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CONGENITAL PULMONARY ARTERY STENOSIS DIAGNOSED AFTER COVID-19-ASSOCIATED PNEUMONIA IN AN ELDERLY PATIENT WITH MULTIVALVULAR PATHOLOGY AND SUBSEQUENT SUCCESSFUL SURGICAL TREATMENT

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Doi: https://doi.org/10.52340/jecm.2025.06.01

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Covid-19-თან ასოცირებული პნევმონიის შემდეგ დიაგნოსტირებული ფილტვის არტერიის თანდაყოლილი სტენოზი ხანდაზმულ პაციენტში მულტისარქვლოვანი პათოლოგიის თანხლებით და შემდგომში წარმატებული ქირურგიული მკურნალობით

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რეზიუმე

ფილტვის არტერიის თანდაყოლილი სტენოზი მეტწილად გამოწვეულია გენეტიკური ფაქტორებით და ემბრიონული განვითარების დეფექტით. გულის სხვა მანკებთან კომბინირების გამო კლინიკური მანიფესტაცია საკმაოდ რთულია, რის გამოც, ბავშვობის ან მოზარდობის ასაკში გამოვლინდება. ხანდაზმულ ასაკში დიაგნოსტირება ძალიან იშვიათია. თანმხლებმა ფაქტორებმა, მაგალითად, ვირუსულმა ინფექციამ შესაძლოა სიმპტომების პროვოცირება გამოიწვიოს.

მოცემულ სტატიაში აღწერილია 69 წლის მამაკაცის კლინიკური შემთხვევა კომბინირებული სარქვლოვანი მანკითა და კოვიდ-19-თან ასოცირებული პნევმონიით, რომელსაც დაუდგინდა ფილტვის არტერიის თანდაყოლილი სტენოზი. შემთხვევა საინტერესო იყო თანმხლები დაავადებითაც, მათ შორის: კომბინირებული სარქვლოვანი მანკი, იშემიური კარდიომიოპათია, ესენციური ჰიპერტენზია, გულის ათეროსკლეროზული ავადმყოფობა, მეორადი პულმონური ჰიპერტენზია, თირკმლის ქრონიკული დაავადება (მესამე სტადია), ჭარბი კალორიების მიღების შედეგად განვითარებული სიმსუქნე, შაქრიანი დიაბეტი ტიპი 2. ჩაუტარდა ქირურგიული ოპერაცია: აორტის და პულმონური სარქვლების ბიო-პროტეზირება, მარჯვენა პარკუჭის გამომავალი ტრაქტის ტრანსანულარული გაფართოება ჩანართის გამოყენებით, მიტრალური სარქვლის პლასტიკა რგოლზე. არაკეთილსაიმედო პროგნოზის მიუხედავად, ჩივილების ინტენსივობა შემცირდა, დინამიკაში მდგომარეობა მნიშვნელოვნად გაუმჯობესდა.

Literature Review. Pulmonary artery stenosis is a common congenital heart defect that involves mild, moderate, or severe narrowing of the right ventricular outflow tract, pulmonary valve, or pulmonary artery, which limits blood flow from the heart to the lungs. Pulmonary stenosis may occur in isolation or in conjunction with other congenital heart anomalies. It is typically diagnosed during childhood or adolescence, while its verification in elderly patients is extremely rare [1,2,3].

Pulmonary stenosis primarily develops due to defects in the formation of the pulmonary valve during the first eight weeks of embryonic development, though the exact cause remains unknown [4]. As a result of the stenosis, pressure increases in the right ventricle, leading to its hypertrophy. Clinical manifestations depend on the severity of stenosis: mild cases may be asymptomatic, while severe cases can present with dyspnea, shortness of breath, fatigue, and cyanosis.

Pulmonary stenosis is frequently associated with other congenital heart defects, which affect hemodynamics and worsen disease progression. In tricuspid valve defects, systolic regurgitation of blood

occurs from the right ventricle into the atrium. Pulmonary stenosis and tricuspid regurgitation are common in Ebstein's anomaly, which further exacerbates the severity of regurgitation. Tricuspid stenosis causes hypertrophy of the right atrium due to impaired blood flow from the atrium to the ventricle. Mitral stenosis and regurgitation lead to increased pulmonary venous pressure, pulmonary hypertension, and heart failure.

