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Case Report

Anaesthesia Section

Brachial Vein Thrombosis Following Abdominal Hysterectomy and Hernioplasty under General Anaesthesia: A Case Report

S SREYA SRI[1], POOJA SHAH[2], SARA MARY THOMAS[3]

ABSTRACT

Upper Extremity Deep Vein Thrombosis (UEDVT) is a relatively rare but serious postoperative complication. While lower limb thrombosis is more common after major pelvic surgery, UEDVT, such as brachial vein thrombosis, can occur and poses significant diagnostic and therapeutic challenges. A 46-year-old female presenting with a fibroid uterus and umbilical hernia underwent an uneventful total abdominal hysterectomy, bilateral salpingo-oophorectomy, and hernioplasty under general anaesthesia. Intraoperatively, standard monitoring was used with an intravenous line secured in the right arm. Six hours postoperatively, the patient developed acute pain, swelling, and bluish discolouration of the left hand, which was unresponsive to analgesics. Although peripheral pulses were palpable and a hand X-ray was normal, a venous duplex ultrasound of the left arm confirmed a brachial venous thrombus. No signs of Pulmonary Embolism (PE) were present. The patient was immediately treated with an intravenous Unfractionated Heparin (UFH) infusion for 24 hours, followed by subcutaneous heparin for three days. Her symptoms resolved within 72 hours, and she was transitioned to oral apixaban. Complete resolution of the clot was confirmed via ultrasound at a 3-month follow-up, and anticoagulation was safely discontinued. This case underscores the importance of maintaining a high index of suspicion for UEDVT in the early postoperative period, even in asymptomatic limbs without direct vascular intervention. Prompt diagnostic ultrasound and immediate anticoagulation are critical to managing upper extremity thrombi and preventing thromboembolic complications.

Keywords: Anticoagulants, Apixaban, Heparin, Thrombophlebitis, Vascular diseases

CASE REPORT

A 46-year-old female presented to the gynaecology outpatient department with a one-month history of intermittent, dull lower abdominal pain, four to five days of generalised body ache, and an 8-month history of irregular menstrual cycles (30-60-day intervals, lasting 10-15 days). Following evaluation, she was diagnosed with a fibroid uterus and an umbilical hernia, and was scheduled for a total abdominal hysterectomy, bilateral salpingo-oophorectomy, and hernioplasty. Physical examination revealed a height of 160 cm and a weight of 65 kg. She was haemodynamically stable: blood pressure 110/70 mmHg, heart rate 98 bpm, and SpO2 100% on room air. Respiratory examination revealed normal bilateral vesicular breath sounds. Abdominal examination demonstrated a firm, irregular mass corresponding to a 16-week gestational size uterus with side-to-side mobility, alongside an everted umbilicus with a 3×3 cm hemispherical defect. Basic laboratory investigations were within normal ranges [Table/Fig-1,2]. She had no surgical history or comorbidities.

The patient was classified as ASA Grade 2 for general anaesthesia. Following informed written consent, a 2-hour fasting protocol for clear liquids was advised. Premedication included oral alprazolam 0.25 mg and ranitidine 150 mg at bedtime. Intraoperatively, standard multiparametric monitoring {ECG, pulse oximetry, Non-Invasive Blood Pressure (NIBP) cuff on the left arm} was established, and intravenous (i.v.) Ringer's lactate was initiated via an 18-gauge cannula in the right arm. Baseline vitals were recorded. Pre-anaesthetic medications included i.v. midazolam 1 mg, glycopyrrolate 0.2 mg, and fentanyl 100 mcg. Following preoxygenation with 100% oxygen via facemask, anaesthesia was induced with i.v. propofol 150 mg. Upon verifying intermittent positive-pressure ventilation, i.v. succinylcholine 75 mg was administered. Following lower limb fasciculations, direct laryngoscopy (mac blade size 3) was performed, and a size 7.5 endotracheal tube was passed under direct visualisation. The cuff was inflated with 4 cc of air, fixed at 20 cm, and position confirmed via auscultation of bilateral equal air entry and End-Tidal CO2 (EtCO2). Controlled ventilation was initiated following i.v. atracurium 30 mg anaesthesia was maintained using sevoflurane (MAC 1) with oxygen and nitrous oxide (1:1 ratio).

