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Adrenal Crisis Secondary to Bilateral Adrenal Hemorrhage in a Patient with Hypercoagulable Disorder

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Abstract

Background: Bilateral adrenal hemorrhage (BAH) is a rare condition that may lead to life-threatening adrenal insufficiency or adrenal crisis if not addressed appropriately. **Case Report:** A 54-year-old male with a history of venous thromboembolism (VTE) on warfarin presented to the hospital with nausea, vomiting, and abdominal and flank pain shortly following colonoscopy preparation. Initial imaging of the abdomen and pelvis was notable for hyperdense thickening of the bilateral adrenal glands raising concerns for hemorrhages, and subsequent magnetic resonance imaging (MRI) confirmed BAH. During hospitalization, the patient was placed on prophylactic heparin, and shortly after decompensating, he became tachycardic, hypotensive, and febrile. This led to heparin reversal followed by administration of a single dose of Hydrocortisone 100 mg and Hydrocortisone 50 mg TID due to concern for adrenal insufficiency. The patient also necessitated sepsis work-up and fluid resuscitation. Repeat CT imaging showed no significant change in hemorrhage size bilaterally. Endocrinology, vascular surgery, hematology/oncology, and rheumatology were consulted for the management of adrenal insufficiency, anticoagulation in the presence of hemorrhage, thrombocytopenia, and hypercoagulable state. Towards the end of his hospital course, the patient had asymptomatic diffuse ST elevations, elevated troponin, and an ejection fraction of 10% - 15%, leading to cardiac catheterization and placement of an intra-aortic pump. During subsequent stay in the ICU, the patient developed hemodynamic shock and was transferred to a facility with a higher level of care and medical support therapies. After this transfer, the patient was stabilized from a cardiac standpoint but developed acute respiratory failure suspected to be secondary to diffuse alveolar hemorrhage and immune thrombocytopenic purpura, necessitating platelet transfusion. He was on continued monitoring from rheumatology given his myocarditis believed to be secondary to his

antiphospholipid antibodies, and was treated with IVIG, rituximab, and hydroxychloroquine. A repeat echocardiogram revealed an improved ejection fraction of 52% and the patient was then discharged on an enoxaparin bridge to warfarin and a cardiac home event monitor. **Discussion:** BAH is a life-threatening condition that should be promptly identified and managed in patients presenting with nonspecific symptoms and a history of hypercoagulability or anticoagulation. In these cases, the risk of AH and subsequent adrenal insufficiency is drastically increased, so immediate imaging as well as initiation of steroid therapy is crucial to stabilize patients and prevent adrenal crisis. A multidisciplinary approach, involving endocrinology, hematology, and cardiology as in this case is also imperative to optimize patient outcomes and increase survival. **Conclusion:** BAH should be considered in patients presenting with a history of VTE and hypercoagulable state when precipitating stressors or predisposing risk factors are present. This case report highlights the importance of clinical awareness of BAH for clinicians to accurately identify and manage it to prevent fatal sequelae and ensure long-term favorable patient outcomes.

Keywords

Bilateral Adrenal Hemorrhage, Adrenal Insufficiency, Antiphospholipid Syndrome, Hypercoagulable State

1. Introduction

Bilateral adrenal hemorrhage (BAH) is a rare condition, with a prevalence of 0.14% - 1.8%, according to post-mortem studies, in which blood collects between the adrenal gland and its capsule due to one or more ruptured adrenal vessels [1]. Potential etiologies include abdominal trauma, sepsis, infection, bleeding disorders, systemic lupus erythematosus (SLE), antiphospholipid syndrome, as well as preexisting adrenal conditions including adrenal tumors [1]. Accounting for about 10% of AHs are bilateral adrenal hemorrhages (BAH), which may have deleterious effects if they progress including adrenal insufficiency, which occurs in roughly 16% - 50% of individuals with BAH [1] [2]. Adrenal insufficiency can occur in patients with BAH if more than 90% of the adrenal cortex is involved [1] [2]. Clinical presentation of BAH can make early diagnosis difficult due to variable presentation and nonspecific symptoms (e.g., abdominal pain, nausea, vomiting, fatigue, hypotension) [2].