Pulmonary artery stenosis associated with ventricular septal defect is commonly diagnosed prenatally [5]. Congenital heart defects are mainly associated with genetic syndromes such as Noonan syndrome - characterized by pulmonary valve stenosis, short stature, webbed neck, and developmental delay; or Tetralogy of Fallot - consisting of pulmonary stenosis, ventricular septal defect, dextroposition of the aorta, and right ventricular hypertrophy. Additionally, missense mutations in the transcription factor exons GATA4 and TBX5, which play essential roles in gene expression during embryonic development, are involved in heart formation and morphogenesis [6].

Studies have shown that right ventricular outflow tract obstruction is most strongly influenced by hereditary factors [7]. Multivalvular heart damage is observed in various conditions such as rheumatic fever, infective endocarditis, and connective tissue diseases [8].

The COVID-19 pandemic highlighted patients with congenital heart defects as a high-risk group. According to the World Health Organization, SARS-CoV-2 enters host cells via angiotensin-converting enzyme 2 (ACE2) receptors. Upon binding, the enzyme undergoes hydrolysis, leading to decreased arterial pressure. During the pandemic, patients struggled with hypertension management due to suppression of antihypertensive effects, unmasking previously compensated and undiagnosed congenital heart defects.

Furthermore, widespread evidence emerged of myocardial toxicity due to the virus, reflected in elevated serum troponin levels and increased risk of myocardial infarction [9,10,11]. In addition, direct damage to endothelial cells of the pulmonary artery was observed, leading to vascular spasms and stenosis progression due to the release of pro-inflammatory mediators - IL-6, IL-1, and TNF-α -and the initiation of chronic inflammation [12,13].

The primary method for diagnosing pulmonary stenosis is transthoracic Doppler echocardiography. It enables the evaluation of stenosis anatomy, etiology, associated lesions, disease severity, and transvalvular pressure gradients. Accurate assessment is necessary to identify the type of valve dysfunction: dysplasia, prolapse, atresia, or agenesis, as well as dilation and hypertrophy of the right atrium and ventricle.

Pulmonary valve evaluation can also be performed through electrocardiography - revealing right axis deviation, right ventricular hypertrophy, right atrial enlargement (p-pulmonale), and arrhythmias (commonly right bundle branch block). Radiography shows right ventricular dilation, post-stenotic dilation of the pulmonary artery, and reduced pulmonary vascular markings [14]. Further diagnostics include transesophageal echocardiography, cardiac catheterization, magnetic resonance imaging, and computed tomography, especially in cases associated with other congenital anomalies [15].

Balloon valvuloplasty has replaced surgical valvulotomy as a minimally invasive procedure. A balloon-tipped catheter is introduced via the femoral vein and guided to the heart using ultrasound. Once at the narrowed valve, the balloon inflates to enlarge the orifice. If balloon valvuloplasty is not possible, surgical valvulotomy is performed to relieve the obstruction. Valve replacement may be considered in patients with severely damaged and dysfunctional valves. Reconstruction of the right ventricular outflow tract is used in cases with significant peripheral pulmonary artery stenosis. The intervention type depends on stenosis severity, anatomical variation, patient age, and associated anomalies [16,17].

Pharmacological treatment options include endothelin receptor antagonists, oral and parenteral prostacyclin analogs, phosphodiesterase-5 inhibitors, beta-blockers, anticoagulants, and diuretics. Lifestyle changes are essential, including reduced table salt and mineral water intake, lipid-lowering diet, and regular walking [18]. Potential complications include pulmonary hypertension, right-sided heart failure, myocardial infarction, and sudden cardiac death [19].

Case Report. A 69-year-old male was hospitalized in the intensive care unit of a cardiology clinic in November 2021 with the following diagnoses: combined valvular heart disease, ischemic cardiomyopathy, essential hypertension, atherosclerotic heart disease, secondary pulmonary hypertension, stage III chronic kidney disease, and obesity caused by excessive caloric intake. The patient had insulin-independent type 2 diabetes mellitus with a 21-year history.

Symptoms: The patient experienced intense, constrictive retrosternal chest pain during regular physical activity, radiating to the left shoulder, relieved by rest; dyspnea, shortness of breath, choking sensation, and palpitations.

