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Case Report

A clinical case of a patient with congenital heart defect and a pacemaker dysfunction

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ABSTRACT

Ebstein anomaly (EA) is a congenital heart defect characterized by malformation of the tricuspid valve, leading to various cardiovascular complications, including arrhythmias and heart failure. This case report presents the clinical journey of a 33-year-old female patient with a complex medical history, including congenital heart disease (EA) and multiple interventions related to her cardiac condition, including a mechanical tricuspid valve prosthesis. Following a recent dual-chamber pacemaker implantation, the patient experienced acute episodes of sharp, electric shock-like pain in the epigastric region, and X-ray imaging revealed dislocation of the atrial electrode. Re-implantation of the atrial electrode was performed; however, the postoperative period was marked by fluctuations in the patient's condition. A multidisciplinary team involving cardiologists, clinical pharmacologists, and cardiothoracic surgeons was assembled to manage her care. The patient's recovery trajectory showed positive dynamics, with stabilization of her symptoms and resolution of pleural effusions over time.

Keywords: Ebstein anomaly, Single-chamber pacemaker implantation, Electric shock waves atrial flutter, AV block, Dislocation of the atrial electrode, Holter monitoring, Thrombotic risk, Anticoagulation therapy

INTRODUCTION

Ebstein anomaly (EA) is uncommon, however significant congenital heart defect, affecting less than 1% of congenital disorders and occurring in approximately 1 in 200,000 live births.[1-5] This condition is characterized by a malformation of the tricuspid valve and impacts the right ventricle (RV).[6] It can manifest as cyanosis in neonates or exercise intolerance in older patients, with symptoms capable of appearing at any age. First described by Wilhelm Ebstein in 1866, EA presents with distinctive features such as the downward displacement of the septal leaflet of the tricuspid valve, an enlarged right atrium, and a thinned, dilated section of the RV.[7,8] It is often associated with other congenital disorders, such as mitral valve prolapse and left ventricular noncompaction.[9,10] Accurate diagnosis relies on advanced imaging techniques, including echocardiography and cardiac magnetic resonance imaging, which are essential for assessing the condition and planning effective surgical interventions.[11] Notably, about 39% of patients may display left heart anomalies, potentially leading to left ventricular dysfunction. Healthcare providers, especially nurses, must be adept at recognizing the signs and symptoms of EA to be sure thorough and effective patient management, given the variable presentations of this condition.

Pathophysiologically Ebstein's anomaly is a congenital cardiac malformation characterized by abnormalities of the right ventricle and the tricuspid valve. In a healthy heart, the tricuspid valve comprises three leaflets named as: the anterior, posterior, and septal leaflets. However, in individuals with Ebstein's anomaly, significant developmental abnormalities are present, primarily resulting from incomplete delamination-an embryological

process where in valve tissue fails to separate adequately from the underlying myocardial structure.[12]

Although the conduction pathways typically remain in a standard configuration, the atrioventricular node may experience compression from aberrant fibrous formations. Besides the right bundle branch might also be compromised by extensive fibrotic tissue. Nonetheless, complete heart block remains a relatively uncommon consequence. The anomalous positioning results in creating an incoherence of the septal tricuspid leaflet among the central fibrous body and the septal fibrous atrioventricular ring, which may account for the elevated occurrence of pre-excitation syndromes in this patient population.

Ebstein's anomaly encompasses a spectrum of morphological manifestations, relocation of the posterior and septal leaflets ranging from minimal to severe cases involving obstructive membranes or muscular shelves. The anterior leaflet typically becomes presented as unwanted and may contain several fenestrations, with short and inadequately formed chordae tendineae.

Conduction abnormalities in individuals who are diagnosed with Ebstein's anomaly commonly exhibit disturbances within the conduction system, usually stemming compression from the atrioventricular (AV) node and associated structural abnormalities. Occurrence of the right bundle branch block is prevalent, and the anatomical features of the atrialized right ventricle augment its potential for arrhythmias.[13] The distinctive findings on an electrocardiogram (ECG) include elevated and broad P waves indicative of right atrial enlargement, along with right bundle branch block and tachyarrhythmia's linked with accessory conduction pathways and Wolf-Parkinson-White syndrome. Prominent symptoms of this condition encompass exertional dyspnea (71.5%), exertional palpitations (36.5%), and cyanosis (30.2%).[14,15] These symptoms may arise at various ages, predominantly between six months and 51 years, with an estimated 48% of patients demonstrating cyanosis. While the presence of atrial paralysis and AV block is infrequent, accessory pathways are frequently observed in this patient population.[16] Preexcitation syndromes affect up to 10% of those affected, and approximately 30% experience symptomatic arrhythmias such as atrial flutter or fibrillation. Patients often present with significant clinical symptoms, including atrial flutter accompanied by abnormal conduction. They may also display bradycardia and second-degree AV block under certain circumstances, highlighting the imperative for diligent monitoring and management. In conclusion, Ebstein's anomaly poses substantial arrhythmic risks and presents a range of clinical symptoms, necessitating comprehensive monitoring and strategic management approach.

