

#### **CASE REPORT**

# From Device to Disease: Tricuspid Valve Replacement in a Patient with Copper T Associated Endocarditis and Thrombotic Thrombocytopenic Purpura

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#### **Abstract**

Background: Infective endocarditis involving the tricuspid valve is rare, typically seen in intravenous drug users or patients with indwelling vascular devices. Intrauterine device (IUD)-associated infective endocarditis is exceedingly uncommon, with very few cases documented. Even more unusual is the concomitant occurrence of thrombotic thrombocytopenic purpura (TTP), a rare and life-threatening thrombotic microangiopathy, in the setting of infective endocarditis. Here, we report a rare case of Copper T-associated tricuspid valve infective endocarditis complicated by TTP requiring surgical valve replacement and aggressive medical therapy.

**Keywords:** Tricuspid valve endocarditis, intrauterine device (IUD), Copper T, thrombotic thrombocytopenic purpura (TTP), infective endocarditis (IE).

#### Introduction

Tricuspid valve endocarditis accounts for only 5% to 10% of all infective endocarditis cases and is predominantly observed in intravenous drug users or patients with indwelling central venous catheters [1,2]. Infections related to intrauterine devices (IUDs), particularly the Copper T, are exceedingly rare, with only isolated case reports describing progression to right-sided endocarditis. The risk of IUD-associated systemic infection is estimated to be

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less than 1 per 1,000 insertions [3,4]. Thrombotic thrombocytopenic purpura (TTP) is a rare thrombotic microangiopathy with an incidence of approximately 3 to 10 cases per million annually. TTP can be idiopathic or triggered by infections, surgeries, or autoimmune processes [5,6].

#### **Case Report:**

A 38-year-old previously healthy female presented with a 2 months history of fever, dyspnoea, and generalized weakness. She had no history of intravenous drug use, cardiac disease, or any recent surgeries. However, on detailed inquiry, she reported a Copper T IUD insertion approximately 5 years prior. A month ago, she developed fever and vaginal discharge. She consulted a gynaecologist at Patan Government Hospital, and the infected Copper T IUD was removed. Her symptoms of fever, dyspnoea, and generalized weakness were persisted after the IUD was removed. Then she was referred to U. N Mehta Institute of Cardiology and research centre, Ahmedabad for further management.

On admission, Physical examination shows Fever, tachycardia, hypotension, tachypnoea, and Systolic murmur best audible at the left lower sternal border as well as mild peripheral oedema with skin lesions of all limbs which revealed signs of sepsis. Diagnosis of TTP based on laboratory findings of microangiopathic haemolytic anaemia (MAHA), thrombocytopenia, elevated lactate dehydrogenase (LDH) and plasmic score. Blood cultures grew methicillin sensitive Staphylococcus aureus confirming Infective endocarditis. Transthoracic echocardiography revealed a severe Tricuspid Regurgitation (TR), vegetations (32\*28 mm) on the tricuspid valve - large echogenic structure attached to Anterior Tricuspid Leaflet, suggestive of vegetation (Figures 1 and 2). Right atrium and ventricle - mild dilated EF- 60% no RWMA at rest and Mild pericardial effusion.



Figure 1: TTE- Apical four chamber view shows large echogenic structure on Tricuspid valve



Figure 2: TTE- Subcostal four chamber view shows large echogenic structure on tricuspid valve

In Pre-operative Management, a haematologist recommended plasmapheresis (3 cycles) to manage TTP, improve platelet count, and prevent haemolysis. Stabilization of Acute Kidney Injury (according to RIFLE criteria) and optimization of hemodynamic in septic shock. Empirically Broad-spectrum antibiotics (MEROPENEM, TEICOPLANIN AND CASPOFUNGIN) for infective endocarditis given for 2 weeks. Despite of medical therapy, the patient developed persistent sepsis and worsening right-sided heart failure, necessitating surgical replacement of the tricuspid valve due to severe TR, failure of medical management, and refractory right-sided heart failure. Cardiac surgery was performed under general anaesthesia, and valve replacement was done using a mechanical prosthesis (Figure 3).

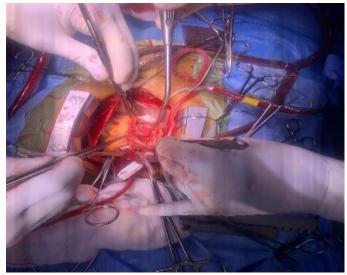


Figure 3: Valve replacement was done using a mechanical prosthesis

In pre-operatively, with stabilization of hemodynamic patient was extubated within 24 hours of surgery. Plasmapheresis (2 cycles) and Corticosteroid therapy were continued post-operatively to prevent recurrence of TTP as advised by haematologist. Intensive

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care monitoring for sepsis, acute kidney injury and respiratory support. The patient underwent renal replacement therapy after consulting a nephrologist because her urine output was decreased during the post-operative period.

