Table 3 – The most frequently reported agents with their generic names

Drugs (in order of frequency)	Leukemia Subtype						
	AML	ALL	CML	CEL	CMML	CLL	HCL
Most frequent	Cyclophosphamide	Lenalidomide	Imatinib Mesylate	Imatinib Mesylate	Cyclophosphamide	Ibrutinib	Cladribine
2nd	Azacitidine	Cyclophosphamide	Nilotinib	Yellow fever vaccine	Azacitidine	Venetoclax	Adalimumab
3rd	Etoposide	Dexamethasone	Dasatinib	Hepatit A vaccine	Prednisone	Rituximab	Rituximab
4rd	Venetoclax	Methotrexate	Ponatinib	Lenalidomide	Lenalidomide	Cyclophosphamide	Cyclosporine
5rd	Lenalidomide	Blinatumomab	Cyclophosphamide	Bleomycin	Mycophenolate Mofetil	Lenalidomide	Infliximab

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Adult Hematology Abstract Categories

Coagulation Diseases
OP 02

MANAGEMENT OF BLEEDING IN A PATIENT WITH ACQUIRED HAEMOPHILIA A: A CASE REPORT

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Objective: Acquired haemophilia A (AHA) is a blood clotting disorder caused by the presence of autoantibodies that interfere with the function of factor VIII, often called factor VIII inhibitor (1). It has a prevalence of about one per million per year. Acquired haemophilia A is usually associated with autoimmune diseases, malignancies, skin disorders, drug interactions, and the postpartum period, and as many as 50% of cases have unknown causes (2). Bleeding often occurs under the skin (purpura/ecchymosis) and in soft tissues, and rarely in the joints (haemarthroses) (3). The presence of Factor VIII inhibitors causes a disruption in the intrinsic coagulation pathway, leading to prolongation of the activated partial thromboplastin time (APTT) parameter, which does not improve after mixing test, and has low Factor VIII activity (2). The management of patients with AHA aims to control bleeding and its complications and to eradicate factor VIII inhibitors. Immunosuppressive agents are usually used to eradicate factor inhibitors. In this study, we report a case of acquired haemophilia A presenting with spontaneous unprovoked bruising and discuss the approach to diagnosis and how to alert the clinician to suspect this potentially rare but devastating disease. **Case report:** A 67-year-old man presented to the emergency department with bruising on his left hand and right leg for approximately 10 days. The patient had no history of trauma. He had a history of hypertension and chronic obstructive pulmonary disease. Approximately 2 months prior to presentation, he had complained of bruising on his arms after taking medication (etodolac, ampicillin-sulbactam) (Figure 1). There had been no bleeding after the cholecystectomy. Physical examination revealed an anteroposterior diffuse haematoma extending from the right inguinal to the popliteal area and a haematoma on the dorsal aspect of the left hand (Figure 2). Other systemic examinations were normal. All laboratory results showed the initial complete blood count; normochromic anaemia, normal platelet count and prolonged activated partial thromboplastin time (APTT) with normal prothrombin time (PT). Liver and renal function tests were within normal limits. The test results showed very low factor VIII activity with normal von Willlebrand factor and fibrinogen levels (factor VIII activity: <0.4%). There was no improvement in the mixing test. The factor VIII inhibitor level was 96 Bethesda Units (BU). Workup for connective tissue disease and malignancy screening were otherwise negative. The patient's USG showed a $21 \times 20 \times 65$ mm haematoma in the muscle planes of the right thigh, which was drained by interventional radiology. The patient was then admitted to the haematology clinic and started on methylprednisolone at 1 mg/kg/day. However, recombinant factor VIIa followed by activated prothrombin complex concentrate was administered to the patient whose bleeding could not be controlled. In addition, the patient whose bleeding could not be controlled received three sessions of plasmapheresis every other day. After three weeks of steroid treatment, cyclophosphamide was added due to lack of response and treatment was continued for approximately 5 weeks. Factor 8 activity returned to normal after approximately two months and no inhibitor was detected after this period. **Conclusion:** Acquired haemophilia A (AHA) is a rare condition in which the body produces autoantibodies against factor VIII (1). Approximately half of cases are associated with a disease, and the others are idiopathic. The most commonly reported associations are autoimmune diseases, solid organ and lymphoproliferative malignancies, skin disorders, medications and pregnancy (2).Patients usually present with subcutaneous and mucocutaneous bleeding, followed by muscular, gastrointestinal, genitourinary and retroperitoneal bleeding (4). Our patient above presented with both subcutaneous and intramuscular haematomas.The first suspicion of this condition is usually based on a coagulation profile. This may show an isolated prolonged APTT ratio in a patient with unexplained bleeding. The platelet count will usually be normal. An isolated prolonged apTT raises two possibilities; either an absolute deficiency of intrinsic pathway coagulation factors, or the presence of antibodies or inhibitors to these factors (5). The BU test is performed to quantify the inhibitor titer and confirm the diagnosis. The stronger the inhibitor, the higher the BU (6). In general, a titer greater than 5 BU indicates a high titer inhibitor. Our patient had a reported inhibitor titer of 96 BU.Bypassing agents such as active recombinant factor VII have shown good results in stopping bleeding, while immunosuppressive drugs such as corticosteroids,

cytotoxic agents (cyclophosphamide, cyclosporine) and rituximab (anti-CD-20 monoclonal antibody) are often used to eradicate inhibitors (7).Our patient, who presented with diffuse bleeding in the right leg and left hand, was also difficult to treat due to high factor inhibitor level and was hospitalised for approximately one month.In conclusion, AHA should be recognised and treated rapidly, as delays in diagnosis can have serious consequences. In our patient, AHA was diagnosed rapidly and treatment and bleeding control were effective.