Regular physical exercise has been linked to minimize the risk of future obesity and ischemic heart disease. For individuals who have congenital heart defects, tailored physical activity recommendations are essential. A study conducted by Werner Budts and colleagues emphasizes the importance of personalized exercise prescriptions for adolescents and adults facing these unique challenges.

In cases related with pacemaker endocarditis which are caused by *Propionibacterium acnes* in adult patients with Ebstein's anomaly, empirical treatment often consists of intravenous antibiotic therapy, typically with ceftriaxone and gentamicin for a duration of 14 days, followed by a course of oral doxycycline upon discharge.[17]

Management strategies for patients suffering from cardiomegaly linked to Ebstein's anomaly may involve diuretic therapy. significant heart-related issues such as non-patent ductus arteriosus aneurysm and bradycardic atrial fibrillation.[18]

Operational interventions for Ebstein's anomaly may include: -Tricuspid valve reconstruction or replacement when feasible.[19] Closure of atrial septal communications, correction of concurrent anomalies (e.g., closure of ventricular septal defects).[20,21] Antiarrhythmia procedures (e.g., maze procedure using cryoablation or radiofrequency ablation).[22] Selective plication of the atrialized right ventricle.[23] Right reduction atrioplasty

The outcomes of tricuspid valve fixation and replacement have been well-documented. For adults where valve repair is not viable, tricuspid valve replacement, particularly with a porcine bioprosthetic valve, is often recommended due to its commendable durability and the absence of the need for chronic warfarin anticoagulation. Unlike mechanical valves, which can have a greater incidence of malfunction and thromboembolic events, porcine bio prostheses usually require only about three months of warfarin anticoagulation, followed by lifelong aspirin therapy.

In older adults, particularly those over 50 years, EA management presents additional complexities. Factors such as significantly enlarged hearts, the prevalence of atrial tachyarrhythmias, atherosclerotic coronary artery disease, and other acquired valve conditions, alongside common comorbidities like diabetes and hypertension, complicate perioperative care. Nonetheless, the longevity of porcine bio prostheses makes valve replacement a pragmatic choice in cases where repair is not feasible.

In terms of pacemaker therapy, all patients generally receive atrioventricular pacemakers that utilize bipolar epicardial steroid-eluting leads.[24-26] This approach optimizes cardiac output and mitigates the risk of atrial arrhythmias stemming from premature atrial beats. The advancement in pacemaker technology has develop to the development of atrioventricular anti tachycardia dual-chamber pulse generators and multisite ventricular pacing capabilities, which are particularly beneficial for patients with dyskinetic ventricles or those requiring automatic cardiac defibrillators for ventricular tachycardia.

During the process of pacemaker implantation, challenges can arise, such as difficulties passing the ventricular lead through the tricuspid valve for right ventricular pacing, as noted in clinical experience. In such cases, alternative leads designed for coronary sinus cannulation can be utilized to facilitate left ventricular pacing.

Traditional trans-venous cardiac pacemakers present several inherent challenges, particularly related to lead and device pocket complications.[27] These issues can arise during the implantation procedure as well as during the subsequent follow-up period.

