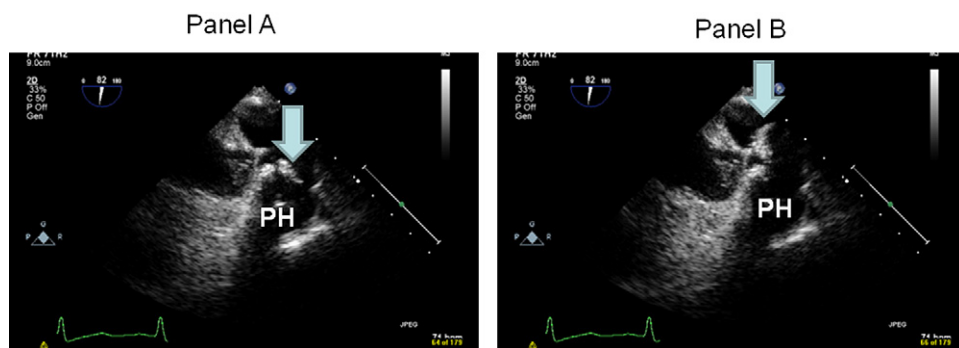


Figure 2 Transesophageal echocardiogram, midesophageal view at 82°, showing the pulmonary homograft (PH) with a mobile 1.8 × 1.1 cm vegetation (arrows) attached to the distal homograft during diastole **(A)** and systole **(B)**.

IE is not well documented on a wide scale, although it accounted for 4% of admissions to a specialized adult congenital heart unit, as observed by Li and Somerville,³ with *Streptococcus viridans* being the most common infective organism. Epidemiologic studies of IE have reported congenital heart disease patients to constitute 11% to 13%

intravenously for 4 weeks in HACEK native valve endocarditis and for 6 weeks in prosthetic valve endocarditis. Because of the rise of β -lactamase-producing strains of HACEK, they are often ampicillin resistant. Virtually all strains are susceptible to ceftriaxone or other third-generation or fourth-generation cephalosporins.⁷

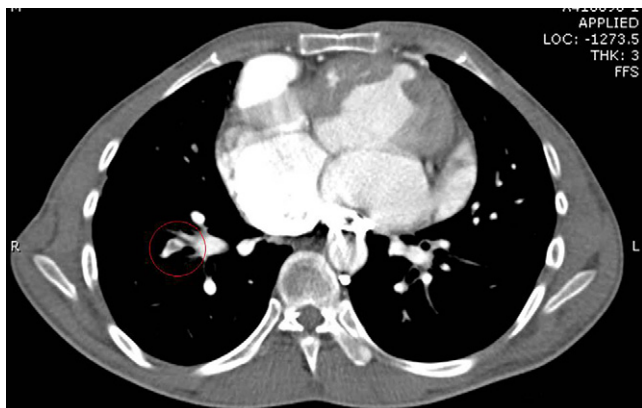


Figure 3 Computed tomographic angiography of the chest showing a pulmonary embolus in the right lower lobe (circle).

ACKNOWLEDGMENT

We would like to thank Dr. Arnold Bayer for his assistance in editing the manuscript.

REFERENCES

1. Dewhirst FE, Paster BJ, La Fontaine S, Rood JI. Transfer of *Kingella indologenes* (Snell and Lapage 1976) to the genus *Suttonella* gen. nov. as *Suttonella indologenes* comb. nov.; transfer of *Bacteriodes nodosus* (Beveridge 1941) to the genus *Dichelobacter* gen. nov. as *Dichelobacter nodosus* comb. nov.; and assignment of the genera *Cardiobacterium*, *Dichelobacter*, and *Suttonella* to *Cardiobacteriaceae* fam. nov. in the gamma division of *Proteobacteria* on the basis of 16S rRNA sequence comparisons. *Int J Syst Bacteriol* 1990; 40:426-33.
2. Jenny DB, Letendre PW, Iverson G. Endocarditis caused by *Kingella indologenes*. *Rev Infect Dis* 1987;9:787-9.
3. Li W, Somerville J. Infective endocarditis in the grown-up congenital heart (GUCH) population. *Eur Heart J* 1998;19:166-73.
4. Warnes CA, Williams RG, Bashore TM, Child JS, Connolly HM, Dearani JA, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Adults With Congenital Heart Disease). *Circulation* 2008;118:e714-833.
5. Morris CD, Reller MD, Menashe VD. Thirty-year incidence of infective endocarditis after surgery for congenital heart defect. *JAMA* 1998;279:599-603.
6. Di Filippo S, Delahaye F, Semiond B, Celard M, Henaine R, Ninet J, et al. Current patterns of infective endocarditis in congenital heart disease. *Heart* 2006;92:1490-5.
7. Baddour LM, Wilson WR, Bayer AS, Fowler VG Jr, Bolger AF, Levison ME, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. *Circulation* 2005;111:e394-433.

Video 1 Cine images of the vegetation by transesophageal echocardiography in the pulmonary homograft. Note the presence of nearby smaller mobile vegetations in the pulmonary homograft.

Video 2 Sixty-four-slice cardiac computed tomographic angiographic multiphase cinematography, axial view, showing the vegetation in the pulmonary homograft.