Objective examination: The skin and visible mucous membranes were pale with cyanotic tinge. The patient was in an orthopneic position. Blood pressure was 120/60 mmHg, heart rate 102 bpm with a regular rhythm. Percussion revealed dullness in the infrascapular regions bilaterally. Cardiac tones were muffled with systolic murmur heard at all auscultation points. Respiratory rate was 28 breaths/min; SpO_2 was 94% (88% without oxygen). Auscultation showed coarse vesicular breath sounds bilaterally, with moist rales and crepitations in the infrascapular areas.

Laboratory findings: Fibrinogen 512 mg/dL, creatinine 139 μmol/L, C-reactive protein 30.8 mg/L, D-dimer 211 ng/mL, prothrombin index 93%.

Instrumental examinations: Electrocardiogram (ECG): Showed evidence of a previous myocardial infarction, left axis deviation, first-degree AV block, and complete left bundle branch block. Chest X-ray: Enhanced pulmonary vascular markings, consolidated hila, widened aortic arch, and moderately enlarged left cardiac border. Echocardiography: Significant dilation of the left atrium and moderate dilation of the remaining chambers. Pulmonary trunk dilation. Calcification of the aortic annulus and cusps (grade II–III). Poor differentiation of the cusps. Fibrosis of the mitral ring and cusps (grade III), calcification (grade I), posterior cusp restriction. Mild eccentric hypertrophy of the interventricular septum and posterior wall. Asynchronous segmental wall motion. Left ventricular systolic dysfunction (EF 33%) mainly due to hypo-akinesis of the septal segments. Right ventricular systolic dysfunction (TAPSE 14 mm). Doppler: Relaxation could not be assessed due to monophasic curve. Severe aortic valve stenosis (maxPG 80 mmHg, mPG 40 mmHg; possibly more due to low flow/low gradient). Moderate pulmonary valve stenosis (maxPG 58 mmHg), probable patent ductus arteriosus. Severe mitral regurgitation (grade IV), moderate tricuspid regurgitation, mild aortic and pulmonary regurgitation. Significant pulmonary hypertension. No pleural effusion.

Past history:

- 2018: Myocardial infarction and coronary stenting with a drug-eluting stent.
- 2020: COVID-19-associated pneumonia. The patient received two doses of the AstraZeneca vaccine.

Prognosis was initially unfavorable. The patient was started on anti-ischemic, antiplatelet, anticoagulant, and dehydrating therapy. Following a therapeutic consultation, antibiotics were added. With treatment, the patient's condition improved: adequate diuresis was achieved, signs of circulatory congestion resolved, and anginal pain was relieved. No arrhythmias or signs of heart failure were noted,

and the patient was able to lie horizontally. Selective coronary angiography revealed no significant coronary artery stenosis. Due to valvular pathologies, a cardiac surgery consultation was recommended.

On January 21, 2022, the patient underwent surgery: bioprosthetic replacement of the aortic and pulmonary valves, transannular enlargement of the right ventricular outflow tract using a patch, and mitral valve annuloplasty (FKSB10, FMSD10, FBSE10).

Surgical details: A median sternotomy was performed. Cannulation of the ascending aorta, superior and inferior vena cava was conducted. After clamping the aorta, an aortic incision was made and cardioplegia was delivered selectively into the coronary ostia (total 2600 mL). The aortic valve leaflets were thickened and calcified. Following hypothermic cardiac arrest, the aortic valve was excised and debrided. A vertical incision was made on the pulmonary artery, revealing fibrotically altered valve leaflets. The valve diameter corresponded to size N19. The pulmonary valve was excised, and the incision extended transannularly into the free wall of the right ventricle.

An atriotomy was performed, and mitral valve reconstruction was carried out using a prosthetic ring (Edwards Lifesciences N32). In the aortic position, a biological valve (SJM-N23) was implanted using mattress sutures. The aortotomy was closed with a double continuous suture. The atriotomy was closed with a continuous suture (Prolene 4/0). After declamping, the heart resumed function spontaneously.