The transvenous lead is the utmost vulnerable element within the pacemaker system, as it operates in a mechanically demanding environment, subjected to repetitive stress during each of the cardiac cycles and shoulder movement. This vulnerability can result in lead failure in a small percentage of patients. Additionally, the lead may interfere with the tricuspid valve leaflet motion or the subvalvular apparatus, potentially leading to significant tricuspid valve dysfunction. Moreover, the extravascular nature of the pulse generator poses a risk of infection following surgical procedures related to the device, as the attached leads may facilitate bacterial entry into the bloodstream, creating a nidus for recurrent bacteremia after initial infections.[28]

Additional complications may include a lack of a practical and long-lasting power source, thromboembolic events, mechanical ectopy, and damage to valvular or subvalvular structures.[29] Myocardial perforation and worsening heart failure may necessitate elective explantation. Vascular access complications, such as bleeding, arteriovenous fistula, pseudoaneurysm, and complications involving vascular closure devices, are of significant concern. Moreover, cardiac effusion and perforation may occur, along with lack of ventricular capture, particularly due to thrombus formation at the tip of leadless pacemakers.[30]

These complications are associated with higher mortality rates, prolonged hospital stays, and the necessity of reinterventions and intensive care. Infections were tracked particularly if they necessitated extended or additional hospitalization, including those from other devices or implants in the cardiac or vascular systems.

Cardiac injuries encompass a range of issues, pericardial effusions, pericarditis, and cardiac tamponade. Bleeding that necessitates blood transfusion in patients with pacemaker systems.[31,32] The economic burden includes extensive drug therapy and implant costs during hospitalization.

CASE REPORT

Upon admission, the 33-year old female patient presented to Grodno State clinical cardiology center with complaints with painful sensations in the epigastric region slightly to the right of the postoperative scar, which she describes as "electric shocks" of varying strength, 5-6 shocks every few hours, more strongly felt in the supine position; shortness of breath when walking; general weakness, heavy menstrual bleeding.

The patient has a history of congenital heart disease-EA. In 2001, a biological prosthesis of the tricuspid valve was implanted. Five years later, in 2006, prosthesis was replaced with a 29 mm mechanical St. Jude prosthesis. On January 12, 2007, she underwent single-chamber pacemaker implantation for grade 2 AV block, Mobitz types 1 and 2. In 2013, pacemaker re-implantation took place.

In April 2020 during a pacemaker checkup atrial flutter was detected for the first time. So electrical cardioversion was performed and restoration of the sinus rhythm was done. In January 2022, she had a Covid-19 infection, against this background, again atrial flutter was detected and the sinus rhythm was restored.

In April 2024 patient was admitted at the Grodno State clinical cardiology center for re-implantation of the pacemaker. During the operation an implantation of an atrial electrode was performed and a single-chamber pacemaker was switched to dual-chamber one. The postoperative period was uneventful and she noted an improvement in her well-being, as well as decrease in shortness of breath.

However, on 09/05/2024 she was felt a series of sharp pain attacks in the epigastric region, to the right of the postoperative scar, which she described as "electric shocks"; she did not feel palpitations, pre-syncope, syncope and she didn’t seek medical help immediately. In the following 3 days, the above complaints recurred (blows of varying strength in series of 5-6 blows every few hours), felt more strongly in the supine position.

During the pacemaker check-up episodes of ineffective atrial stimulations were detected X-ray showed the dislocation of the atrial electrode (Figure 1). So, the atrial electrode was temporarily disconnected and the patient hospitalized for the further examinations.

An EchoCG (M+B mode+Doppler+color map+tissue Doppler examination on 09/11/2024 was done and it indicated that the hemodynamic parameters of the prosthesis were unchanged. Tricuspid regurgitation grade was 2-3. Also, dilation of the right ventricle cavities and both atria was determined in the cavity of the left ventricle, alongside with diastolic dysfunction of the left ventricular myocardium.

Holter monitoring on 09/16/2024 showed sinus rhythm with periods of atrial rhythm with complete intraventricular block (QRS width-140 ms). AV block grade 1 (PQ interval duration up to 300 ms) and episodes of pacemaker rhythm (AsVp) with heart rate of 50 bpm were also determined.