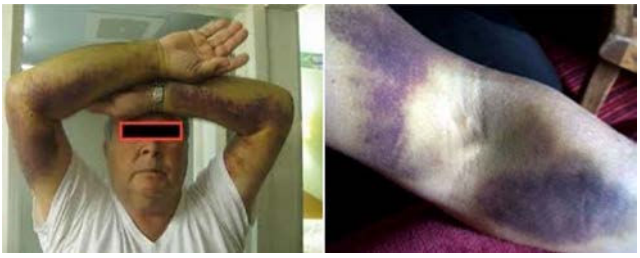

Figure 1: Ecchymosis on the forearms of both arms

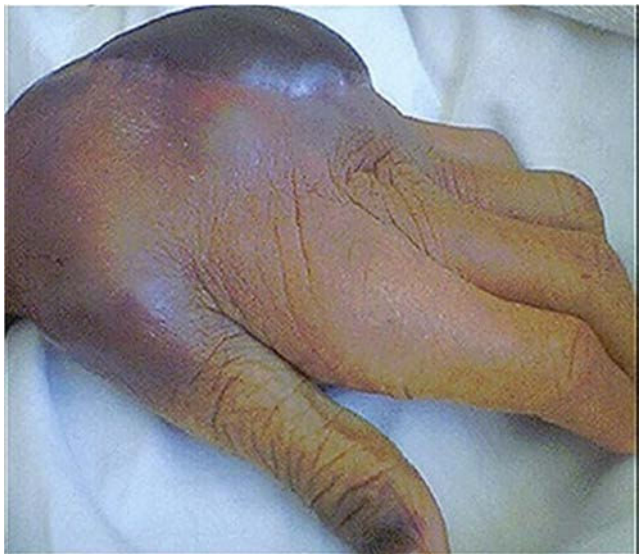

Figure 2:Ecchymosis on the dorsal left hand

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OP 03

NEWLY DIAGNOSED HEREDITARY FACTOR V DEFICIENCY IN A PATIENT PRESENTING WITH DEEP VEIN THROMBOSIS: A Rare Case

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Objective: Factor V (FV) is a crucial regulator of hemostasis, functioning as both a procoagulant and an anticoagulant glycoprotein within the coagulation cascade. In plasma, FV exists as an inactive precursor, which is activated by thrombin or factor Xa into its procoagulant form, FVa, contributing to the formation of the prothrombinase complex. Additionally, FV isoforms generated via alternative splicing, notably FV-short, exhibit anticoagulant properties by interacting with tissue factor pathway inhibitor-alpha (TFPIα), thus playing a role in regulating blood coagulation. Factor V deficiency is a rare condition characterized by a bleeding tendency, with a prevalence of approximately 1:1,000,000 in the general population. Clinical presentations can range from mild mucosal bleeding to severe hemorrhagic events. However, in rare cases, thrombotic complications may also occur, highlighting the heterogeneous clinical spectrum of FV deficiency. In this case report, we will discuss a patient who presented with deep vein thrombosis (DVT) and was diagnosed with FV deficiency. **Case report:** A 43-year-old female presented to the emergency department of Dicle University Hospital on July 21, 2024, with complaints of pain and swelling in her left leg. Physical examination and lower extremity venous Doppler ultrasonography revealed an echogenic thrombus within the distal iliac, femoral, popliteal, and proximal deep crural veins, leading to a diagnosis of deep vein thrombosis (DVT). Initial coagulation tests showed prolonged prothrombin time (PT) of 246 seconds, INR of 2.21, and activated partial thromboplastin time (APTT) of 38.2 seconds, with PT being more significantly prolonged. The complete blood count was normal. The patient was admitted to the cardiovascular surgery clinic for thrombolytic therapy and underwent thrombectomy and thrombolysis. Anticoagulant therapy with bemiparin sodium (HIBOR) was initiated. Due to abnormal coagulation tests, hematology consultation was requested. Further investigations showed normal lupus anticoagulant levels (1.16). Factor assays revealed a Factor V level of <6% (normal: 70-120%), while other factor levels were within normal limits. Anticardiolipin IgM and IgG were negative, and homocysteine was normal. APC resistance was measured at 0.64 seconds (normal: 0.91-1.19). Both Factor V Leiden and Factor II mutations were homozygous normal. Antithrombin III activity, as well as Protein C and S levels, were normal. ANA and anti-dsDNA tests were negative. A repeat Factor V level confirmed it remained <6%. The patient had no history of bleeding episodes and had not experienced bleeding complications during previous childbirths or minor surgeries. This case highlights the rare identification of Factor V deficiency in a patient presenting with deep vein thrombosis, an uncommon association, illustrating the complexity and variable clinical manifestations of Factor V deficiency. **Dıscussıon:** Factor V deficiency is a rare coagulopathy, typically characterized by a bleeding tendency, with an incidence of approximately 1:1,000,000 in the general population. The disease is inherited in an autosomal recessive manner and is caused by various mutations in the FV gene. As FV plays a critical role in both procoagulant and anticoagulant pathways, its deficiency usually manifests with symptoms such as mucosal bleeding, postoperative hemorrhage, and menorrhagia. However, paradoxically, some patients with FV deficiency have been reported to develop thrombotic events. In this patient, who presented with complaints of deep vein thrombosis, the absence of a bleeding history and lack of hemorrhagic complications during past surgical procedures highlights the