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Case Report

Warfarin-Induced Supratherapeutic Anticoagulation Presenting as an Iliopsoas/Iliacus Haematoma with Lower Gastrointestinal Haemorrhage

Subhasis Mishra, Raghavendra Shivaram Hegde*

Department of Pharmacy Practice, H.S.K. College of Pharmacy, Bagalkot, Karnataka, INDIA.

ABSTRACT

Warfarin remains one of the most frequent causes of serious bleeding-related hospital admissions because of its narrow therapeutic index and unpredictable inter-individual response, and the iliopsoas/iliacus compartment is an under-recognised site of anticoagulant-related haemorrhage that can be mistaken radiologically for an abscess. A 44-year-old man with an eleven-year history of warfarin therapy for deep vein thrombosis presented with a fifteen-day history of bleeding per rectum together with one day of abdominal pain and vomiting. He was severely anaemic (Haemoglobin 4.2 g/dL) with a critically elevated international normalised ratio of 9.23. Ultrasonography and computed tomography demonstrated a rapidly enlarging collection in the left iliacus muscle that was radiologically favoured as an abscess, but the absence of fever together with the coagulation profile pointed instead to an expanding haematoma. Critically, pharmacy dispensing records confirmed that the patient had been concurrently self-administering warfarin alongside a newly prescribed rivaroxaban regimen-a medication-reconciliation failure that was the proximate cause of the supratherapeutic anticoagulant state. Management included withholding warfarin, intravenous Vitamin K, multiple packed red cell transfusions, empirical antibiotic cover, and ultrasound-guided drainage, with gradual clinical improvement. Intravenous Vitamin K was selected as the reversal agent in preference to fresh frozen plasma or four-factor prothrombin complex concentrate, which was appropriate given the patient's haemodynamic stability and the absence of a requirement for emergency surgical intervention. Causality was assessed using the Naranjo Adverse Drug Reaction Probability Scale-with the INR applied as a validated pharmacodynamic surrogate for toxic warfarin exposure-yielding a score of 8 and classifying the event as a probable adverse drug reaction. Iliopsoas/iliacus haematoma is a potentially life-threatening complication of supratherapeutic warfarin therapy that can closely mimic an abscess on imaging. Clinicians and pharmacists must correlate any newly detected intra-abdominal collection with the coagulation profile before assuming an infective cause, and must ensure structured anticoagulant reconciliation at every point of transition between anticoagulant classes.

Keywords: Adverse Drug Reaction, Anticoagulant-Associated Haemorrhage, Iliopsoas Haematoma, Naranjo Scale, Supratherapeutic INR, Warfarin.

Correspondence:
Dr. Raghavendra S. Hegde
PharmD, Assistant Professor, Department of Pharmacy Practice, H.S.K. College of Pharmacy, Bagalkot, Karnataka, INDIA.
Email: raghavhegde28@gmail.com

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INTRODUCTION

Warfarin, a Vitamin K antagonist, remains widely prescribed for the prevention and treatment of venous thromboembolism, atrial fibrillation, and mechanical heart valve thrombosis. Its narrow therapeutic index, extensive drug-food and drug-drug interactions, and dependence on regular monitoring of the International Normalised Ratio (INR) make it one of the drugs most frequently implicated in serious Adverse Drug Reactions (ADRs), the majority of which are haemorrhagic in nature (Ansell *et al*., 2008; Brunton *et al*., 2018).

Gastrointestinal and intracranial bleeding are the most commonly reported sites of warfarin-related haemorrhage, but retroperitoneal and iliopsoas compartment haematomas are an under-recognised, clinically important complication. They often present with non-specific abdominal, groin, or hip pain rather than overt bleeding, a feature that can delay diagnosis and lead imaging findings to be misread as an abscess rather than a haematoma. We describe a case of a long-standing warfarin user who developed an expanding iliacus muscle haematoma together with lower gastrointestinal bleeding in the setting of a critically elevated INR, and discuss the causality assessment and

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the clinical and medication-safety learning points relevant to pharmacy practice and pharmacovigilance.