Upon clinical suspicion, imaging modalities such as computed tomography (CT) and MRI may be used to identify lesions [1] [3]. In the context of BAH, swift diagnosis and treatment are necessary to prevent the patient from declining to adrenal insufficiency, or worse, a life-threatening adrenal crisis [4]. Acute management for an adrenal crisis involves glucocorticoid replacement (e.g., hydrocortisone) and fluid resuscitation, with mineralocorticoid replacement (e.g., fludrocortisone) as needed. Dosing should be monitored closely through clinical signs

and symptoms to ensure that the patient is appropriately treated [4].

Hypercoagulable states, such as antiphospholipid syndrome, have been well documented as a common risk factor for bilateral adrenal hemorrhage [2]. Interestingly, cessation of anticoagulation has been mentioned as a potential trigger for adrenal hemorrhage [5] [6]. Additionally, the usage of anticoagulants such as heparin has been associated with adrenal hemorrhage in the context of heparin-induced thrombocytopenia, which is a pro-thrombotic state [2]. Anticoagulants have also been associated with adrenal hemorrhage in the context of increased bleeding risk [7]. Furthermore, direct oral anticoagulants (DOACs) and warfarin have been increasingly recognized as precipitants of BAH, which is more evident in patients with underlying thrombotic disorders [8].

In this case, we present a 54-year-old male who initially presented with nausea and vomiting with abdominal and flank pain. He was later diagnosed with BAH complicated by adrenal crisis and myocarditis.

2. Case Report

The patient is a 54-year-old male with a past medical history of unprovoked VTE, dural sinus thrombosis on warfarin, hiatal hernia, and gastroesophageal reflux disease. He presented to the hospital with complaints of nausea and vomiting along with abdominal and flank pain that began shortly after he was preparing for his colonoscopy, which was scheduled for that afternoon. In the five days prior to the colonoscopy, the patient was holding his warfarin, in line with preparation for the procedure. The colonoscopy was intended as a health maintenance screening for the presence of colorectal neoplasms.

In the emergency department, the patient got a CT scan which showed esophageal thickening, concerning esophagitis for a neoplasm. Furthermore, the CT also showed bilateral adrenal hyperintensities, suspicious for either a hemorrhage or mass. His labs remained normal aside from mild leukocytosis, with a white blood cell count of 12,000. At this point, the patient remained hemodynamically stable with unremarkable bloodwork. Though there was a pending MRI from the previous day, the patient was determined to be stable, and clinical judgement was made to initiate the patient on anticoagulation due to his history of venous thromboembolism.

On day 2, the patient's vitals became unstable with systolic blood pressures in the 90 s and tachycardia with rates in the 120 s. Additionally, the patient became febrile at 103 degrees Fahrenheit. Results of the MRI imaging from the previous day were consistent with AH Heparin was reversed with protamine sulfate and the patient was started on hydrocortisone 100 mg once, followed by hydrocortisone 50 mg three times daily for adrenal insufficiency (**Figure 1**). Septic workup was also performed, and the patient was started on intravenous fluids and antibiotics which led to hemodynamic stabilization. Repeat CT imaging showed no significant changes in size of the BAH.

On day 3, the endocrinology service was consulted for recommendations and clearance of the patient for esophagogastroduodenoscopy (EGD). They