Parameters	Observed value	Normal range
Haemoglobin	12.5	12.0 to 15.5 g/dL
Total leucocyte count	6300	4,000-11,000 cells/µL
platelets count	3.68×10^5 cells/µL	150,000 to 450,000 cells /µL
Blood urea	16 mg/dL	15 to 40 mg/dL
Serum creatinine	0.6 mg/dL	0.5 to 1.1 mg/dL
Random blood sugar	92 mg/dL	70 to 140 mg/dL
serum sodium	136 mEq/L	135 to 145 mEq/L
serum potassium	4.6 mEq/L	3.5 to 5.0 mEq/L
serum chloride	101 mEq/L	96 to 106 mEq/L
HIV	Negative	
HbsAg	Negative	
HCV	Negative	
ECG	Normal sinus rhythm	
chest X-ray	Normal lung fields	
USG abdomen	The uterus is bulky in size measuring 9.5×5.5×4.8 cm with altered hypoechoic masses largest measuring 2.6×1.7 cm in the anterior myometrium. Both ovaries are normal Right adnexal mass with altered echogenicity measuring 5.7×8 cm subserosal fibroid	
CECT abdomen and pelvis	Benign etiology lesion multiple uterine fibroid likely umbilical hernia	

[Table/Fig-1]: Preoperative investigations of patients.

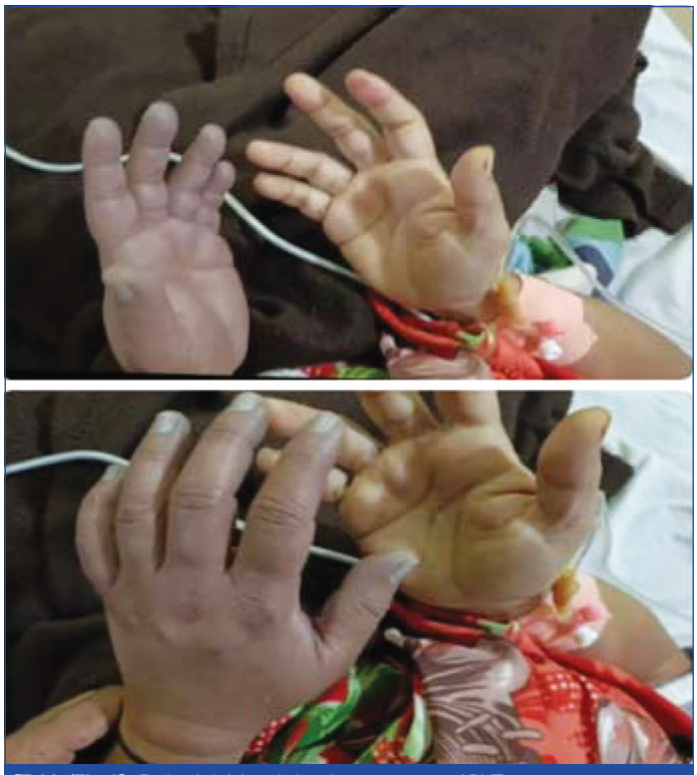
The surgery proceeded uneventfully, lasting four hours with an estimated blood loss of 350 mL. Following skin closure, a bilateral Transverse Abdominis Plane (TAP) block was administered under aseptic conditions: a 23-gauge spinal needle was inserted midway

PT	12	11 to 13.5 seconds
INR	1	0.8 to 1.1
CA125	20 U/mL	0 to 35 U/mL
D dimer	>4000 ng/mL	less than 500 ng/mL
APTT	36 sec	25 to 35 seconds
USG duplex left arm	Left internal jugular vein, subclavian vein, axillary vein show normal colour flow, compression and grey scale appearance. Left brachial vein shows thrombosis with greyscale imaging showing echogenic material completely obstructing the lumen of the vein.	

[Table/Fig-2]: Postoperative investigations of patient.

between the costal margin and iliac crest, and after confirming double-pop loss of resistance, 15 cc bupivacaine (0.5%) with 15 mcg dexmedetomidine was injected per side. Upon detecting adequate tidal volume and eye-opening, neuromuscular blockade was reversed with i.v. glycopyrrolate 0.6 mg and neostigmine 3 mg. Extubation was uncomplicated. In the Post-Anaesthesia Care Unit (PACU), 2 L/min oxygen was delivered via nasal prongs, i.v. 0.45% dextrose normal saline was infused at 80 mL/hr, and NPO status was maintained for six hours.