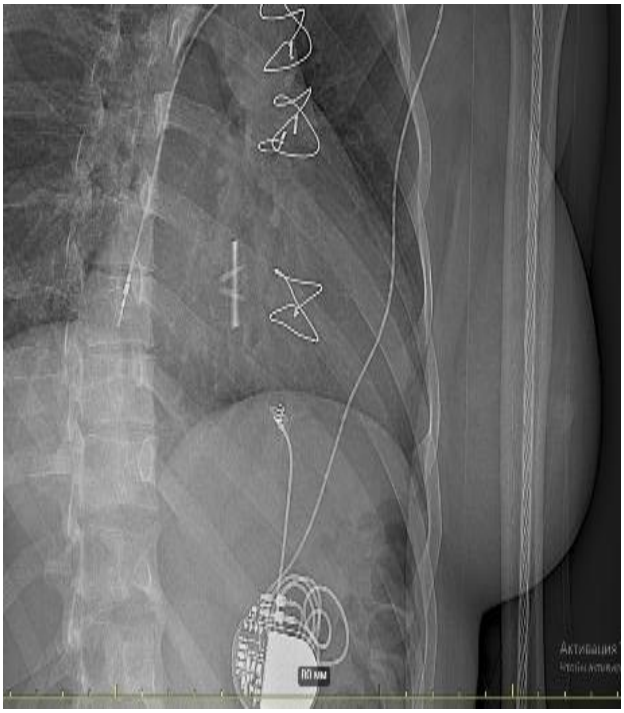

Figure 1: Dislocation of the atrial electrode on X-ray.

On 09/18/2024 patient was consulted by the Council of cardiologists and cardio surgeons. Consilium stated that patient had high thrombotic risk due to mechanical prosthesis of tricuspid valve and also high risk of bleeding associated with pacemaker electrode repositioning. Decisions included reposition of the atrial pacemaker electrode and warfarin dosage reduction to 1 tablet per day; INR monitoring and administration of low molecular weight heparin (LMWH) 12 hours prior to surgery if INR drops below the target.

Next day 09/19/2024 re-implantation of the atrial electrode of a dual-chamber pacemaker operation was performed. The patient underwent general anesthesia, the wound in the epigastric region was dissected, the pacemaker was removed, the atrial electrode was disconnected. After performing general anesthesia, the wound on the left in the subclavian region was dissected. The electrode was released from fixation in soft tissues. The atrial electrode was extracted, and the electrode was removed.

By puncture, an intraducer was installed in the left v. subclavia and through it a pericardial electrode was installed in the interatrial septum, the stimulation threshold of the atrial electrode is -0.5V, sensitivity 2.5mV. A subcutaneous tunnel was formed bluntly from the subclavian region on the left to the epigastric region. The pacemaker was placed in the bed, the electrode was connected to it, the rhythm was imposed in the DDD=60 bpm mode. The atrial electrode stimulation threshold is -0.5V, sensitivity is 2-2.5mV, the ventricular electrode stimulation threshold is -2.75V, >5.0mV. and finally, intraducer was removed sutured in layers. No complications were noted.

On the same day patient had chest X-ray and it's was all normal without focal and infiltrative shadows (Figure 2).

After the operation patient had complaints on the pain in the area of postoperative wounds and pain along the ribs and epigastric region, her body temperature increased to subfebrile with general weakness. The next day morning temperature was 37.3. The wounds were bandaged, and the dressings were dry in the morning.

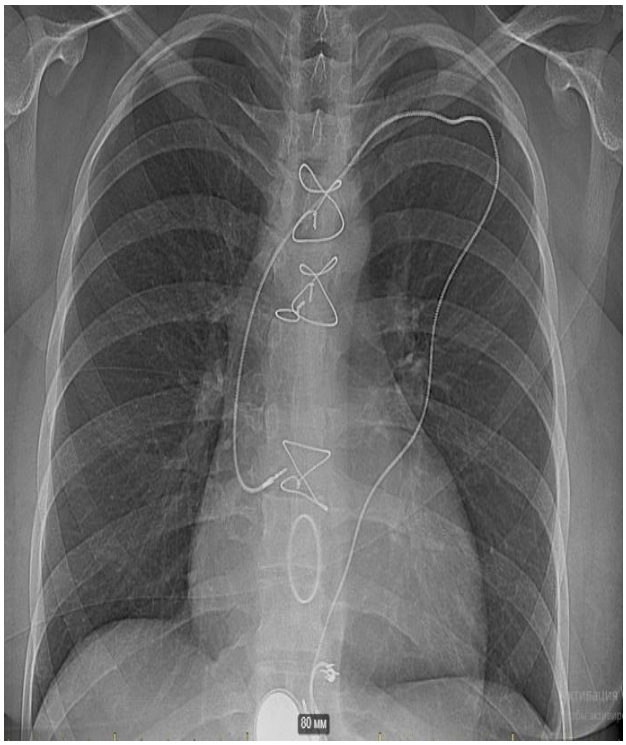

Figure 2: Patients X-ray after the operation.

The next day patient underwent pacemaker check-up. No disturbances in pacemaker were found. Throughout the recovery phase, the patient's condition was closely monitored, and additional tests were conducted to ensure that the patient was recovering well. The ECG showed a regular heartbeat, and the ST segment had returned to normal. The echocardiography revealed no abnormalities and local myocardial contractility index was 1.

