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WHEN TWO CHAMBERS COLLIDE: A RARE CASE OF AN ACQUIRED GERBODE DEFECT IN INFECTIVE ENDOCARDITIS

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ABSTRACT

Introduction: The Gerbode defect is a rare cardiac anomaly characterised by abnormal communication between the left ventricle and right atrium. Although primarily congenital, it can also be acquired, typically as a complication of myocardial infarction or infective endocarditis. This report examines a unique case of multivalvular infective endocarditis caused by group B Streptococcal bacteraemia, which led to the development of an acquired Gerbode defect.

Case description: A 64-year-old male patient presented with symptoms of fever and dyspnoea and was diagnosed with group B Streptococcal bacteraemia. Transoesophageal echocardiography (TOE) and cardiac magnetic resonance were used to assess the extent of valvular involvement and detect any underlying structural abnormalities. Comprehensive clinical, microbiological and imaging evaluations were performed to confirm the diagnosis.

Discussion: A transthoracic echocardiography revealed multivalvular vegetations and a newly detected left ventricle-to-right atrium shunt, consistent with a Gerbode defect. Cardiovascular magnetic resonance imaging confirmed the acquired defect. Surgery was considered but deemed too high a risk due to the likelihood of postoperative dialysis and a greater than 50% morality risk. After discussing these risks, the patient and family opted for conservative management, and palliative care was initiated.

Conclusion: This case highlights the rare development of an acquired Gerbode defect in the context of multivalvular infective endocarditis due to group B Streptococcus. Early diagnosis using advanced imaging techniques is critical for proper management. Surgical intervention remains a key therapeutic option, and a multidisciplinary approach is essential for optimising patient outcomes in such complex cases.

KEYWORDS

Gerbode defect, group B *Streptococcus*, infective endocarditis

LEARNING POINTS

- An acquired Gerbode defect (left ventricular to right atrial shunt) is a rare but critical complication of infective endocarditis.
- Infective endocarditis caused by group B *Streptococcus* in adults, though uncommon, carries a high risk of complications and mortality, especially in prosthetic valve patients.
- Early transoesophageal echocardiography is essential for identifying structural complications of infective endocarditis.

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INTRODUCTION

Named after the cardiothoracic surgeon Frank Gerbode, the Gerbode defect is a rare cardiac anomaly characterised by an abnormal connection between the left ventricle and the right atrium[1]. Although typically congenital, Gerbode defects can also be acquired. An acquired Gerbode defect is further classified as acquired iatrogenic and acquired non-iatrogenic forms. Non-iatrogenic Gerbode defects most commonly arise as a complication of myocardial infarction or infective endocarditis[1]. Although rare, infective endocarditis is the second most common cause of acquired left ventricle–right atrium shunts. This report presents a case of multivalvular infective endocarditis due to group B Streptococcal bacteraemia, which led to the development of a Gerbode defect, emphasising the diagnostic challenges and the critical clinical decisions involved.

CASE DESCRIPTION

This is a case of a 64-year-old male with a past medical history of transitional cell bladder cancer treated with radical cystectomy and ileal conduit formation. He had severe aortic stenosis, atrial fibrillation on warfarin following apixaban failure, hypertension, hyperlipidaemia and a recently diagnosed squamous cell carcinoma of the anus. He presented with fever and shortness of breath, and left-sided pleuritic chest pain, left-sided abdominal pain and dysuria.

On admission to the emergency department, the patient was febrile (39.2 °C) and tachycardic with heart rate of 118 bmp. An electrocardiogram showed the patient in atrial fibrillation with rapid ventricular response, though he remained haemodynamically stable. Urinalysis revealed marked pyuria and bacteriuria. His initial troponin level was 0.01 ng/l; computed tomography of the abdomen revealed hydroureteronephrosis and perinephric fat stranding, indicative of pyelonephritis.

Blood cultures grew *Streptococcus agalactiae* (group B Streptococcus), and urine cultures were positive for *Pseudomonas aeruginosa*. Given the clinical context, the source of sepsis was thought to be pyelonephritis, while group B Streptococcus was suspected to originate from the patient's anal carcinoma. The patient was started on cefepime 2,000 mg IV every 12 hours, but persistent fevers warranted further investigation. Given high clinical suspicion for infective endocarditis, particularly in the setting of persistent fever and sepsis despite broad-spectrum antibiotic use, transthoracic echocardiography was pursued, which revealed severe aortic stenosis with thickened leaflets but no visible vegetation.

Despite appropriate antibiotic therapy, the patient's condition deteriorated, leading to episodes of complete heart block that necessitated upgrade to the coronary care unit and temporary pacemaker placement. The patient subsequently underwent arterial line and central venous catheter placement, followed by elective intubation prior to transoesophageal echocardiogram. This was performed with colour flow Doppler imaging and revealed a fistula between

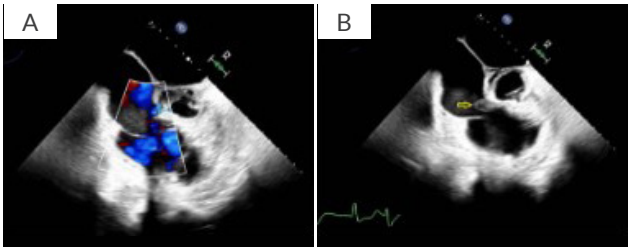


Figure 1. Transoesophageal echocardiogram mid-oesophagal view showing A) aorto-atrial fistula and B) vegetation on the tricuspid valve.

