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A Double-Edged Sword of Transfusion: A Post-Hysterectomy Plot Twist - A Case Report

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ABSTRACT

Red blood cell (RBC) transfusion effectively and rapidly corrects severe anaemia but is increasingly recognised to carry short- and long-term risks that extend beyond immunological and infective hazards, including immunomodulation, altered coagulability, and endothelial glycocalyx injury. We describe a 40-year-old woman with severe menorrhagia-related anaemia (haemoglobin 6.9 g/dl) who received five units of packed red blood cells before elective total abdominal hysterectomy. Ultrasound-guided internal jugular vein cannulation failed bilaterally, and a femoral venous catheter was placed; a further internal jugular attempt precipitated acute respiratory distress requiring conversion to general anaesthesia. Hysterectomy was otherwise uneventful. Postoperatively, the patient developed right lower-limb deep vein thrombosis at the femoral catheter site, followed by septic shock, splenic and cerebral infarction, and multi-organ dysfunction; blood and drain cultures grew Serratia marcescens and Escherichia coli respectively. Despite maximal supportive care, she died on postoperative day 5. This case illustrates the sequential, and ultimately fatal, convergence of transfusion-associated hypercoagulability and immunomodulation with catheter-related thrombosis and nosocomial sepsis in a single perioperative course. It highlights the need for judicious transfusion thresholds, preferential avoidance of femoral venous access, and heightened postoperative surveillance for thrombotic and septic complications in transfused surgical patients.

Keywords: *Red blood cell transfusion; venous thromboembolism; endothelial glycocalyx; hysterectomy; perioperative sepsis; case report*

INTRODUCTION

Anaemia is a common indication for red blood cell (RBC) transfusion in the perioperative setting, particularly in gynaecological patients presenting with chronic blood loss from menorrhagia. While transfusion effectively and rapidly corrects haemoglobin deficits and reduces the immediate risks associated with severe anaemia, it is not without hazard. Modern blood banking has largely eliminated the risks of ABO incompatibility and transfusion-transmitted infection through improved screening and cross-matching protocols. However, current international guidance continues to favour restrictive transfusion thresholds in haemodynamically stable patients, **[1]** reflecting a growing body of evidence that RBC transfusion is

independently associated with increased short- and long-term morbidity, including nosocomial infection, bleeding complications, and mortality, through mechanisms that are only partly understood. Proposed mechanisms include transfusion-related immunomodulation (TRIM), storage-lesion-related changes in donor erythrocytes, and disturbance of the recipient's haemostatic balance. **[2,3]** Of particular interest is the observation that RBC transfusion may be associated with a reduction in coagulability accompanied by degradation of the endothelial glycocalyx - a delicate layer lining the vascular endothelium that normally maintains an anti-thrombotic, anti-adhesive surface. **[4]** Paradoxically, despite this reduction in measured coagulability, clinical and epidemiological data suggest that perioperative RBC transfusion is associated with an increased risk of venous thromboembolism (VTE), **[5]** suggesting that endothelial injury and a systemic inflammatory response may outweigh any anticoagulant effect in vivo. We report a case in which multiple transfusion-associated complications — thrombosis, sepsis, and multi-organ dysfunction — occurred sequentially in a single patient following major gynaecological surgery, resulting in a fatal outcome, and discuss the interplay of transfusion, surgery, and anaesthesia in the pathogenesis of these complications.

CASE REPORT

A 40-year-old woman, gravida 4 para 1 living 1 (G4P1L1), presented with menorrhagia and abdominal pain of several months' duration and was found to have severe anaemia. She was scheduled for elective total abdominal hysterectomy. Initial haemoglobin was 6.9 g/dl, and serum ferritin was markedly elevated at 1000 ng/ml. She received five units of packed red blood cells preoperatively, after which her haemoglobin improved to 12 g/dl. Baseline biochemical investigations, electrocardiography, and chest radiography were unremarkable. Transthoracic echocardiography demonstrated trivial mitral and mild tricuspid regurgitation, with no other structural or functional abnormality.