Under working heart conditions with cardiopulmonary bypass support, mattress sutures were placed for the pulmonary valve prosthesis, and a biological valve (SJM-N25) was implanted. A patch for the defect between the right ventricle and pulmonary artery was created from Dacron prosthesis. The patch was sutured in place using continuous sutures, and the biological valve was fixed to the patch. The patient was weaned from cardiopulmonary bypass with inotropic support. Drainage of the mediastinum and pericardial cavities was performed. Two temporary pacing wires were attached to the right ventricle. Drains and electrodes were brought out subxiphoidally. Hemostasis was achieved, and the wound was closed anatomically in layers using absorbable sutures. The valve leaflets of the aortic and pulmonary valves were sent for histopathological examination.

Follow-up and clinical progression:

31.01.2022 – Mild to moderate dilation of all cardiac chambers was observed. Mild eccentric hypertrophy of the posterior wall. Asynchronous movement of septal segments. Significantly reduced left ventricular systolic function (EF 28%) due to hypokinesis of the septal segments. Right ventricular systolic function was within normal limits. Systolic pressure in the pulmonary artery was normal. Pericardial effusion was present. Bilateral pleural effusion measured 2 cm.

13.12.2023 – The patient presented with complaints of fatigue during moderate physical exertion and palpitations. No other complaints were reported. Re-evaluated by a cardiologist and continued treatment included ACE inhibitors, aldosterone receptor blockers, antiarrhythmics, microcirculatory enhancers, antithrombotics, hypoglycemics, proton pump inhibitors, and lipid-lowering agents.

13.09.2024 – Cardiac chamber dimensions remained stable, with a tendency toward regression of moderate dilation. Left ventricular systolic function improved with EF reaching 40%. Partial recovery of segmental myocardial kinetics and reduced asynchrony were noted. Mild eccentric hypertrophy of the left ventricular posterior wall persisted. Right ventricular systolic function remained within normal limits. Systolic pressure in the pulmonary artery was normal. No pericardial effusion was visualized. Bilateral pleural effusion was reduced to 1 cm. Table N1 shows the dynamics of blood test data from 2021 to 2024 during treatment in the clinic.

Table N1. Blood Test Dynamics Over Time

Date	08.11.21	15.12.21	21.01.22	31.01.22	13.12.23	13.09.24
Na^+ (mmol/L)	139.5	134	139.5	135	140	135
K^+ (mmol/L)	4.66	4.28	4.66	4.48	3.9	3.6
Glucose (mmol/L)	4.0	5.8	11.7	4.0	4.0	4.0
C-reactive protein (CRP) (mg/L)	30.8	8.8	9.8	76.3	5.2	3.0
Prothrombin Index (%)	93	94	93	81	85	79
Troponin (μg/L)	0.13	0.097	0.026	0.01	0.015	0.01
Creatinine (μmol/L)	139	108	94	77	70	65
Fibrinogen (mg/dL)	512	512	451	721	175	150
ESR (mm/h)	25	90	27	85	35	20