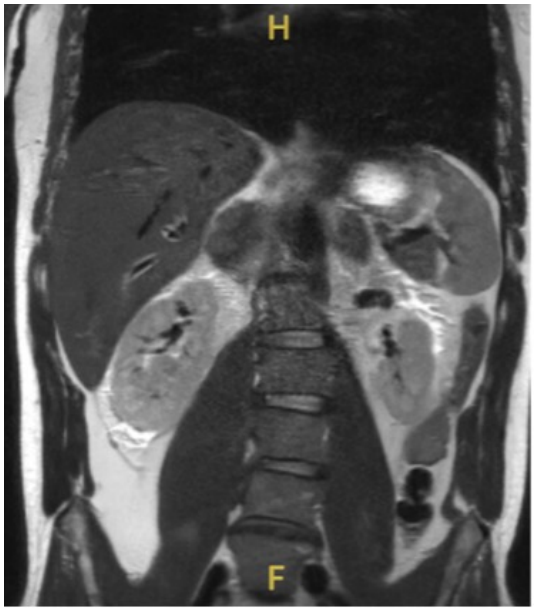

Figure 1. MRI Abdomen and Pelvis. This abdominal MRI demonstrates bilateral adrenal abnormalities with minimal enhancement and signal changes consistent with hemorrhage as opposed to solid lesions. Furthermore, there are mildly enlarged retroperitoneal lymph nodes observed, which are more numerous than we would typically expect. Additionally, the liver shows signs of steatotic liver disease. The findings described in the imaging are consistent with acute bilateral adrenal hemorrhage in the appropriate clinical context. Follow-up imaging is necessary to assess for stability or resolution.

recommended a hydrocortisone taper, continuing with 50 mg three times daily for another day, followed by 25 mg every 8 hours the next day, then decreasing hydrocortisone to 10 mg in the morning and 5 mg in the afternoon while starting fludrocortisone 0.05 mg daily. The decrease in dosage was tolerated well by the patient. Furthermore, endocrinology recommended for the patient to have a basic metabolic panel done a week later and to be seen in the clinic for follow-up in 4 - 6 weeks, with plans to repeat the MRI of the abdomen and pelvis in 2 - 3 months to ensure resolution of the BAH. On day 4, vascular surgery was also consulted for recommendations of how to manage the patient's anticoagulation in the setting of BAHs. Their recommendation was to place an inferior vena cava (IVC) filter, which was placed without complications. Additionally, the patient underwent an EGD without complications as well. On day 5, hematology service was consulted for thrombocytopenia after platelets decreased to 70,000 platelets/mcL. There was concern for possible hypercoagulable state due to the patient's history of dural sinus thrombosis in addition to unprovoked DVT/PE.

Hypercoagulability workup was started by the hematology service, which included haptoglobin, cardiolipin antibody, antiphospholipid antibody, antithrombin III antigen, Factor 5 Leiden, APTT ANA, and JAK2 V617F mutation. Haptoglobin was elevated at 316 mg/dL. ESR was elevated at 48 mm/hr. Meanwhile, antithrombin III antigen (at 87%), Factor 5 Leiden, and APTT (at 34.6 sec) were all negative. JAK2 V617F mutation was not detected in whole blood. The

Antiphospholipid Antibody Panel including beta-2 glycoprotein IgG and IgM (at 16.1 SGU and 9.0 SGU, respectively), cardiolipin antibody IgG and IgM (at 15.3 GPL and 5.7 GPL, respectively), Dilute Russell's Viper Venom Time (DRVVT), and lupus anticoagulant were also completed with elevations being noted in the DRVVT ratio at 2.05 and lupus anticoagulant at 52.5 sec. Additionally, the Antinuclear Antibody (ANA) titers were drawn and were positive at a titer of 1:640 with a speckled pattern. The lab findings were consistent with an underlying autoimmune pattern and thus required further investigation. Furthermore, rheumatology service was consulted due to the elevated lupus anticoagulant. Rheumatology work-up included following up on MPO and PR3 reflex to ANCA for vasculitis concerns and IgG subclasses for IgG4-related diseases. The MPO and PR3 reflex to ANCA was negative at 0.0 AU/mL, and the IgG subclasses for IgG4-related diseases were also negative (IgG1: 380 mg/dL, IgG2: 176 mg/dL, IgG3: 38 mg/dL, IgG4: 27 mg/dL). Rheumatology believed that there could be a possibility for antiphospholipid syndrome considering the elevated lupus anticoagulant and history of clotting disorders, and recommended outpatient follow-up following discharge. CT-guided bone marrow aspiration and biopsy from the left medial posterior iliac bone were completed due to progressing thrombocytopenia and showed hypercellular bone marrow for age with megakaryocyte hyperplasia without atypia. Following clearance from hematology service, low-dose heparin therapy was resumed due to concerns of hypercoagulation.