CASE REPORT

Patient Information

A 44-year-old male resident of Bagalkot, Karnataka, presented to the surgery casualty department of a tertiary care teaching hospital.

Past Medical and Medication History

The patient had a well-documented eleven-year history of Vitamin K antagonist therapy. He was admitted at an outside hospital from 4 to 11 July 2015, at 30 years of age, with left lower-limb deep vein thrombosis; venous duplex at that admission demonstrated thrombosis of the left common iliac, external iliac, common femoral, superficial femoral and popliteal veins with extension into the long saphenous vein. Anticoagulation was initiated with warfarin, bridged with subcutaneous enoxaparin during the inpatient stay, and continued thereafter on an outpatient basis.

Outpatient follow-up through 2015-2016 showed fluctuating INR control (recorded values ranging from 1.3 to 3.65 across visits) requiring repeated warfarin dose titration. During this period, he also developed iron-deficiency anaemia (haemoglobin nadir 7.9 g/dL, with a peripheral smear showing microcytic hypochromic cells and a serum ferritin of 3.75 ng/mL), which was treated with oral iron and folate supplementation. Thrombophilia-directed screening at the time showed a mildly elevated serum homocysteine of 21.7 micromol/L, a recognised contributory risk factor for venous thromboembolism in a patient with no other major provoking factor for his index DVT. Serial venous Doppler studies in 2016, 2018, and 2020 documented progressive chronic post-thrombotic change, with partial recanalisation of the previously thrombosed segments, saphenofemoral and saphenopopliteal junction incompetence, and secondary varicose veins with multiple incompetent perforators, consistent with established post-thrombotic syndrome (Figure 1).

He had hypertension, reportedly on irregular treatment, with no documented history of diabetes mellitus, epilepsy, tuberculosis, or bronchial asthma. Lower-limb venous Doppler studies in December 2024 and April 2026 confirmed persistence of chronic deep vein thrombosis of the left common femoral, superficial femoral, and popliteal veins with partial (30-60%) recanalisation, together with secondary varicose veins and incompetent perforators, consistent with the longer trajectory above. In April 2026, he was managed at an outside hospital with compression stockings, tablet aspirin 75 mg once daily life-long, tablet rivaroxaban 10 mg twice daily for 45 days, and topical betamethasone, for symptomatic chronic DVT and varicose disease.

History of Presenting Illness

The patient presented with a one-day history of diffuse, non-radiating abdominal pain and one episode of non-bilious, non-projectile vomiting, superimposed on a fifteen-day history of bleeding per rectum. There was no fever, burning micturition, or loose stools. He had a remote history of a ureteric calculus three years earlier with spontaneous resolution.

Physical Examination on Admission

Vitals

Blood pressure 130/80 mmHg, pulse rate 74-78/min, afebrile. Per abdomen: soft, with tenderness in the left iliac fossa and no guarding or rigidity. The cardiovascular and respiratory systems were unremarkable, and the patient was conscious and oriented. A provisional diagnosis of suspected left renal calculus was made, and the patient was admitted to the surgery male ward for further evaluation.

Investigations

Haematological Trend

The reference ranges used at the treating laboratory were Haemoglobin 13.0-18.0 g/dL, total WBC count 4,000-11,000/cumm, and platelet count 1,50,000-4,00,000/cumm. The trend over admission is summarised in Table 1.

Coagulation Profile

The critically prolonged prothrombin time and INR on admission, improving but still markedly elevated three days later despite Vitamin K administration, was the pivotal laboratory finding in this case (Table 2).