Six hours postoperatively, the patient reported pain, swelling, and bluish discolouration of the left hand [Table/Fig-3] unresponsive to i.v. paracetamol. There was no local trauma. Radial, brachial, and axillary pulses were palpable. An X-ray of the hand was normal. Immediate cardiothoracic surgery consultation was obtained, and a venous duplex ultrasound of the left arm revealed a brachial venous thrombus. No signs of PE were present. UFH was initiated at an infusion rate of 1000 IU/hr, monitored via 6-hourly activated Partial Thromboplastin Time (aPTT). After 24 hours, the infusion was transitioned to subcutaneous UFH 5000 IU every eight hours for three days. Symptoms resolved within 72 hours. On postoperative day 5, she was switched to oral apixaban 5 mg twice daily and discharged on day 10. Outpatient follow-ups occurred at 1, 3, and 6 months. Repeat ultrasound at three months confirmed full clot resolution. At six months apixaban was discontinued.



[Table/Fig-3]: Patient's left hand showing symptoms of DVT.

DISCUSSION

The UEDVT is the formation of a thrombus in the jugular, subclavian, brachiocephalic, axillary, radial, or ulnar veins, driven by Virchow's triad: venous stasis, vessel wall injury, and hypercoagulability. Once rare, UEDVT now accounts for 4% to 10% of all Venous Thromboembolic (VTE) events. Clinical presentation classically involves acute unilateral limb swelling, oedema, and heaviness. Primary causes include anatomical abnormalities like cervical ribs, shoulder girdle syndrome, Paget-Schroetter syndrome, or clavicle fractures and shoulder dislocations. Secondary causes include mechanical trauma from Central Venous Catheters (CVCs), Peripherally Inserted Central Catheters (PICCs), or pacemaker leads [1]. Postoperatively, UEDVT stems from surgical trauma, malignancy, and prolonged perioperative immobility that induce venous stasis and systemic inflammatory pathways [2].

In this patient, UEDVT presented within six hours postoperatively despite no prior risk factors or intraoperative complications. Genetic screening for Factor V Leiden, antiphospholipid antibodies, prothrombin G20210A mutation, and Protein C, Protein S, or antithrombin III deficiencies was omitted due to the absence of personal or family thrombotic history [3]. Notably, Mustafa S et al., observed that 19% of radiologically proven upper-limb DVT cases lacked identifiable risk factors [4].

Surgery under general anaesthesia lasting longer than 30 minutes represents a recognised transient VTE risk factor due to immobility and reduced blood flow [5]. This patient's 4-hour surgery contributed to this risk. Furthermore, Marschang P et al., reported an UEDVT caused by automated blood pressure cuff monitoring in a patient lacking other risk factors [6]. In this case, the NIBP cuff was applied to the affected left arm throughout the perioperative period, inflating every five minutes intraoperatively and every 30 minutes postoperatively. Additionally, uterine and adnexal masses elevate VTE risk [7]. While uterine leiomyomas primarily cause lower-limb distal DVTs via pelvic vein compression, associated Abnormal Uterine Bleeding (AUB) can trigger hypercoagulability through compensatory polycythemia and thrombocytosis [7]. However, preoperative testing ruled out polycythemia and thrombocytosis, and the patient was not taking prothrombotic oestrogen therapies.

Skoko M et al., reported UEDVT as an initial manifestation of occult malignancy, where inflammatory cytokines (TNF-α, IL-6) caused endothelial dysfunction without direct venous compression [8]. Postoperative VTE incidence is roughly doubled in patients with active malignancy compared to those with benign pathology [8]. Malignancy induces hypercoagulability via prothrombotic factors (e.g., tissue factor) that alter natural anticoagulant pathways [8]. Preventing UEDVT requires high clinical suspicion across all perioperative phases. Preoperatively, identifying active malignancies, anatomical variations, and family history is critical [1]. Stratification tools, such as the Caprini Risk Assessment Model, guide thromboprophylaxis planning [9].

Intraoperatively, anaesthesia selection and positioning modulate venous return. General anaesthesia induces vasodilation and abolishes muscle pump action, fostering stasis. While regional anaesthesia minimises these effects, general anaesthesia with mechanical ventilation was necessary here due to the extended operative time and combined pelvic and upper abdominal surgical fields (requiring deep muscle relaxation for hernia repair and visceral pain control) maintaining haemodynamics and minimising operative duration reduce stasis [10]. Detecting UEDVT intraoperatively is challenging, but limb swelling and plethysmographic changes on pulse oximetry offer early diagnostic clues. A meta-analysis by Locker T et al., noted that while plethysmography lacks sufficient sensitivity as a standalone tool, it serves as a valuable early indicator [11]. Postoperatively, mechanical prophylaxis (e.g., intermittent pneumatic compression or graduated compression stockings) reduces lower-limb stasis [10]. Case literature emphasises investigating underlying neoplasia when acute UEDVT occurs without obvious catheter-related triggers [8].