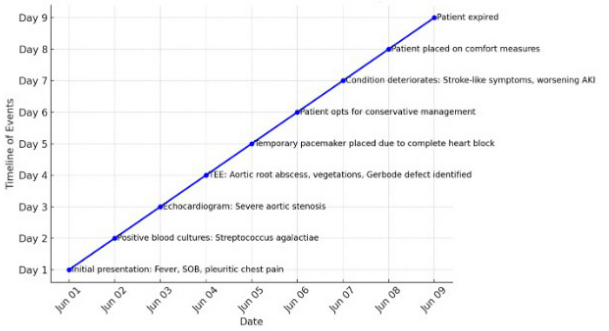


Figure 2. Timeline depicting the patient's clinical course.

the left ventricle outflow tract and the right atrium *(Fig. 1A)*, consistent with a Gerbode defect. Additionally, an aortic root abscess with vegetations on the anterior mitral leaflet, tricuspid septal leaflet *(Fig. 1B)*, and aortic valve leaflets were also identified, consistent with multivalvular infective endocarditis.

The patient's condition further deteriorated with neurological symptoms, including anomia and left upper extremity weakness, likely due to septic emboli. The severity of his condition, including worsening acute kidney injury and persistent high fever, prompted a multidisciplinary discussion about the risks and benefits of surgical intervention. Cardiac surgery was considered, but the risks – including a high likelihood of requiring postoperative dialysis and a greater than 50% mortality risk – were deemed too high. After discussing these risks, the patient and his family opted for conservative management. Consequently, the patient was transitioned to palliative care. His condition further declined, and he subsequently passed away. The timeline of the case is summarised in *Fig. 2*.

DISCUSSION

An acquired non-iatrogenic Gerbode defect most commonly arises from myocardial infarction, particularly involving the right coronary artery, as well as infective endocarditis[1]. Several theories attempt to explain the pathophysiology behind Gerbode defect development in the setting of infective endocarditis. One theory suggests that an underlying, closed congenital defect may be re-perforated due to bacterial invasion[2].

In our patient's case, a previously documented intact atrial septal defect was noted on a prior transthoracic echocardiogram. Based on this finding, it is plausible that bacterial invasion resulted in perforation of the existing

atrial septal defect, supporting the theory that infective endocarditis-induced Gerbode defects may occur in the presence of an underlying congenital anomaly. This case underscores the importance of thorough history-taking in patients with suspected infective endocarditis, as a history of congenital wall defects should raise clinical suspicion for a Gerbode defect.

Accurate diagnosis of a Gerbode defect is critical, as its clinical presentation may mimic that of tricuspid regurgitation or ventricular septal defects on physical examination, potentially leading to diagnostic errors. Differentiating a Gerbode defect murmur from those of tricuspid regurgitation or ventricular septal defects requires meticulous echocardiographic evaluation. This should include comprehensive colour flow Doppler mapping of the left ventricular outflow tract and right atrium, as well as transoesophageal echocardiography, which offers superior sensitivity for detecting abscesses and other intracardiac complications.

Cardiac magnetic resonance imaging and three-dimensional echocardiography serve as adjunct diagnostic modalities, providing detailed anatomical insights regarding the defect's size, location and shunt flow quantification[3]. These imaging tools are especially valuable when surgical or interventional closure is being considered.

Although *Staphylococcus aureus* remains the leading cause of infective endocarditis in North America, *S. agalactiae* (group B *Streptococcus*) is increasingly recognised for causing severe complications, including thromboembolic events, heart failure and cardiogenic shock[4]. As highlighted in our patient, *S. agalactiae* bacteraemia should not be underestimated and warrants prompt investigation, given that mortality rates for *S. agalactiae*-induced infective endocarditis can reach up to 50%, emphasising the need for early recognition and tailored management strategies[5].

In this case, the decision against surgical intervention was guided by the patient's frailty and high operative risk. While surgical closure is generally recommended, particularly in cases with significant shunt flow or associated complications, management decisions must be individualised based on a careful assessment of risks and benefits.

CONCLUSION

This case highlights the importance of considering acquired Gerbode defects in patients with infective endocarditis, particularly when complicated by multivalvular involvement. *S. agalactiae*, while an uncommon cause of native valve infective endocarditis, can lead to severe and life-threatening complications, including the development of a Gerbode defect. Prompt recognition and a multidisciplinary approach are essential in managing such complex cases, although the decision between surgical and conservative management must be carefully weighed based on individual patient factors and preferences.

REFERENCES

1. Saker E, Bahri GN, Montalbano MJ, Johal J, Graham RA, Tardieu GG, et al. Gerbode defect: a comprehensive review of its history, anatomy, embryology, pathophysiology, diagnosis, and treatment. *J Saudi Heart Assoc* 2017;**29**:283–292.
2. Thurman J. On aneurysms of the heart with cases. *Med Chir Trans* 1838;**21**:187–438.
3. Taskesen T, Prouse AF, Goldberg SL, Gill EA. Gerbode defect: another nail for the 3D transesophageal echo hammer? *Int J Cardiovasc Imaging* 2015;**31**:753–764.
4. Mahdi M, Ozer M, Nagra B, Aufiero P, Kantharia B. Acquired Gerbode defect in a patient with infective endocarditis of bicuspid aortic valve. *Cureus* 2021;**13**:e15352.
5. Rollán MJ, San Román JA, Vilacosta I, Sarriá C, López J, Acuña M, et al. Clinical profile of Streptococcus agalactiae native valve endocarditis. *Am Heart J* 2003;**146**:1095–1098.