On day 10, ST segment elevations were found on telemetry, which alerted staff to run an ECG, which showed ST-elevations in the inferior and lateral leads with reciprocal depression suggestive of inferior lateral acute infarction, although the patient was not complaining of chest pain or associated symptoms at the time. Troponin levels were elevated at 50 ng/mL and cardiac catheterization showed patent coronary arteries with evidence of coronary artery disease. With additional careful review of EKG, diffuse ST elevations were noted without T wave inversions, as well as possible PR elevation in aVR, which in conjunction with the elevated troponin levels were suggestive of myopericarditis. The patient underwent a transthoracic echocardiogram which showed an ejection fraction of 10% - 15% with diffuse regional wall abnormalities. Due to these findings, an intra-aortic balloon pump was placed. After the cardiac catheterization, he was moved to the ICU for further management. The patient had worsening hemodynamics, including signs of hypoxia and hypotension with systolic blood pressures in the 80s consistent with shock. He also developed an acute kidney injury (AKI) with a rapid increase in serum creatinine concentrations from 0.99 mg/dL to 2.00 mg/dL within 24 hours, and metabolic acidosis with compensatory respiratory alkalosis. Cardiology concluded that the patient developed acute fulminant myocarditis which resulted in early cardiogenic shock. Ultimately, the decision was made to transfer the patient to a higher level of care and advanced mechanical support therapies.

In the Cardiac ICU of the higher care facility, the patient was weaned off the

intra-aortic pump and transitioned to afterload management care (Hydralazine and Isosorbide Dinitrate), and eventually Valsartan long-term therapy. Following an incident of acute respiratory failure in the setting of suspected Diffuse Alveolar Hemorrhage (DAH) and Immune Thrombocytopenic Purpura (ITP), the patient received a platelet transfusion. Meanwhile, Rheumatology continued to follow up on the patient with IVIG, Rituximab, and Hydroxychloroquine. A repeat echocardiogram revealed improved left ventricular function of 52%. Likewise, the patient got a Cardiac MRI which was consistent with ischemic cardiomyopathy and myocardial infarction with nonobstructive coronary arteries. Following stabilization, Endocrinology and Rheumatology both decided on a prednisone taper of 60 mg for 2 weeks, followed by 40 mg for 2 weeks, with a plan for outpatient Endocrinology and Rheumatology follow-up for medication management. The patient was discharged on an enoxaparin bridge to warfarin and a cardiac home event monitor.

Three months post-discharge, the patient was re-evaluated by Vascular Surgery, which revealed that the patient had resumed anticoagulation therapy with Coumadin without any signs of recurrent bleeding. Furthermore, the patient continued to face chronic lower extremity symptoms such as edema, discoloration, and intermittent calf pain of the bilateral lower extremities, which was more prominent in the limb affected by their prior DVT episodes. The provider of this visit concluded that the patient's lower extremity edema was likely due to post-thrombotic syndrome with his history of DVT. From this presentation, it was recommended for the patient to increase their compression stockings to 20 to 30 milligrams of mercury and elevate the extremities. Additional follow-up was scheduled for 3 weeks later. The persistence of the patient's symptoms despite the use of Coumadin suggests ongoing venous insufficiency, emphasizing the importance of long-term vascular surveillance for patients with a history of recurrent thrombotic events and anticoagulation-associated complications. With the patient's history, initial presentation, and condition at three months follow-up, continuing close follow-up and adding on adjunctive interventions like compression stockings and extremity elevation may be warranted to prevent further exacerbation and complications.

3. Discussion

Adrenal hemorrhage is a rare but often life-threatening condition in which there is bleeding within the adrenal capsule. It has a mortality risk of around 15%, with a potential higher risk if not accurately identified and managed promptly [9]. It results from both traumatic and nontraumatic sources and often presents with nonspecific symptomatology, innovations in imaging technology have improved detection in hospitalized patients [4]. This makes clinical diagnosis more frequent, as historically this was a pathology mostly described post-mortem [10]. The adrenal glands are susceptible to hemorrhage given their vascular supply and architecture. This vascular architecture is characterized by an abundant arterial supply

with a high rate of arterial inflow that is contrasted with limited venous drainage through a singular vein. During periods of hemodynamic stress, an increase in adrenocorticotropic hormone (ACTH) promotes blood flow to the adrenal glands while simultaneously a catecholamine surge constricts venous circulation, increasing pressure, promoting platelet aggregation and eventually leading to rupture of thin-walled vessels resulting in hemorrhage and infarction [4].

