

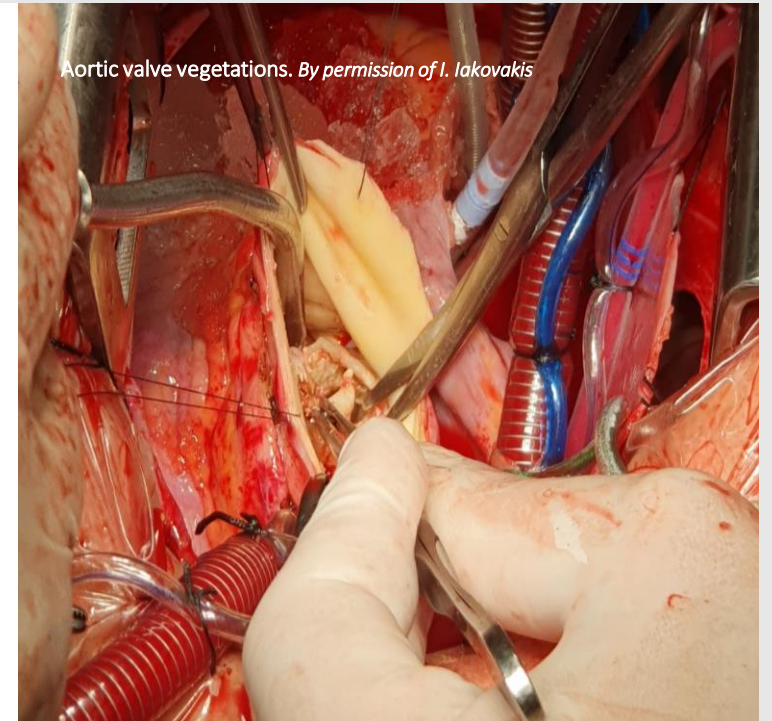
# An intriguing case of unruptured right coronary sinus aneurysm, complicated with aortic valve endocarditis and acute heart failure.

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**Introduction:** A Valsalva sinus aneurysm (SVA) is a rare asymmetrical dilatation of the aortic root between the annulus and the sino-tubular junction,<sup>1,2,3</sup> with estimated prevalence is ~0.09% in the general population.<sup>2,3</sup> Their size and possible protrusion into nearby structures defines the treatment strategy, however, surgery seems to be the gold standard for preventing life threatening complications, such as rupture or thrombus formation<sup>2,3</sup>

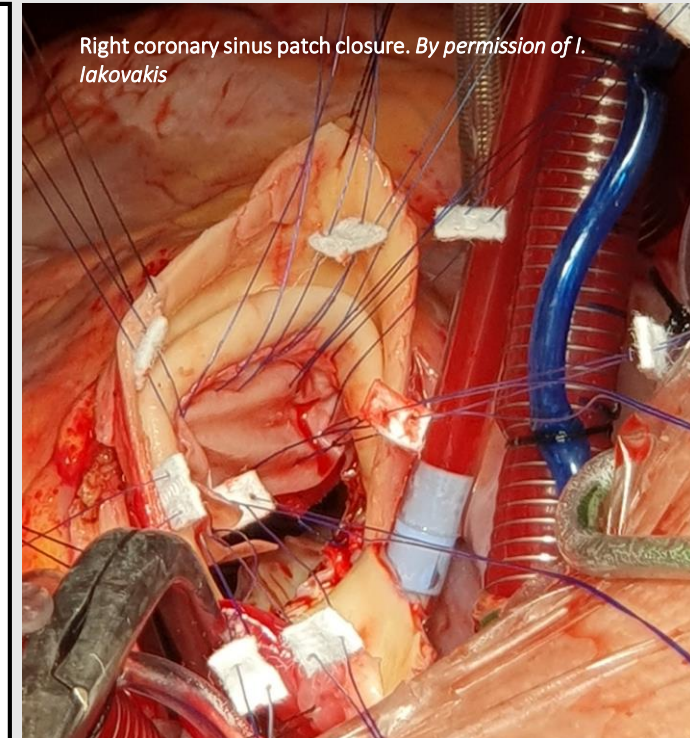
**Case presentation:** A 50 year old male with a history of untreated severe aortic stenosis presented in the Emergency Department with acute pulmonary edema. Transthoracic echocardiography revealed a dilated left ventricle (LVEDd=62mm) with concentric hypertrophy and mid-range reduced ejection fraction (40%). The aortic valve was tri-leaflet and calcified with severe regurgitation and stenosis, while a mobile structure 2x1,5 cm was noticed with possible protrusion into a small cavity by the right coronary sinus. The patient was immediately admitted in the cardiovascular Intensive Care Unit, set on intravenous diuretics, and 3 pairs of blood cultures were acquired. A transesophageal echocardiography was further on performed and it confirmed severe aortic regurgitation with rupture of the right coronary cusp and extension into the sino-tubular junction. Dilation of the aortic root at 52mm and right Valsalva sinus aneurysm were also described. The patient was set on an initial treatment for aortic valve endocarditis with ceftriaxone. Further CT investigation confirmed the right SVA and additional coronary angiography showed no coronary calcification, but aberrant origin of the right coronary artery from the non-coronary cusp. During his stay in the ICU the patient's already critical clinical condition further deteriorated (elevation in CRP, WBC, procalcitonin) developing decompensated heart failure with need for intravenous inotropes and vasoconstrictors. Moreover, respiratory and renal failure were established. A Covid-19 infection was diagnosed and repeated CT scanning showed multiple bilateral infiltrations, the patient required mechanical ventilation and he was urgently intubated. As a result, surgery was delayed. In the meanwhile, blood cultures developed *Streptococcus mitis* and pharmacological treatment was adjusted to ceftriaxone and vancomycin, according to antimicrobial susceptibility testing, besides remdesivir and dexamethasone for the Covid-19 infection