Tissue of tricuspid valve vegetation was sent from OT which grew STENOTROPHOMONAS MALTOPHILIA. Antimicrobials adjusted as it was only sensitive to MINOCYCLINE. On 2 D echo findings Tricuspid valve prosthesis in situ, No residual regurgitation, No pericardial effusion and Gradual improvement in renal parameters and hemodynamic post-surgery. Complete recovery of platelet counts and resolution of TTP after plasmapheresis with stable respiratory and cardiovascular function as showed in Figures 4 and 5.



Figure 4: Healed skin lesion



Figure 5: Healed skin lesion

The patient showed gradual hematologic and clinical recovery. The patient was discharged home in a stable condition on oral antibiotic MINOCYCLINE for total of six week after surgery, scheduled for haematology follow-up for TTP surveillance. At 3month follow-up, she remained asymptomatic with normal valve function and no recurrence of TTP.

#### **Discussion:**

Infective endocarditis (IE) involving the tricuspid valve is an uncommon clinical entity, accounting for approximately 5-10% of all IE cases, and is most frequently observed in intravenous drug users, patients with intracardiac devices, or those with longterm central venous access [7]. Isolated tricuspid valve endocarditis in the absence of these risk factors is rare. Even more exceptional is the occurrence of IE in association with intrauterine devices (IUDs), such as the Copper T, which are not traditionally implicated in systemic infections or cardiac complications [8].

The pathogenesis of IUD-associated IE is not well understood, but it is hypothesized that ascending genital tract infections can provide a portal of entry for pathogens into the bloodstream, leading to hematogenous seeding of the endocardium in susceptible individuals. Although pelvic inflammatory disease is a more commonly reported complication of IUDs, systemic complications such as endocarditis are exceedingly rare, with only a handful of case reports documented in the literature [9].

The present case underscores an exceptionally rare clinical scenario of tricuspid valve IE associated with a Copper T IUD, with no history of Intra Venous Drug User or vascular access device. This raises important questions regarding host susceptibility, microbial virulence, and potential hematogenous dissemination from genital tract flora in the presence of an IUD. The identification of the causative organism and its correlation with urogenital flora could lend further support to the proposed pathogenesis, though such data may not always be available. An even more unusual aspect of this case is the development of thrombotic thrombocytopenic purpura (TTP) in the setting of active infective endocarditis. TTP is a rare but potentially fatal thrombotic microangiopathy characterized by pentad of microangiopathic haemolytic anaemia, thrombocytopenia, neurologic dysfunction, renal impairment, and fever [10]. The majority of TTP cases are related to a deficiency of ADAMTS13, either congenital or acquired due to inhibitory autoantibodies

Infectious triggers for secondary TTP have been documented, particularly in bacterial infections, but its association with IE remains sparsely reported. The presumed mechanism involves systemic inflammation and endothelial injury leading to excessive von Willebr and factor (vWF) release and microvascular thrombosis in the absence of adequate ADAMTS13 activity [12]. In such settings, distinguishing TTP from disseminated intravascular coagulation (DIC) or sepsisassociated thrombocytopenia is crucial, as management differs significantly. Timely diagnosis and initiation of plasma exchange, immunosuppression, and antimicrobial therapy are critical to survival [6].

Our patient required aggressive therapy including plasmapheresis, corticosteroids, antibiotics, and ultimately surgical valve replacement due to refractory infection and valvular destruction. Surgical intervention in tricuspid valve IE is typically reserved for patients with persistent bacteraemia, large vegetations (>20 mm), septic emboli, or right heart failure [1]. The successful outcome highlights the importance of a multidisciplinary approach in managing such complex cases.

#### **Conclusion:**

This case underscores the rare but serious potential for systemic and cardiac complications arising from intrauterine device (IUD) use, particularly in the context of copper-T insertion. The development of tricuspid valve endocarditis with concurrent thrombotic thrombocytopenic purpura (TTP) represents a unique clinical intersection that demands prompt recognition and

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aggressive, multidisciplinary management. Early diagnosis through echocardiography, blood cultures, and peripheral smear, along with timely initiation of antibiotics, plasma exchange, and surgical intervention, is vital in improving patient outcomes. Clinicians should maintain a high index of suspicion for infective endocarditis in patients with unexplained fever, thrombocytopenia, and intravascular devices—even when the device is extra-cardiac.

**Conflict of interest:** None

**Ethical Consideration:** None

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