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# Recurrence of Gerbode Defect despite Surgical Repair in a Patient with Bacterial Endocarditis

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**Abstract** Gerbode defect refers to an abnormal communication of the left ventricle (LV) with the right atrium (RA), causing a severe left-to-right shunt. Symptoms of gerbode defect depend on the degree of communication between LV and RA. This defect can be congenital or acquired. Some etiologies of acquired gerbode defect include cardiac trauma, myocardial ischemia, a complication of prior cardiac surgery, or bacterial endocarditis. Regardless of the etiology, surgical intervention involving the closure of the defect is the definitive treatment of this rare anatomical anomaly. We present the case of a young man with an acquired gerbode defect as a complication of bacterial endocarditis for which he underwent surgical correction. He later had a recurrence and worsening of left-to-right shunt via the gerbode defect a few days after the first surgery, requiring another surgical intervention. To our knowledge, recurrence of this defect after a surgical repair has so far not been reported in the literature. Therefore, we conclude that patients presenting with symptoms of severe right heart failure in whom prior surgical repair of the gerbode defect is performed should prompt clinicians to rule out recurrence of this defect.

Keywords: Gerbode defect, AV block, Left-to-right shunt, Hepatic congestion, right heart failure

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## 1. Case Report

A 25-year-old relatively healthy male presented to the hospital complaining of chest pain for 5 days. Prior to chest pain, the patient was experiencing fever, chills, myalgia, shortness of breath. EKG performed at the time showed first-degree AV block and physical exam was significant for a grade III/VI holosystolic murmur. Transthoracic Echocardiogram (TTE) performed showed flail aortic leaflet concerning for a vegetation, evidence of ventricular septal defect (VSD), moderate to severe aortic insufficiency (Figure 1). One of two blood cultures was positive for methicillin-sensitive Staph. Aureus (MSSA) and he was being treated with vancomycin and gentamicin initially which was later modified to oxacillin after cultures and sensitivities were complete. A repeat echo two days later revealed worsening of aortic insufficiency, persistent VSD, and moderate purulent pericardial effusion (Figure 2). The patient had no history of abnormal heart valves, congenital cardiac defects, intravenous drug use, or recent dental procedure.

During his admission, the first-degree AV block slowly progressed to a third-degree AV block. The patient subsequently underwent surgery for repair of VSD, and

aortic insufficiency. During the surgery, the patient was found to have a large hemorrhagic pericardial effusion, large vegetation on the aortic annulus, and a sizable VSD in the region of the membranous septum, which was later identified as having direct communication between left ventricular outflow tract (LVOT) and RA (Gerbode defect) (Figure 3). Inspection of the aortic valve revealed complete destruction of the right non-coronary leaflets. The annulus of the aortic valve in that area was also damaged. The tricuspid valve looked relatively uninvolved. A segment of autologous pericardial tissue was used to patch the VSD. A 24 mm homograft was used for aortic root replacement. Echocardiogram performed after the surgery showed mild aortic insufficiency, trivial peri-valvular regurgitation, and no VSD. Postoperatively, there was a discussion of a possible AV synchronous permanent pacemaker placement (PPM), but the patient spontaneously converted to sinus rhythm and appeared to have some conduction recovery without the need for a PPM. He was later discharged home on a 14-day extended

He returned to the hospital 10 days later complaining of palpitations in his chest and neck along with dyspnea on exertion. His physical exam was significant for tachycardia, a harsh grade V/VI holosystolic murmur across the entire precordium, a unilateral pulsating right