## The operation

Examination of the aortic root revealed widespread infection of the area with multiple large vegetations on the valve cusps and root and rupture of an extremely friable right aortic cusp. A complete destruction of the aortic annulus between the left coronary-to-right coronary and right coronary-to-non coronary commissures was observed. Indeed, a very friable muscle (part of the interventricular septum (IVS) was all that could be seen in the position of the right aortic annulus. In addition, a large RCSA was identified with a large (2x3cm) opening “mouth” towards the aortic root, ending blindly within the right ventricle (RV) but without communication with the RV cavity. Notably, the walls of the RCSA and the RV were firmly fixed to each other and were in fact unified to a single structure. The anomalous origin of the RCA from the non-coronary sinus was also seen. The calcified and infected aortic valve cusps were excised and a thorough debridement of all infected and necrotic tissues along with the vegetations was carried out. Then, the left ventricular cavity, the aortic root and the cavity of the RCSA aneurysm were washed with copious amounts of normal saline. All removed material was sent for microbiological culture. Next, the destroyed right aortic annulus was reconstructed placing multiple Teflon strips on the upper and inner surface of the IVS and suturing them with non-absorbable polypropylene sutures, forming, thus, a neo-right aortic annulus. Then, an appropriately sized piece of bovine pericardium was sutured (with interrupted and continuous 4/0 prolene sutures) in 4 directions: 1) downwards, onto the neo-aortic annulus, 2) upwards, onto the upper edge of the mouth of the RCSA, 3) medially, into the left-to-right aortic commissure and 4) laterally, into the left-to-non aortic commissure. Thus, the “mouth” of the RCSA was closed, forming a new aortic wall (from bovine pericardial patch). Subsequently, a 23mm bioprosthetic pericardial aortic valve (Edwards RESILIA) was implanted using 12 non-inverted 2/0 ethibond mattressed pledgetted sutures (there were concerns that a mechanical prosthesis implanted in the environment of an extensively reconstructed aortic root and annulus might not have been functional in terms of unimpeded leaflet mobility, therefore, a bioprosthesis was considered as a much safer option in these circumstances and was chosen instead).

The patient was returned in cardiac ICU sedated, intubated and mechanically ventilated, supported by inotropes and vasoconstrictors, having satisfactory urine output. Heart failure treatment was uptitrated. His clinical condition, as well as laboratory exams gradually improved. The patient was uneventfully extubated on the sixth postoperative day and was gradually weaned from inotropes and vasopressors. He was then transferred to the Thoracic Surgery Clinic for completion of his therapy.



### Conclusions:

The treatment approach of SVAs is challenging by itself. Intervention is not usually indicated when so many comorbidities occur at the same time, however, in our case, concomitant aortic valve endocarditis and acute aortic valve regurgitation defined surgery as the only life-saving approach.

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