Anticoagulation is the core management strategy. The i.v. heparin is preferred acute postoperative therapy due to its rapid onset and

Study	Age and gender	Possible cause	Veins involved	Initial management	Long-term management
Chen AWY et al., [13]	38/M	Prolonged immobility of upper arms	Axillary vein	Subcutaneous low molecular weight heparin	Oral warfarin for 3 months
Chen S et al., [14]	79/M	Peripheral catheter (picc) associated UEDVT post craniotomy for subdural haemorrage	Axillary, brachial, radial ulnar	Nadroparin calcium at 4,100 IU subcutaneously once daily. the Nadroparin dosage was escalated to 4,100 IU twice daily, subsequently switched to enoxaparin at 6,000 IU twice	Oral Rivaroxaban (20 mg once daily). Till discharge
Skoko M et al., [8]	55/F	Left segmentectomy and axillary dissection for breast cancer	Brachial vein	Subcutaneous Dalteparin (LMWH), at a dose of 7,500 IU daily, adjusted based on body mass. Dalteparin dosage was reduced to 5,000 IU daily. Oral Warfarin was started concurrently on Day 6 to overlap with LMWH therapy.	On Day 10, Dalteparin was discontinued, and the patient was maintained solely on oral Warfarin.
Marschang P et al., [6]	60/F	Repeated blood pressure cuff monitoring for 24 hours	Subclavian axillary and brachial vein	Initial treatment with Enoxaparin 60 mg subcutaneously BD shifted to oral Acenocoumaral (Vitamin k antagonist) after dose titration with INR	12 months therapy with oral Acenocumaral Dose titrated to INR
Current case's finding	46/F	post total abdominal hysterectomy for uterine fibroids and umbilical hernia repair	Brachial vein	Infusion of Unfractionated Heparin (UFH) 1000 IU/hr for 24 hours Switched to 5000 IU subcutaneously thrice daily for. 3 days	On day 5 oral Apixaban 5 mg twice daily started and continued till 6 months

[Table/Fig-4]: table comparing various regimens used for treatment of UEDVT [6,8,13,14].

ease of reversal with protamine if surgical bleeding occurs [12]. In this case, immediate UFH infusion titrated via aPTT monitoring was justified. For cancer-associated thrombosis, Low-Molecular-Weight Heparin (LMWH) or UFH is standard [12] supporting initial heparin stabilisation. A single, unified treatment protocol for UEDVT remains unestablished; [Table/Fig-4] [6,8,13,14] outlines common literature-reported regimens. Untreated UEDVT carries significant morbidity. Pulmonary Embolism (PE) occurs in 2% to 6% of cases [1], presenting a critical mortality risk. Long-term sequelae include Post-Thrombotic Syndrome (PTS)- characterised by chronic pain, oedema, and hyperpigmentation from valvular insufficiency- which occurs in up to 5% of primary and higher rates of secondary UEDVT [15]. Prompt anticoagulation is vital to mitigate both PE and PTS risks [15].

CONCLUSION(S)

This case demonstrates an acute UEDVT developing within six hours of prolonged gynaecological surgery in the absence of CVC or baseline risk factors. Prompt diagnosis via venous duplex ultrasound and stepwise anticoagulation enabled complete thrombus resolution at six months without PE or haemorrhagic complications. Clinicians must maintain a high-index of suspicion for UEDVT when encountering acute postoperative limb swelling and critically evaluate intraoperative monitoring practices.

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PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Anaesthesia, Smt. B.K. Shah Medical Institute and Research Center, Dhiraj General Hospital, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
2. Professor, Department of Anaesthesia, Smt. B.K. Shah Medical Institute and Research Center, Dhiraj General Hospital, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
3. Professor, Department of Anaesthesia, Smt. B.K. Shah Medical Institute and Research Center, Dhiraj General Hospital, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
S Sreya Sri,
B5, 203, Springwood Residency, O. P. Road, Near Nand Duplex,
Behind Reliance Mall, Vadodara-390020, Gujarat, India.
E-mail: sreyasrisirpurkar1@gmail.com

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