Imaging

Ultrasonography of the abdomen (13-06-2026) showed an unremarkable liver, spleen, pancreas and kidneys, no renal calculus or hydronephrosis, mild ascites, and a hypoechoic, thick-walled collection in the left iliacus muscle measuring 6.9 × 3.8 × 3.9 cm (approximately 53 cc), favoured as likely an abscess; Contrast-Enhanced Computed Tomography (CECT) was advised for further evaluation. CECT of the abdomen and pelvis performed the following day (14-06-2026) showed a bulky, heterogeneously enhancing collection in the left iliacus muscle measuring approximately 11.0 × 7.1 × 6.0 cm (estimated volume approximately 251 cc), with adjacent peritoneal free fluid and fat stranding, displacing the oedematous left psoas muscle medially, and was again reported as 'likely intramuscular abscess.' Additional findings included mild free fluid in the bilateral paracolic gutters, bilateral perinephric fluid and fat stranding, sub-mucosal oedema of the rectum, sigmoid, descending and ascending colon and ileal loops (suggested reactive enterocolitis), bilateral simple renal cortical cysts, a non-obstructing right renal calculus (approximately 8.7 × 6.2 mm), mild prostatomegaly

(approximately 26 cc), bilateral varicoceles, hepatic calcified granulomas, a T10 vertebral haemangioma, and mild left pleural effusion with adjacent basal collapse-consolidation. The collection in the left iliacus muscle nearly quintupled in measured volume between the ultrasound and the CT scan performed approximately twenty-four hours later, a trajectory more consistent with an actively expanding haematoma than a static infective abscess, particularly in a patient who remained afebrile throughout admission and who had a critically elevated INR at presentation.

Other Investigations

Liver function tests, renal function tests, and serum electrolytes were within normal limits except for a mildly reduced serum albumin (3.2 g/dL); random blood sugar was 198 mg/dL. Urine routine examination showed 3+ blood and 1+ protein with 2-5 pus cells per high-power field, consistent with the patient's bleeding tendency rather than an isolated urinary pathology. Serology for HIV, hepatitis B surface antigen, and hepatitis C virus was non-reactive. A twelve-lead electrocardiogram showed sinus tachycardia (rate approximately 106-108/min) with a mildly prolonged corrected QT interval, a pattern consistent with a physiological response to acute, severe blood-loss anaemia rather than a primary cardiac event. The patient's blood group was typed as A positive, and multiple units of packed red cells were transfused during admission without documented transfusion reactions.

Treatment and Hospital Course

The patient was kept nil-by-mouth initially, progressing to a liquid and then a regular diet as tolerated. He was managed with intravenous fluids (dextrose-normal saline, Ringer's lactate, and normal saline), empirical intravenous antibiotics (initially a ceftriaxone-sulbactam combination with metronidazole, later broadened to piperacillin-tazobactam as part of ongoing clinical reassessment), a proton pump inhibitor (pantoprazole), and an antiemetic (ondansetron), pending clarification of the nature of the iliacus collection.

In view of the markedly elevated INR and active clinical bleeding, the offending anticoagulant was withheld and intravenous Vitamin K 10 mg was administered. Severe symptomatic anaemia was corrected with multiple packed red cell transfusions over several days, each preceded by antihistamine and corticosteroid premedication with close vital-sign monitoring; no transfusion reactions were recorded. Repeat coagulation studies on day three showed a falling but still elevated INR (5.22), reflecting the expected slow correction of Vitamin K antagonist effect. Stool occult blood testing, repeat haemogram and peripheral smear, and renal function tests were repeated serially to track ongoing blood loss and recovery. An ultrasound-guided pigtail catheter was placed for image-guided drainage of the iliacus/retroperitoneal collection, with antibiotic cover continued empirically given the radiological ambiguity between haematoma and abscess.

Analgesia during the early part of admission was provided with parenteral diclofenac. In a patient who was simultaneously coagulopathic and actively bleeding, a non-steroidal anti-inflammatory agent carries an avoidable additional bleeding risk and is not the preferred analgesic choice in this setting; this point is revisited as a medication-safety learning point in the Discussion.