On 09/23 the ultrasound of the pleural cavities and pericardium showed no fluid in the pericardial cavity

In the left pleural cavity, fluid was determined as a layer of fluid above the dome of the diaphragm up to 18 mm thick, volume 200 ml. In the right pleural cavity, fluid was determined as a layer of fluid above the dome of the diaphragm up to 18 mm thick, volume 200 ml.

Ultrasound of the pacemaker bed was also performed on 09/23. On the left in the subclavian region, in the projection of the wound, imbibition of soft tissues was visible (Figure 3).

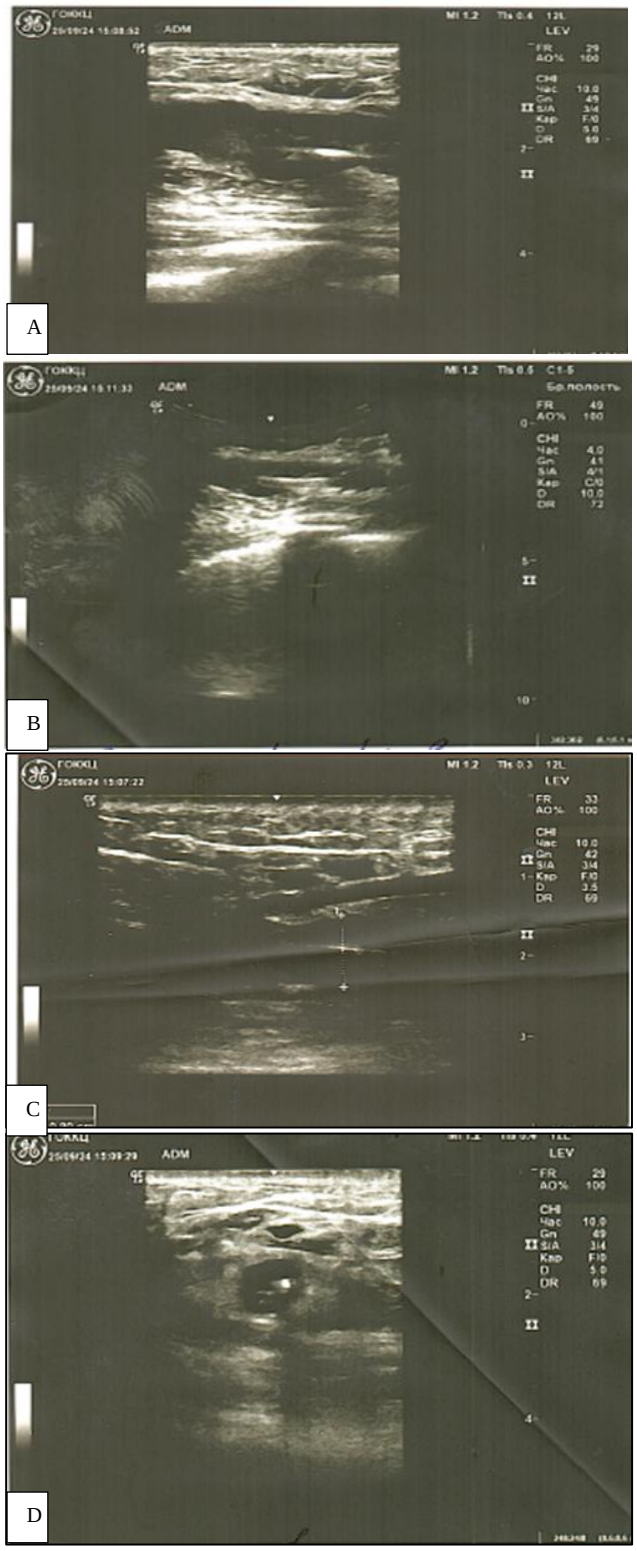

Figure 3 (A-D): Ultrasound of the pacemaker bed and electrode channel. Ultrasound of the pacemaker bed (linear sensor). Ultrasound of the pacemaker bed (convex sensor) on the left in the subclavian region, in the projection of the pacemaker bed subcutaneously, a fluid formation 47×14 mm in size is located. Ultrasound of pacemaker electrode in longitudinal position and ultrasound of pacemaker electrode in transverse position.

Along the entire length of the subcutaneous tunnel of the electrode, a strip of fluid with thickness of 9 mm is visible.