sided jugular venous distension (JVD), and a pulsating liver. He was also found to have some right upper quadrant abdominal pain. Continuous telemetry morning showed variable heart block ranging from Mobitz type I to third-degree AV block. A repeat TTE showed moderate aortic regurgitation, the flow of blood from around the aortic valve prosthesis into the RA suggestive of recurrent gerbode defect, and severe tricuspid regurgitation with a mobile vegetation on the tricuspid septal leaflet. Patient then underwent transesophageal echocardiogram (TEE) for further examination and was found that the tricuspid vegetation that was identified on TTE was, in fact, a dehisced patch repair of the gerbode defect in the RA with a recurrence and worsening of VSD from LVOT to RA (Figure 4 & Figure 5). He subsequently underwent his second cardiac surgery with replacement of the aortic valve and root with a 23 mm homograft valve conduit and a bovine pericardial patch repair of the VSD along with tricuspid valve repair. Postoperatively, he continued to remain in complete heart block and subsequently underwent placement of Micra leadless PPM a few days after the surgery.



**Figure 1.** TTE with parasternal long axis showing flail aortic leaflet and purulent pericardial effusion

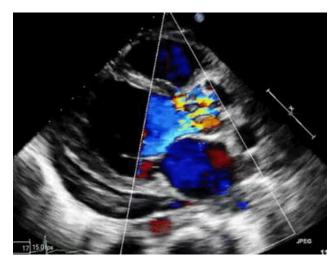


Figure 2. TTE parasternal long axis showing severe aortic regurgitation and persistent VSD



Figure 3. Surgical image showing the VSD between the LV and RA



Figure 4. TEE 4 chamber view showing vegetation on the tricuspid valve and the VSD

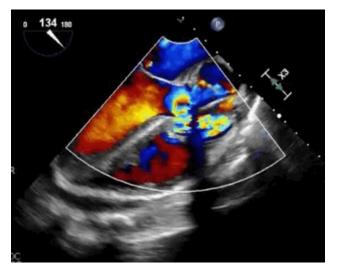


Figure 5. TEE long axis view of the LVOT at 135 degrees showing the LV to RA defect and VSD

#### 2. Discussion & Conclusion

Gerbode defect is an extremely rare cardiac anatomical anomaly that refers to an abnormal connection and shunting of blood from LV to RA. The defect can be congenital or acquired later in life due to trauma, prior cardiac surgery, myocardial infarction, or endocarditis [1]. There have been a few documented reports of this abnormal shunting dating as far back as 1838, but it wasn't until 1958 that Gerbode et al. coined the term "Gerbode defect" describing it as an anatomical defect in the ventricular-septal wall with an associated defect in the septal leaflet of the tricuspid valve [2]. The exact mechanism of acquired gerbode defect due to endocarditis is still under debate, but it is hypothesized that colonization of the valve leaflets by the offending microbial agent erodes through the septal leaflet of the [tricuspid] valve and also through the AV septum causing a direct communication between the left ventricle and RA [2,3].

Symptoms of this defect vary widely depending on the degree of communication between the two chambers. Some people can be asymptomatic while others with a larger defect can manifest symptoms of severe right heart failure such as, dyspnea on exertion, chest pain, lower extremity edema, etc [4]. A physical exam can be strikingly remarkable for a harsh holosystolic murmur heard throughout the precordium [5]. In addition to this murmur, our patient also demonstrated a unilateral (right-sided) pulsating jugular vein that was thought to be directly reflecting the LV pressures [6]. Due to the severe left-to-right shunt in our patient, he was also found to have a pulsating liver, thought to be due to liver engorgement and hepatic congestion [6]. Some degree of AV block is also associated with Gerbode defect, most likely due to the disruption of the electrical conduction system and close proximity of the defect to the AV node [4]. Definitive treatment of this defect includes surgical closure of the LV-RA communication and as noted per Prifti et al., recurrence of the defect after a surgical repair has not been reported so far [3].

We conclude that, although rare, gerbode defect can recur despite surgical correction, and patients presenting with symptoms of right heart failure, especially younger ones with a history of prior surgical correction of the gerbode defect, should prompt clinicians to rule out the possibility of recurrence of the communication between LV and RA. Perhaps recurrence occurs due to the repair being challenging given the friable nature of the tissue.

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