AH can arise from a variety of etiologies including trauma, anticoagulation, pregnancy, autoimmune conditions, hypercoagulable state, infections, neonatal stress, and adrenal tumors. Trauma is a primary cause, particularly in right-sided AH due to blunt abdominal injury in which there is liver-adrenal compression. Anticoagulation therapy may lead to AH via thrombocytopenia and increased venous pressure. AH has also been associated with states of hypercoagulability such as Systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS). Other associated causes include post-infection Waterhouse-Friderichsen syndrome, neonatal stress, and adrenal tumors such as pheochromocytoma and myelolipoma. These etiologies pose risks, each with unique imaging and management approaches depending on the underlying pathology and severity [1].

In many cases, unilateral AH is clinically silent, however bilateral adrenal hemorrhage is often associated with fatal primary adrenal insufficiency in 16% - 50% of patients [1]. This happens when the hemorrhage causes significant damage to the adrenal cortex tissue, which in turn causes electrolyte imbalances and hypotension. Two principal characteristics of BAH have been described, biochemical evidence of hemorrhage with a fall in hemoglobin and hematocrit as well as hormonal appearance of adrenal insufficiency as demonstrated by hyponatremia and hyperkalemia [11]. Extensive damage to the cortex also interferes with adrenal hormone production, a sequela that can potentially lead to an Addisonian crisis. Recognizing and treating this is vital because it can be fatal, yet it presents with non-specific symptoms such as abdominal pain, hypotension, weakness, and vomiting as the patient presented in this case. Adrenal insufficiency is usually diagnosed by laboratory confirmation of mineralocorticoid deficiency and measuring plasma renin and aldosterone levels. Imaging is necessary, with CT used to show adrenal enlargement as well as hyperdensity, such as in this case in which the patient's CT showed bilateral adrenal hyperintensities, and MRI to detect hemorrhagic patterns as well as the timing of the hemorrhage. It is imperative to administer IV corticosteroids, specifically hydrocortisone, to stabilize the patient's blood pressure and mimic physiological stress response if adrenal insufficiency is suspected. In refractory cases, surgical adrenalectomy or embolization may be necessary [12].

In this case, several overlapping predisposing factors likely contributed to the development of BAH. As such, this case highlights the importance of considering the interplay of several risk factors in patients with multiple complex health issues as they heighten the possibility and severity of a BAH. This patient's, previously diagnosed venous thromboembolisms with underlying hypercoagulability, cessation

of warfarin therapy, and the stress of colonoscopy preparation combined to create a risk factor profile that likely contributed to subsequent BAH. Complex clinical scenarios such as this one highlight the necessity of thorough planning of anticoagulation strategies to mitigate the risk of BAH in patients with predisposing risk factors. These factors have been documented as potential risk factors for the development of AH. Specifically, as aforementioned BAHs are rare, but they are often encountered in patients with predisposing factors such as coagulopathies and anticoagulant therapy. Moreover, warfarin therapy is an established risk factor for spontaneous hemorrhage, especially in highly vascular organs like the adrenal glands. In a study on patients requiring anticoagulation after knee arthroplasty and no previous need for it, the implementation of anticoagulant therapy with agents such as heparin and warfarin increased the risk of AH 5- to 10-fold [13]. While there is little documented in literature regarding the relationship between warfarin cessation and development of BAH, studies have shown that anticoagulation therapy presents an increased risk of BAH with one study showing anticoagulation as the etiology for approximately 11% of BAH cases [7]. Similarly, having a history of VTE predisposed this patient to BAH. This potential link between BAH and VTE may be due to increased susceptibility of the adrenal vasculature to thrombosis. VTE often results from a prothrombotic state and thus the presence of multiple VTEs in a patient increases their risk of adrenal vein thrombosis leading then to AH. An underlying hypercoagulable state due to the presence of antiphospholipid antibodies, which was present in this case, has a strong association with the occurrence and recurrence of BAH. The presence of antiphospholipid antibodies has been described as the most common identifiable risk factor for AH, as well as the most common etiology of BAH [7]. Patients that have these are likely to develop AH in the presence of stress or precipitating factors like infection or anti-coagulation [14]. In many cases, AH and subsequent primary adrenal insufficiency may be the first manifestation of antiphospholipid syndrome and the most common associated endocrine complication [15].