In view of poor peripheral venous access, ultrasound-guided central venous catheterization was planned prior to induction of spinal anaesthesia. Bilateral attempts at internal jugular vein (IJV) cannulation were unsuccessful, and a central venous catheter was subsequently placed successfully in the right femoral vein. During a further attempt at left IJV cannulation, the patient developed acute respiratory distress. She was managed with rapid sequence induction and endotracheal intubation, and the anaesthetic plan was converted from neuraxial to general anaesthesia. An arterial blood gas sample drawn intraoperatively clotted before analysis and could not be reported. Total abdominal hysterectomy proceeded under general anaesthesia and was otherwise uneventful. The patient was extubated at the end of surgery and shifted to the recovery area in stable condition.

Figure 1: Clinical photograph showing the dressing over the right-sided central venous catheter insertion site.

Figure 2: Contrast-enhanced CT of the neck (coronal reformat) obtained during work-up of the catheterisation-related chest pain, showing the great vessels of the neck and thoracic inlet at the site of the internal jugular vein cannulation attempts.

On the night following surgery, the patient developed swelling and pain in the right lower limb. Doppler ultrasonography confirmed a right lower-limb deep vein thrombosis (DVT); the femoral venous catheter was removed and pharmacological thromboprophylaxis was initiated.

She subsequently developed chest pain, which was temporally related to the earlier left IJV catheterization attempt; a cardiac workup, D-dimer, and Protein C and S levels were within normal limits, making an acute coronary or primary thrombophilic cause less likely. The following day, she developed septic shock requiring mechanical ventilation and inotropic support. Computed tomography pulmonary angiography (CTPA) revealed splenic infarcts, and computed tomography of the brain showed a left temporal infarct. By postoperative day 4, liver function tests and the coagulation profile had worsened, raising suspicion of sepsis- or ischaemia-induced hepatitis. Blood cultures grew Serratia marcescens, and drain fluid cultures grew Escherichia coli. Despite maximal supportive management, the patient suffered a cardiac arrest and died on postoperative day 5.

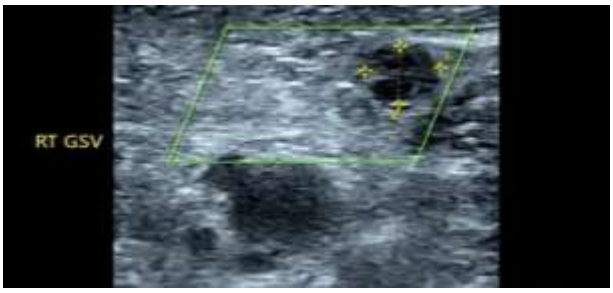

Figure 3: Doppler ultrasonography of the right lower limb showing non-compressible, echogenic thrombus within the right great saphenous vein (RTGSV), confirming deep vein thrombosis.

Figure 4: Contrast-enhanced CT of the abdomen showing a wedge-shaped area of non-enhancement in the spleen, consistent with splenic infarction.

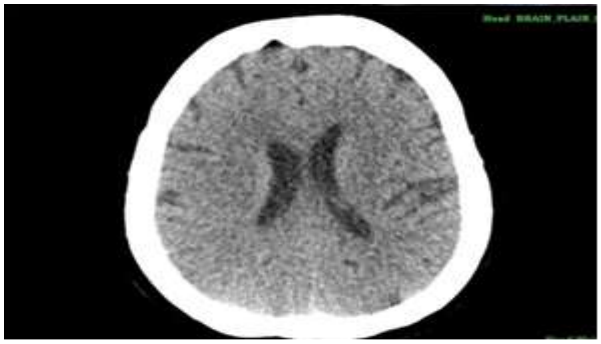

Figure 5: Plain CT of the brain showing a hypodense area in the left temporal region, consistent with an acute infarct.