In her blood tests procalcitonin level was less than 0.05; however, CRP was elevated up to 39.6 mg/l. In venous blood cultures no microbial growth was detected.

During the next 2 days the same complaint of pain appeared along the ribs and the temperature was increased to 38C. However, on X-ray no lung infiltration was found. During this period patient was receiving ceftriaxone 1.0 mg intravenously 2 times a day for antibacterial purposes.

On 09/23 the patient again was consulted by the council of cardiologists, cardio surgeons and clinical pharmacologists. Taking into account the patient's persistent hyperthermic syndrome despite the ongoing antibacterial therapy, the surgical intervention performed, the ultrasound data of the pleural cavities and the pacemaker bed, a very high risk of infectious complications, it was recommended to prescribe teicoplanin to the patient for antibacterial purposes at a dosage of 400 mg intravenously by drip 2 times a day for 2 days (loading dose), followed by a transition to a maintenance dose of 400 mg intravenously once a day, under the control of thermometry, levels of complete blood count, urine analysis, bacterial blood test, procalcitonin, ultrasound of the pleural cavities and pericardium.

The next day on 09\24 the patient reports a condition with slow positive dynamics, pain in postoperative wounds and along the ribs decreased. Body temperature readings indicated mild hypothermia in morning (35.9°C) dressing in the left subclavian region was slightly soaked with blood. Due to the continuing negative dynamics of fluid accumulation, colchicine 0.5 mg twice daily was added.

On the 09/26 the patient showed positive dynamics, reporting improved general well-being, reduced pain, and increased physical activity. Morning temperature was stable at 36.0°C, with normothermia maintained throughout the day. So, the antibiotic therapy with teicoplanin 400 mg intravenously was continued once daily, with monitoring of body temperature.

The following Table 1 and 2 show before and after operation laboratory results.

Upon the discharge condition of the patient was satisfactory, with positive dynamics. From September 26, 2024, normothermia has been maintained, postoperative wounds were healing well, sutures were removed on September 30, 2024. In the epigastric region, left subclavian region, and in the area of both mammary glands, hematomas in the resolution stage remained. According to ultrasound of the pleural cavities and pericardium, there was no fluid at discharge. Positive dynamics of laboratory parameters of the complete blood count and biochemical blood count were observed, INR remained within the target values (2.5-3.5).

Table 1: Complete blood count.

Date	09/11/2024	09/19/2024	09/23/2024	09/30/2024
ESR (mm/hour)	5 (Normal)	4.23	10	22 High
WBC (10^9/L)	9.19 (Normal)	15.8 (Elevated)	8.49 (Normal)	7.28 Normal
RBC 10^{12}/L	4.29 (Normal)	4.23 (Normal)	3.29	3.66 Low
HGB (g/L)	134 g/l (Normal)	131 g/l (Normal)	106 g/l (Low)	113 Low
HCT%	40.1% (Normal)	39.1% (Normal)	31% (Low)	34.9 (Low)

Table 2: Biochemical blood tests which data need to be keep.

Date	09/11/2024	09/19/2024	09/23/2024	09/27/2024
Urea	5.46 mmol/l (Normal)	3.6 mmol/l (Normal)	3.4 mmol/l (Normal)	2.97 mmol/l (Normal)
Creatinine	74 µmol/l (Normal)	53 µmol/l (Normal)	54 µmol/l (Normal)	62 µmol/l (Normal)
C-reactive protein	6.6 mg/l (Slightly elevated)		39.6 mg/l (Significantly elevated)	14.2 (Elevated)

After postoperative care and monitoring, the patient was discharged from the hospital with a stable condition. The patient was directed to follow up with the treating physicians periodically for further management and monitoring.

Also, her recommendation included-Observation by a cardiologist at the outpatient clinic of the State regional cardiological center, a primary care physician at the place of residence, if indicated-consultations with a cardio surgeon of the Regional cardiological center. Daily thermometry (morning, evening) throughout the recovery period. Monitoring of general and biochemical blood tests 5 days after discharge. Monitoring of INR levels in dynamics. Limitation of physical activity along the left arm and shoulder for 3 months. Warfarin 2.5 mg, alternate taking 2.5 and 3 tablets, dose selection under control of target INR values (2.5-3.5) Under the supervision of a GP at the place of residence. Colchicine 0.5 g, 1 ablet 2 times a day in the morning and in the evening for 7 days after discharge, followed by complete cancellation. Continuation of dressing of postoperative wounds, local applications of heparin-containing ointment to the area of postoperative hematomas 2-3 times a day. Pacemaker check-up after 3 months (January 2025) at the state regional cardiology center.