Management of patients with overlapping conditions, as in this case, can present significant clinical challenges. There needs to be a balancing act between proper management of anticoagulation therapy while addressing acute adrenal insufficiency and hypercoagulability which often calls for a multidisciplinary approach. For the patient in this case, timely identification of adrenal insufficiency secondary to BAH as well as prompt treatment with glucocorticoids likely prevented progression to fatal adrenal crisis. Given the patient's underlying risk factors as well as the presence of acute adrenal insufficiency secondary to BAH, it is likely that lifelong steroid replacement will be warranted [12].

Long-term outcomes for patients with concurrent glucocorticoid and anticoagulation therapy in patients with acute adrenal insufficiency and underlying hypercoagulability can be complex. Glucocorticoid therapy has been linked to a potential hypercoagulable state due to an elevation in plasminogen activator inhibitor 1 which increases levels of coagulation factors along with impairment of fibrinolytic

capacity [16]. Because of this, management of these patients usually necessitates a multidisciplinary approach. Endocrinology manages long-term steroid therapy and monitors for recurrence of adrenal insufficiency, while hematology oversees the anticoagulation therapy. Frequent follow-up with cortisol and aldosterone monitoring will also most likely be essential to ensure adequate adrenal function. In this case, collaboration between endocrinology, vascular surgery, and hematology services was imperative to ensure appropriate anticoagulation management and long-term regular follow-up to monitor for adverse effects and adjust treatment as needed.

4. Conclusion

This case highlights the importance of accurately recognizing and managing bilateral adrenal hemorrhage in patients presenting with nonspecific symptoms such as abdominal pain and nausea, in the context of a history of venous thromboembolism and possible stress due to a procedure, such as a colonoscopy as in this case. BAH should be part of the differential diagnosis in any patient with a similar history and presenting with symptoms ranging from weakness to hypotension. When suspected, rapid imaging and prompt treatment with systemic corticosteroids are crucial to prevent progression to primary adrenal insufficiency and subsequent adrenal crisis.

Consent

For this case report, informed consent was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Badawy, M., Gaballah, A.H., Ganeshan, D., Abdelalziz, A., Remer, E.M., Alsabbagh, M., *et al.* (2021) Adrenal Hemorrhage and Hemorrhagic Masses; Diagnostic Workup and Imaging Findings. *The British Journal of Radiology*, **94**, Article ID: 20210753. https://doi.org/10.1259/bjr.20210753

[2] Aloini, M.E., Manella, S., Biondo, I., Maggio, R., Roberto, G., Ricci, F., *et al.* (2023) Bilateral Adrenal Hemorrhage: Learning Notes from Clinical Practice and Literature Review. *Frontiers in Endocrinology*, **14**, Article 1233710. https://doi.org/10.3389/fendo.2023.1233710

[3] Alshahrani, M.A., Bin Saeedan, M., Alkhunaizan, T., Aljohani, I.M. and Azzumeea, F.M. (2018) Bilateral Adrenal Abnormalities: Imaging Review of Different Entities. *Abdominal Radiology*, **44**, 154-179. https://doi.org/10.1007/s00261-018-1670-5

[4] Elhassan, Y.S., Ronchi, C.L., Wijewickrama, P. and Baldeweg, S.E. (2022) Approach to the Patient with Adrenal Hemorrhage. *The Journal of Clinical Endocrinology & Metabolism*, **108**, 995-1006. https://doi.org/10.1210/clinem/dgac672

[5] Meade-Aguilar, J.A., Figueroa-Parra, G., Yang, J.X., Langenfeld, H.E., González-Treviño, M., Dogra, P., *et al.* (2024) Clinical Presentation and Outcomes in Patients with Antiphospholipid Syndrome-Associated Adrenal Hemorrhage. a Multicenter Cohort