Scheme N1. Dynamics of Laboratory Indicators by Year

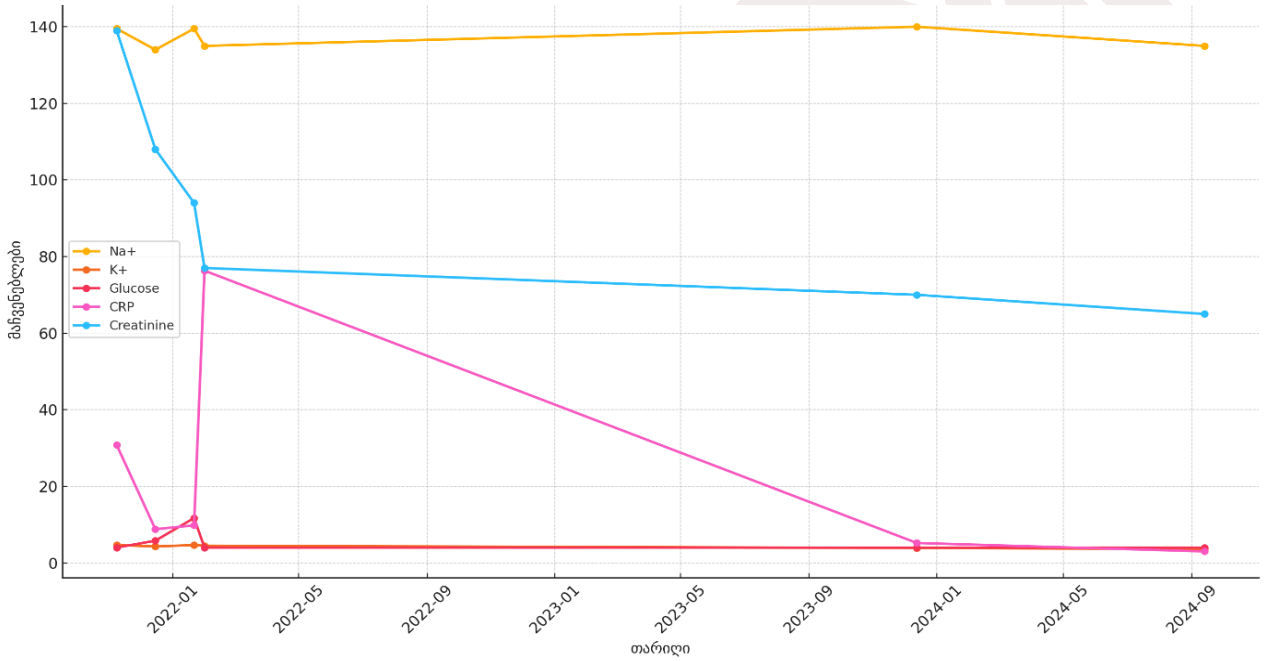


Table N2. Dynamics of Instrumental Indicators from 2021 to 2024

Date	ECG	Chest X-ray	Echocardiography	Coronary Angiography
08.11.2021	Myocardial infarction	Pulmonary-bronchovascular pattern intensified, hilar consolidation, clear sinuses. Aortic arch widened, left cardiac border moderately enlarged.	Dilated pulmonary trunk. Mild to moderate pulmonary valve stenosis. Pulmonary regurgitation. Small amount of pleural effusion. Severe mitral regurgitation.	Anterior descending artery – medially implanted stent without restenosis. Circumflex artery – ostial stenosis 30%, medial 40%.

15.12.2021	Acute subendocardial infarction	Intensified bronchovascular markings, hilar consolidation, clear sinuses. Aortic arch widened, left cardiac border moderately enlarged.	Severe mitral valve defect. Tricuspid valve insufficiency. Reduced systolic function of ventricles.	Right coronary artery – 40% medial stenosis. First marginal branch – 60% ostial to proximal stenosis.
21.01.2022	–	Lungs expanded, intensified bronchovascular pattern, hilar consolidation.	Biological prosthesis on mitral valve. Trivial valve regurgitation. Minor pleural effusion.	No hemodynamically significant coronary stenosis observed.
31.01.2022	–	Same as previous findings.	Left ventricular systolic dysfunction. No pericardial effusion. Normal prosthetic valve function.	No significant restenosis.
13.12.2023	–	Postoperative changes in mediastinal shadow, central venous catheter.	Left ventricular dilatation. Severe regurgitations. Normal prosthetic valve function.	Not relevant.
13.09.2024	–	Bilateral pleural effusion.	Normal prosthetic valve function. No pericardial effusion.	–

Dynamics of chest X-ray data by year:

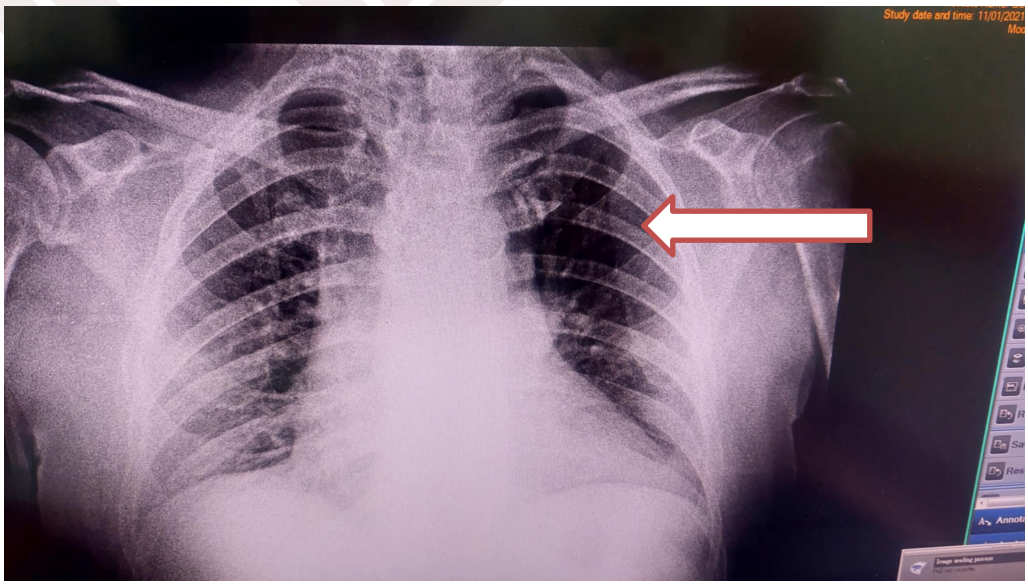


Figure 1. Chest X-ray – 11.01.2021

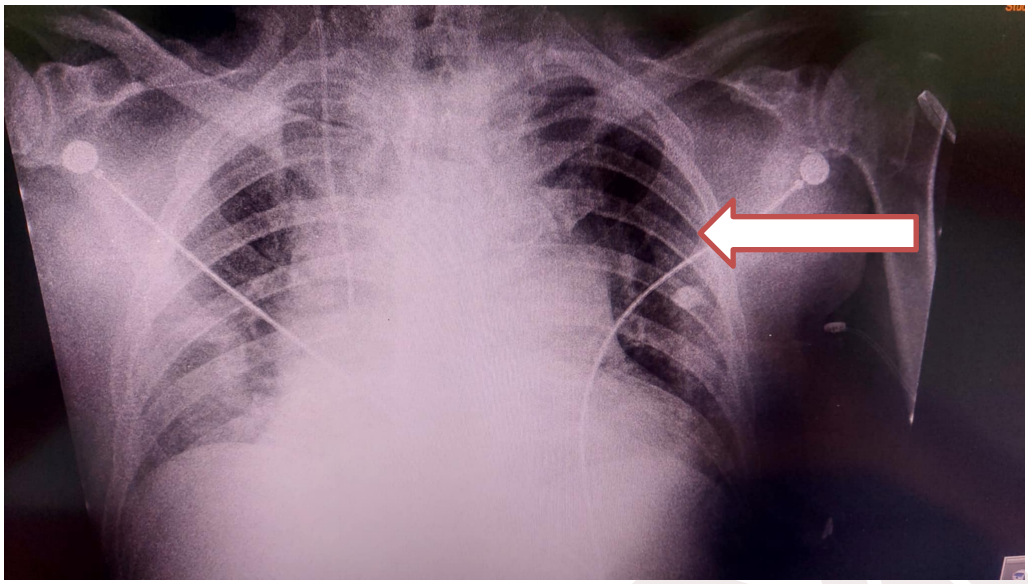


Figure 2. Chest X-ray – 22.01.2022

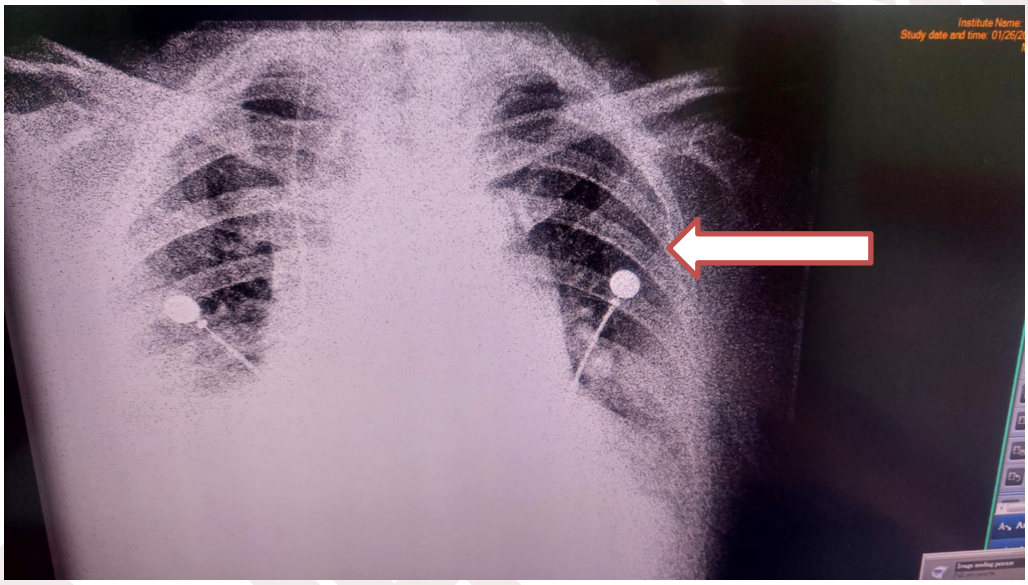