DISCUSSION

This case illustrates a cascade of transfusion- and catheter-related complications culminating in multi-organ thrombotic and septic injury after an otherwise routine major gynaecological procedure. RBC transfusion is known to induce a state of immunosuppression through TRIM and, seemingly paradoxically, to be associated with perioperative hypercoagulability. **[2,3,6]** Transfused blood undergoes storage-related changes, and the interaction between transfused erythrocytes, the recipient's immune system, and the vascular endothelium may result in degradation of the endothelial glycocalyx - a key regulator of vascular permeability, leukocyte adhesion, and coagulation. **[4]** Loss of glycocalyx integrity exposes underlying pro-thrombotic subendothelial structures and disrupts the normal anti-coagulant surface of the endothelium, providing a plausible mechanistic link between transfusion and the increased venous thromboembolic risk reported in several cohort studies and meta-analyses. **[5]**

In the present case, several additional factors likely compounded this baseline transfusion-associated risk: major pelvic surgery itself is a recognised risk factor for VTE; failed IJV cannulation with eventual femoral venous catheterization introduced a further prothrombotic focus, given the higher thrombotic risk associated with femoral compared with subclavian or internal jugular catheter sites demonstrated in a large multicentre randomised trial **[7]**; and the patient's severe pre-existing anaemia and iron overload (reflected in a markedly elevated ferritin) may themselves have contributed to an inflammatory, procoagulant milieu. The subsequent development of septic shock with Serratia marcescens bacteraemia and Escherichia coli-positive drain cultures, together with splenic and cerebral infarcts, suggests a combination of septic embolic phenomena and a systemic inflammatory response superimposed on the initial thrombotic insult — a sequence consistent with TRIM increasing susceptibility to nosocomial infection. **[2,6]**

This case underscores several practical teaching points for the perioperative physician. First, the decision to transfuse should weigh the immediate benefit of haemoglobin correction against a less visible, delayed prothrombotic and immunosuppressive risk, particularly in patients undergoing major surgery; current guidance supports restrictive, haemoglobin-triggered transfusion in stable patients rather than correction to supraphysiological targets. **[1]** Second, femoral venous access, although sometimes unavoidable in the setting of difficult IJV cannulation, carries a higher thrombotic risk and should be exchanged for an alternative site as soon as clinically feasible. **[7]** Third, clinicians should maintain a low threshold for VTE surveillance and early pharmacological or mechanical thromboprophylaxis in transfused surgical patients, especially those with additional risk factors such as prolonged central venous catheterization or difficult vascular access. Finally, once thrombotic and septic complications appear

together, as in this patient, early multidisciplinary escalation - involving haematology, critical care, and infectious disease specialists - is essential, though outcomes may remain poor despite timely intervention.

This report is limited by its retrospective, single-case nature, which precludes establishing a definitive causal pathway between transfusion and the patient's subsequent complications; sepsis, catheter-related thrombosis, and pre-existing anaemia-related inflammation may each have contributed independently. Nonetheless, the temporal sequence - transfusion, followed by femoral catheterization, followed by DVT, followed by septic shock and multi-organ infarction - is consistent with mechanisms described in the transfusion medicine literature **[2-6]** and reinforces the need for prospective, VTE-focused cohort studies in transfused surgical populations.

CONCLUSION

RBC transfusion, while often life-saving, is associated with reduced coagulability and degradation of the endothelial glycocalyx. The physiological changes described are typically small to moderate in magnitude but may translate into meaningful clinical harm - including venous thromboembolism, immunosuppression, and infection - in vulnerable perioperative patients. This case reinforces the need for judicious, guideline-concordant transfusion practice, **[1]** careful selection of vascular access sites, heightened postoperative vigilance for thrombotic and septic complications, and further prospective research to define effective prophylactic strategies in this population.

Declaration By Authors

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