Outcome and Follow-up

Following cessation of the offending anticoagulant and Vitamin K reversal, the patient's INR showed a sustained downward trend and active bleeding settled, with haemoglobin stabilising on transfusion support and the patient remaining afebrile and haemodynamically stable through the remainder of the admission. He was discharged on his pre-admission antiplatelet and anticoagulant regimen of aspirin and rivaroxaban, with the offending Vitamin K antagonist permanently discontinued, along with the other discharge medications he had been maintained on. Physiotherapy was advised for the affected limb and hip region to support functional recovery from the iliacus haematoma, and the patient was scheduled for outpatient review to monitor resolution of the collection and continued surveillance of his underlying chronic venous disease.

DISCUSSION

This case is best characterised as a probable adverse drug reaction: warfarin-induced supratherapeutic anticoagulation manifesting as a major, atypical bleed into the iliacus/psoas compartment with concurrent lower gastrointestinal haemorrhage, occurring against a long-standing history of Vitamin K antagonist use for DVT.

Several features support this causal attribution. First, the temporal relationship is plausible, since the patient was a long-term warfarin user and a critically elevated INR of 9.23 was documented at the time of acute presentation with bleeding. Second, dechallenge, that is, withholding the anticoagulant together with Vitamin K administration, was followed by a falling INR and haemodynamic improvement, consistent with reversal of the pharmacological effect. Third, no convincing alternative cause for the bleeding diathesis, such as primary haematological disease, malignancy, or trauma, was identified in the available records. Fourth, the rapid expansion of the iliacus collection on serial imaging is itself objective evidence of an active bleed.

A key medication reconciliation issue central to this case requires explicit resolution. Although the patient had been prescribed rivaroxaban 10 mg twice daily together with aspirin 75 mg daily at an outside hospital in April 2026, pharmacy dispensing records confirm that he continued to purchase and self-administer tablet

warfarin 3 mg from a community pharmacy during the same period, up to and including the time of his acute admission in June 2026. Direct oral anticoagulants such as rivaroxaban do not elevate the INR to a clinically significant degree; an INR of 9.23 is pharmacologically attributable exclusively to supratherapeutic Vitamin K antagonist activity (Holbrook *et al*., 2012). The documented concurrent dispensing of warfarin therefore establishes that the patient was inadvertently taking both agents simultaneously, with warfarin-not rivaroxaban-being the causative drug responsible for the haemorrhagic ADR. This represents a preventable medication-reconciliation failure at the point of anticoagulant transition: the prescribing clinician initiated a direct oral anticoagulant without confirming or documenting cessation of the long-standing Vitamin K antagonist, and no clinical pharmacist review or patient counselling appears to have been performed to identify the duplication. The critical role of structured anticoagulant reconciliation-including explicit discontinuation of the prior agent, patient education on the incompatibility of concurrent anticoagulants, and early INR follow-up during any transition-is a central learning point of this case.

The concurrent use of aspirin alongside an anticoagulant for a purely venous thromboembolic indication is also notable, since combined antiplatelet-anticoagulant therapy substantially increases bleeding risk without an established additional antithrombotic benefit in isolated venous disease, and should be revisited during medication review (Ansell *et al*., 2008).

A related, smaller prescribing-safety point from this admission is the early use of parenteral diclofenac for analgesia in a patient who was both actively bleeding and severely coagulopathic. Non-steroidal anti-inflammatory drugs inhibit platelet function and can aggravate gastrointestinal mucosal bleeding, and are best avoided in this clinical setting in favour of paracetamol; this represents a further, modifiable medication-safety learning point alongside the central anticoagulant-reconciliation issue (Brunton *et al*., 2018).