Study and Systematic Literature Review. *Clinical Immunology*, **260**, Article ID: 109906. https://doi.org/10.1016/j.clim.2024.109906

[6] Aldaajani, H., Albahrani, S., Saleh, K. and Alghanim, K. (2018) Bilateral Adrenal Hemorrhage in Antiphospholipid Syndrome. *Saudi Medical Journal*, **39**, 829-833. https://doi.org/10.15537/smj.2018.8.22437

[7] Dogra, P., Chinthapalli, M., Sandooja, R., Rahimi, L., Iniguez-Ariza, N.M., Foster, T., *et al.* (2024) Adrenal Hemorrhage: A Comprehensive Analysis of a Heterogeneous Entity—Etiology, Presentation, Management, and Outcomes. *Mayo Clinic Proceedings*, **99**, 375-386. https://doi.org/10.1016/j.mayocp.2023.10.002

[8] Al-Rawi, S., Seidahmed, M., Emam, S.S. and Othman, E.S. (2023) A 65-Year-Old Man with Bilateral Adrenal Hemorrhage Following Prophylaxis for Postoperative Deep Vein Thrombosis with Rivaroxaban. *American Journal of Case Reports*, **24**, e939816. https://doi.org/10.12659/ajcr.939816

[9] Sakalkale, A., Choi, C.C., Krawitz, R. and Yeung, J.M.C. (2024) Two Cases of Atraumatic Adrenal Hemorrhage: A Review of Active Management, Conservative Management, and Challenges Faced. *Radiology Case Reports*, **19**, 2395-2401. https://doi.org/10.1016/j.radcr.2024.03.005

[10] Simon, D.R. and Palese, M.A. (2009) Clinical Update on the Management of Adrenal Hemorrhage. *Current Urology Reports*, **10**, 78-83. https://doi.org/10.1007/s11934-009-0014-y

[11] Caron, P., Chabannier, M., Cambus, J., Fortenfant, F., Otal, P. and Suc, J. (1998) Definitive Adrenal Insufficiency Due to Bilateral Adrenal Hemorrhage and Primary Antiphospholipid Syndrome. *The Journal of Clinical Endocrinology & Metabolism*, **83**, 1437-1439. https://doi.org/10.1210/jcem.83.5.4833

[12] May, C.J.H., Asia, M., Karavitaki, N., Arlt, W., Guest, P. and O'Reilly, M.W. (2017) Bilateral Adrenal Haemorrhage. *QJM*, **110**, 169-171. https://doi.org/10.1093/qjmed/hcw212

[13] LaBan, M.M., Whitmore, C.E. and Taylor, R.S. (2003) Bilateral Adrenal Hemorrhage after Anticoagulation Prophylaxis for Bilateral Knee Arthroplasty. *American Journal of Physical Medicine & Rehabilitation*, **82**, 418-420. https://doi.org/10.1097/01.phm.0000064741.97586.e4

[14] Vella, A., Nippoldt, T.B. and Morris, J.C. (2001) Adrenal Hemorrhage: A 25-Year Experience at the Mayo Clinic. *Mayo Clinic Proceedings*, **76**, 161-168. https://doi.org/10.1016/s0025-6196(11)63123-6

[15] Bouki, K., Venetsanaki, V., Chrysoulaki, M., Pateromichelaki, A., Betsi, G., Daraki, V., *et al.* (2023) Primary Adrenal Insufficiency Due to Bilateral Adrenal Hemorrhage-Adrenal Infarction in a Patient with Systemic Lupus Erythematosus and Antiphospholipid Syndrome: Case Presentation and Review of the Literature. *Hormones*, **22**, 521-531. https://doi.org/10.1007/s42000-023-00463-5

[16] Coelho, M.C.A., Santos, C.V., Neto, L.V. and Gadelha, M.R. (2015) Adverse Effects of Glucocorticoids: Coagulopathy. *European Journal of Endocrinology*, **173**, M11-M21. https://doi.org/10.1530/eje-15-0198