DISCUSSION

This case report presents the clinical journey of a 33-year-old female patient with a complex medical history, including congenital heart disease (EA) and multiple interventions related to her cardiac condition.[1-3] The patient's presentation, characterized by epigastric pain, shortness of breath together with general weakness, highlights the multifaceted challenges faced by individuals with significant cardiac history undergoing procedural interventions.

The patient underwent several significant cardiac procedures, including the replacement of a tricuspid valve prosthesis and multiple pacemaker implantations.[19] Her history of atrial flutter, particularly following a COVID-19 infection, raises concerns about the impact of viral infections on cardiac rhythm disturbances and thromboembolic risks. Her management was further complicated with the existence of a mechanical tricuspid valve due to the inherent thrombotic risks associated with mechanical prostheses, necessitating careful anticoagulation management.

Following the recent dual-chamber pacemaker implantation, the patient experienced acute episodes of sharp, electric shock-like pain in the epigastric region, indicative of potential complications.[24-26] The dislodgment of the atrial electrode detected in the time of pacemaker check-up is a critical finding that may explain her symptoms.[33] The decision to disconnect the electrode and conduct further evaluations was prudent, considering the potential for significant complications such as cardiac tamponade or ineffective pacing.[31,32]

The postoperative period was marked by fluctuations in the patient's condition, including episodes of pain, fevers, and fluid accumulation in the pleural cavities. The ultrasound findings of pleural effusion and the appearance of hematomas in the surgical region underscore the complexity of her recovery, particularly taking into attention her anticoagulation therapy with warfarin.

Laboratory results indicated elevated inflammatory markers (CRP) and variations in haemoglobin levels, suggesting an ongoing inflammatory process or potential bleeding complications. The use of imaging, including echocardiography and ultrasound, was essential in monitoring the patient’s cardiac function and the status of fluid collections, guiding clinical decisions regarding the management of her postoperative complications.[11]

The multidisciplinary approach involving cardiologists and cardiothoracic surgeons was crucial in managing the patient’s care. The decision to reduce warfarin dosage and initiate LMWH prior to the atrial electrode repositioning

was a well-considered strategy to mitigate thrombotic risks while addressing the potential for bleeding complications.[30]

The introduction of teicoplanin for potential infections, coupled with colchicine to manage pleural effusion, reflects an evidence-based approach to addressing the patient's inflammatory and infectious concerns. Regular monitoring of INR and CRP levels further demonstrates a commitment to personalized medicine, ensuring that anticoagulation therapy is tailored to the patient's evolving clinical status.

The patient's recovery trajectory showed positive dynamics, with stabilization of her symptoms and resolution of pleural effusions over time. The discharge plan emphasized the importance of follow-up care, including regular monitoring of INR, dietary modifications, and restrictions on physical activities. Such measures are vital in preventing complications such as bleeding or thromboembolism, patient who holds a mechanical heart valve and recent surgical intervention.

CONCLUSION

This case highlights the intricate interplay between congenital heart disease, cardiac interventions, and postoperative complications, particularly regarding the management of patients with mechanical heart valves and pacemaker systems. The patient's experience with electrode displacement serves as a critical reminder of the potential complications associated with device implantation, underscoring the need for vigilant monitoring and timely intervention.

The dislodgment of the atrial electrode not only contributed to the patient's acute symptoms but also emphasized the importance of routine device check-ups in identifying issues that may not present immediately but can have significant clinical ramifications. This case reinforces the necessity for healthcare professionals to maintain a high index of suspicion for complications, particularly in patients with complex medical histories.

Furthermore, the multidisciplinary approach adopted in this case incorporating cardiologists, cardiothoracic surgeons, and clinical pharmacologists demonstrates the value of collaborative decision-making in optimizing patient outcomes. The careful management of anticoagulation, especially in the context of potential thromboembolic risk associated with mechanical prostheses, is crucial and should be tailored to individual patient needs.

Future research should focus on establishing standardized protocols for monitoring and managing pacemaker electrodes, particularly in patients with complex cardiac histories. Additionally, the exploration of innovative techniques for electrode fixation and placement could enhance the stability and longevity of these devices, potentially reducing the incidence of dislocation and associated complications.

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