Iliopsoas/iliacus haematoma is an uncommon but recognised and potentially life-threatening complication of anticoagulant therapy. It often presents non-specifically with abdominal, flank, hip, or groin pain, sometimes mimicking renal colic or appendicitis, as occurred here with an initial provisional diagnosis of renal calculus, and can be complicated by femoral nerve compression or haemorrhagic shock if unrecognised. A high index of suspicion is warranted in any anticoagulated patient presenting with iliac fossa pain and otherwise unexplained anaemia, and imaging findings of a heterogeneous collection should be interpreted alongside the coagulation profile rather than in isolation, since haematomas can closely mimic abscesses radiologically (Jameson *et al*., 2018).

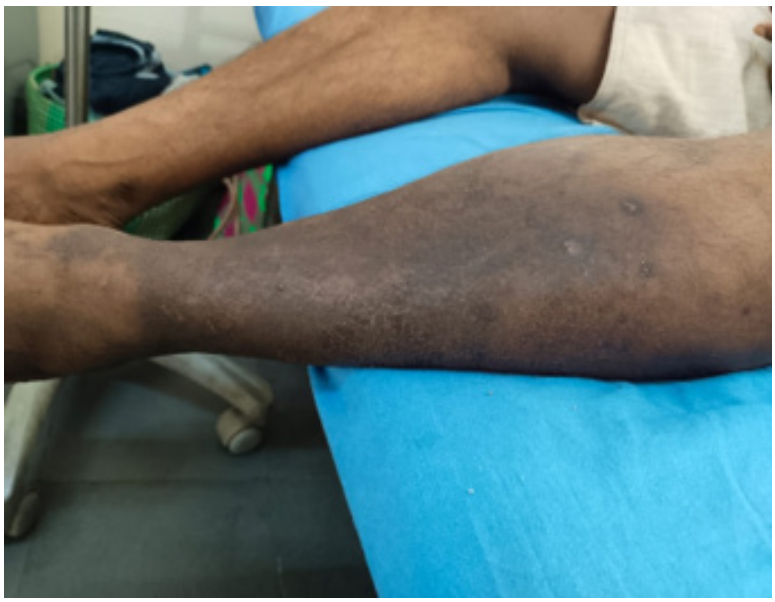

Figure 1: Clinical photograph of the left lower limb showing hyperpigmentation and skin changes of chronic venous insufficiency / post-thrombotic syndrome, consistent with the long-standing left lower-limb DVT described in the Case Report.

Table 1: Haematological Trend During Admission.

Date/Time	Haemoglobin (g/dL)	Total WBC Count (/cumm)	Platelet Count (/cumm)
13-06-2026, 08:44	4.6	21,200	3,79,000
13-06-2026, 11:24	4.2	15,200	3,13,000
16-06-2026, 12:36	6.2	9,100	3,52,000

Table 2: Coagulation Profile During Admission.

Date	PT Test (sec)	PT Control (sec)	APTT (sec)	INR
13-06-2026	120.0	13.0	Not recorded	9.23
16-06-2026	67.9	13.0	65.5 (control 30)	5.22

Table 3: Naranjo Adverse Drug Reaction Probability Scale.

Question	Yes	No	Do Not Know
1. Are there previous conclusive reports on this reaction?	+1		
2. Did the adverse event appear after the suspected drug was administered?	+2		
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist administered?	+1		
4. Did the adverse reaction reappear when the drug was readministered?			0
5. Are there alternative causes that could on their own have caused the reaction?		+2	
6. Did the reaction reappear when a placebo was given?			0
7. Was the drug detected in the blood or other body fluids in a concentration known to be toxic?	+1*		
8. Was the reaction more severe when the dose was increased, or less severe when decreased?			0
9. Did the patient have a similar reaction to the same or a similar drug in any previous exposure?			0
10. Was the adverse event confirmed by any objective evidence?	+1		

*Footnote: The INR of 9.23 was used as a validated pharmacodynamic surrogate for supratherapeutic warfarin concentration, consistent with established international practice for Vitamin K antagonist monitoring and ADR causality assessment (Holbrook *et al*., 2012). Total score: 8, classified as a 'Probable' adverse drug reaction (Naranjo score 5-8).