Figure 3. Chest X-ray – 26.01.2022

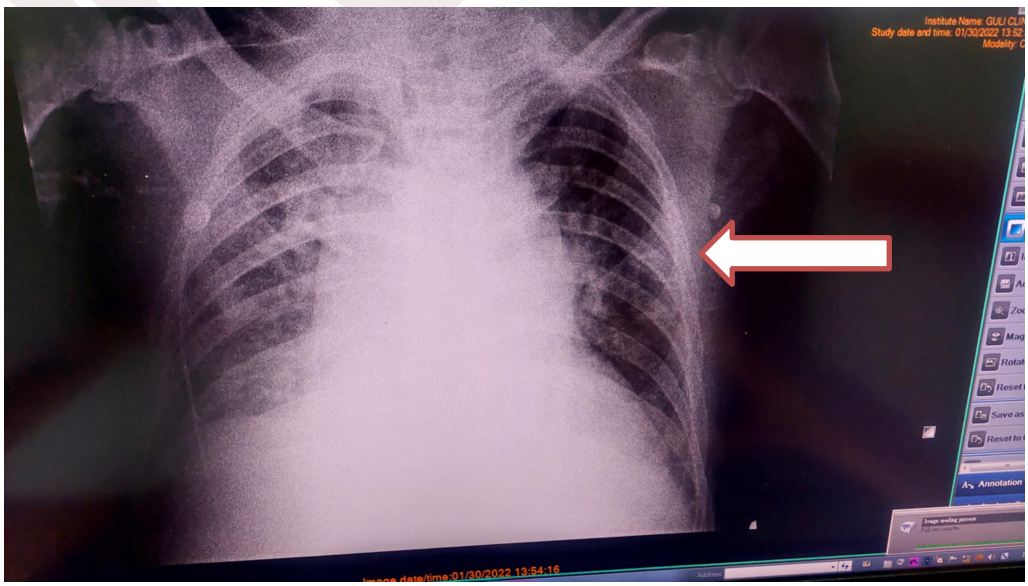
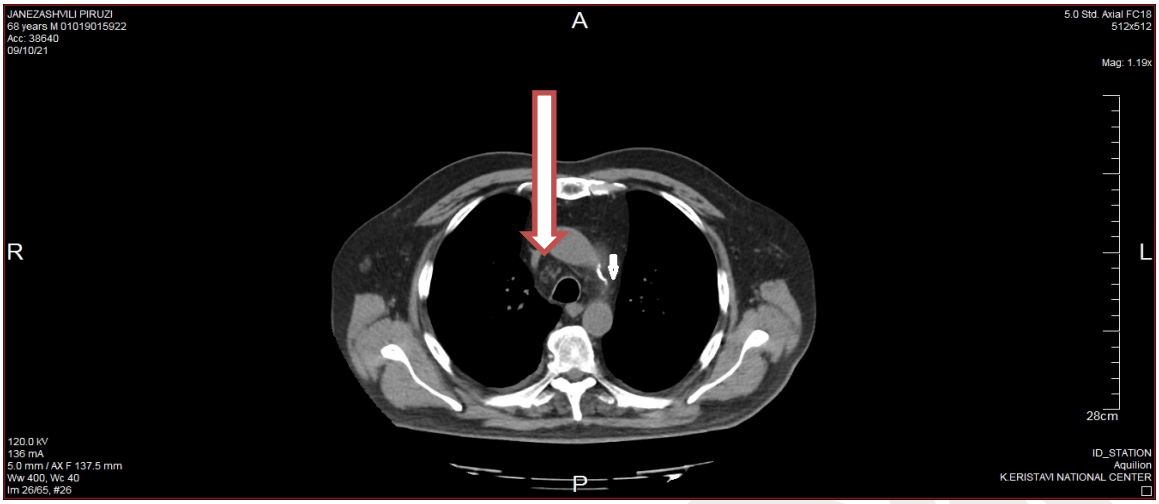
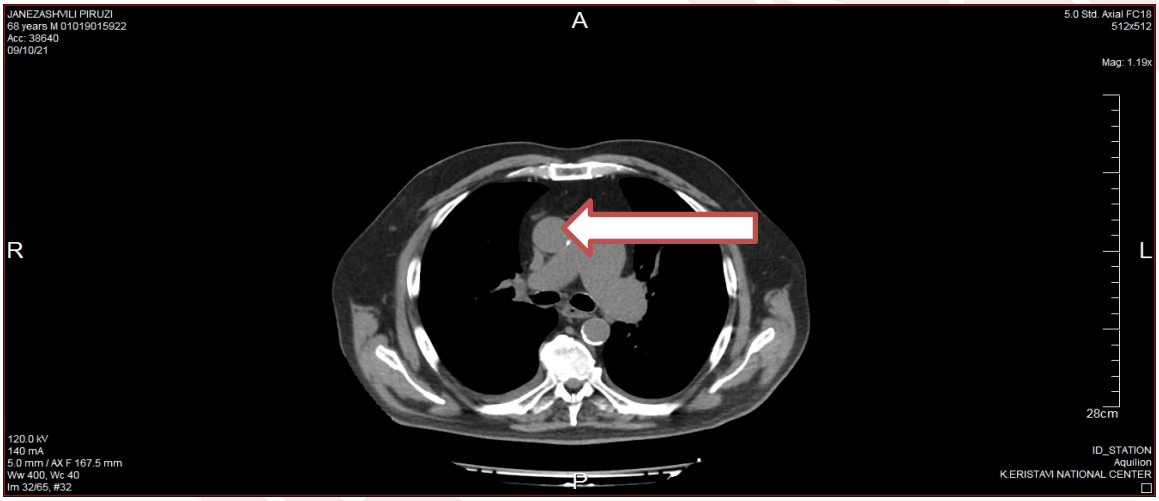


Figure 4. Chest X-ray – 30.01.2022

CT1 - Ductus Botalli



CT-2 - Pulmonary Trunk Dilation



Laboratory and instrumental data (Tables 1 and 2) reflect the clinical course from 2021 through 2024, demonstrating improved inflammatory markers, renal function, and cardiac performance. Radiological imaging (X-rays and CT) supported postoperative anatomical changes and reduction in pleural fluid.

Prognosis. Discussion of long-term outcomes remains complex due to the initial severity of the disease. However, clinical remission has been achieved, and no additional complications have been identified to date.

Conclusion. This case is notable from both theoretical and clinical perspectives. It emphasizes the role of COVID-19-associated pneumonia in the decompensation and diagnosis of previously unrecognized congenital pulmonary artery stenosis. Post-surgical management has transformed the acute pathology into a chronic and controllable condition, with sustained remission achieved through multidisciplinary care.

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CONGENITAL PULMONARY ARTERY STENOSIS DIAGNOSED AFTER COVID-19-ASSOCIATED PNEUMONIA IN AN ELDERLY PATIENT WITH MULTIVALVULAR PATHOLOGY AND SUBSEQUENT SUCCESSFUL SURGICAL TREATMENT

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SUMMARY

Congenital pulmonary artery stenosis is predominantly caused by genetic factors and embryonic developmental defects. Due to its association with other congenital heart anomalies, clinical manifestations are often complex and typically present during childhood or adolescence. Diagnosis at an advanced age is extremely rare. Contributing factors such as viral infections can provoke symptom onset.

This paper presents the clinical case of a 69-year-old male patient diagnosed with congenital pulmonary artery stenosis following COVID-19-associated pneumonia. The case was further complicated by comorbidities including combined valvular defect, ischemic cardiomyopathy, essential hypertension, atherosclerotic heart disease, secondary pulmonary hypertension, stage III chronic kidney disease, obesity caused by excessive caloric intake, and type 2 diabetes mellitus. The patient underwent surgical treatment including bioprosthetic replacement of the aortic and pulmonary valves, transannular patch enlargement of the right ventricular outflow tract, and mitral valve annuloplasty. Despite the initially unfavorable prognosis, symptom severity decreased and the patient's condition improved significantly during follow-up.

Keywords: Pulmonary stenosis, Covid-associated, combined defect, balloon valvuloplasty