Causality Assessment: Naranjo Adverse Drug Reaction Probability Scale

Causality was assessed using the Naranjo Adverse Drug Reaction Probability Scale (Naranjo *et al*., 1981), summarised in Table 3. The Naranjo algorithm was selected because it is the most widely validated and reproducible structured instrument for ADR causality assessment in clinical pharmacy practice, and its step-wise scoring approach is particularly appropriate when the pharmacological mechanism of the adverse event is well established (Naranjo *et al*., 1981).

Naranjo question 7 asks whether the drug was detected in the blood or other body fluids in a concentration known to be toxic. No direct serum warfarin assay was performed, as this test is not routinely available in the clinical setting. A serum warfarin level is, however, not required to establish toxic drug exposure: the INR is a validated, internationally standardised pharmacodynamic biomarker of Vitamin K antagonist activity, reflecting the degree of anticoagulant effect directly and proportionately (Holbrook *et al*., 2012). An INR of 9.23-more than four times the upper limit of the standard therapeutic range of 2.0-3.0 for DVT-constitutes objective evidence of a supratherapeutic pharmacodynamic effect that is pharmacologically equivalent to toxic drug exposure. The use of a validated pharmacodynamic endpoint as a surrogate for direct drug measurement is established practice in ADR causality assessment for drugs whose therapeutic and toxic effects are monitored by a functional assay rather than by plasma concentration, and this approach has been widely accepted in published Naranjo-scored case reports involving Vitamin K antagonists (Ansell *et al*., 2008; Holbrook *et al*., 2012). Accordingly, a score of +1 was assigned for question 7.

Reversal Strategy: Vitamin K Versus Fresh Frozen Plasma and Prothrombin Complex Concentrate

The treating team elected intravenous Vitamin K 10 mg as the sole pharmacological reversal agent, without use of Fresh Frozen Plasma (FFP) or Four-Factor Prothrombin Complex Concentrate (4F-PCC). This decision warrants explicit justification. Current international guidelines stratify warfarin reversal strategy by haemorrhage severity and the urgency of INR correction (Holbrook *et al*., 2012). FFP and 4F-PCC are recommended primarily for life-threatening bleeding requiring immediate INR normalisation, for emergency surgical intervention, or for intracranial haemorrhage, where the speed of reversal is critical to survival. In this case the patient, although severely anaemic (Haemoglobin 4.2 g/dL) and with active bleeding per rectum, remained haemodynamically compensable throughout: he was alert and oriented, afebrile, and without features of hypovolaemic shock. The iliacus collection was managed by image-guided pigtail catheter drainage rather than emergency open surgery, and no urgent operative intervention was required. Under these circumstances, intravenous Vitamin K with supportive packed red

cell transfusion represents a guideline-concordant, appropriate reversal strategy; 4F-PCC would have been reserved for a situation requiring immediate, complete reversal within hours (Holbrook *et al*., 2012). FFP was not administered; while FFP can transiently correct the INR, it carries risks of volume overload and transfusion-related complications that are avoided by targeted Vitamin K plus PRBC transfusion in a haemodynamically stable patient. The INR trajectory-falling from 9.23 on day one to 5.22 by day three-confirmed an adequate pharmacological response to Vitamin K, validating the clinical decision. Had haemorrhage escalated or emergency surgical intervention been required, transition to 4F-PCC would have been the appropriate next step.

CONCLUSION

This case illustrates that warfarin continues to be a major and sometimes atypical cause of serious bleeding ADRs even after years of apparently stable therapy, and that the iliopsoas/iliacus compartment is an important but easily overlooked site of anticoagulant-related haemorrhage capable of closely mimicking an abscess on imaging. Critically, this case demonstrates that the concurrent inadvertent continuation of warfarin during a prescribed transition to a direct oral anticoagulant-a preventable medication-reconciliation failure-was the proximate trigger for a critically supratherapeutic INR of 9.23 and the resulting dual haemorrhage. It highlights the absolute necessity of explicit anticoagulant discontinuation confirmation, structured patient education, and clinical pharmacist review at every point of anticoagulant transition. Causality, established by a Naranjo score of 8, was justified using the INR as a validated pharmacodynamic surrogate for toxic warfarin exposure, consistent with established international practice. Reversal with intravenous Vitamin K and supportive transfusion was appropriate and guideline-concordant given the patient's haemodynamic stability; FFP and 4F-PCC remain reserved for life-threatening or surgically urgent scenarios. Prompt recognition, structured reversal, and image-guided drainage achieved clinical stabilisation, underscoring the importance of interdisciplinary care in managing severe anticoagulant-related haemorrhage.

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ABBREVIATIONS

4F-PCC: Four-Factor Prothrombin Complex Concentrate; **ADR:** Adverse Drug Reaction; **APTT:** Activated Partial Thromboplastin Time; **CECT:** Contrast-Enhanced Computed Tomography; **DVT:** Deep Vein Thrombosis; **FFP:** Fresh Frozen Plasma; **GI:** Gastrointestinal; **HIV:** Human Immunodeficiency Virus; **INR:** International Normalised Ratio; **IV:** Intravenous; **NSAID:** Non-Steroidal Anti-Inflammatory Drug; **PRBC:** Packed Red Blood Cells; **PT:** Prothrombin Time; **QTc:** Corrected QT Interval; **USG:** Ultrasonography; **VKA:** Vitamin K Antagonist; **WBC:** White Blood Cell.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images. A copy of the written consent is available for review by the Editor. No institutional ethics committee approval was sought, consistent with the convention for single, fully de-identified case reports; the report otherwise adheres to the principles of the Declaration of Helsinki.

AUTHORS' CONTRIBUTION

Subhasis Mishra was involved in clinical data collection, literature review, and drafting of the manuscript. Raghavendra S. Hegde conceptualised the case report, performed the Naranjo causality assessment, critically reviewed the manuscript for important intellectual content, and supervised the work as corresponding author. Both authors read and approved the final manuscript.

REFERENCES

Ansell, J., Hirsh, J., Hylek, E., Jacobson, A., Crowther, M., & Palareti, G. (2008). Pharmacology and management of the Vitamin K antagonists: American College of Chest Physicians evidence-based clinical practice guidelines (8th edition). Chest, 133(6 Suppl), 160S-198S. https://doi.org/10.1378/chest.08-0670

Brunton, L. L., Hilal-Dandan, R., & Knollmann, B. C. (Eds.).(2018). Goodman & Gilman's the pharmacological basis of therapeutics (13th ed.). McGraw-Hill Education.

Holbrook, A., Schulman, S., Witt, D. M., Vandvik, P. O., Fish, J., Kovacs, M. J., Svensson, P. J., Veenstra, D. L., Crowther, M., & Guyatt, G. H. (2012). Evidence-based management of anticoagulant therapy: Antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest, 141(2 Suppl), e152S-e184S. https://doi.org/10.1378/chest.11-2295

Jameson, J. L., Fauci, A. S., Kasper, D. L., Hauser, S. L., Longo, D. L., & Loscalzo, J. (Eds.).(2018). Harrison's principles of internal medicine (20th ed.). McGraw-Hill Education.

Naranjo, C. A., Busto, U., Sellers, E. M., Sandor, P., Ruiz, I., Roberts, E. A., Janecek, E., Domecq, C., & Greenblatt, D. J. (1981). A method for estimating the probability of adverse drug reactions. Clinical Pharmacology & Therapeutics, 30(2), 239-245. https://doi.org/10.1038/clpt.1981.154

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