CASE REPORT

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Esophageal ulcer associated with mild hemophilia A: case report

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Abstract

In this paper, we present the case of a 68-year-old male with personal medical history of coagulopathy issues, who presented to our Emergency Room (Emergency County Hospital, Arad, Romania) with bleeding of the superior tract of the digestive system; the case was difficult to manage, thus warranting the intervention of the Department of Gastroenterology. Endoscopy was performed to localize the site of bleeding and to stop the hemorrhage. This procedure was not successful. The patient was transferred to our Intensive Care Unit where different medications, such as proton pump inhibitor, hemostatic agent and prokinetic drugs were administered. Unfortunately, our attempt to stop bleeding failed; this led us to expand our investigation. We focused on a possible hemophilia as the cause of bleeding, which was confirmed as hemophilia A through the coagulometry test after a period of three days. Patient medical history and coagulation test led us to believe that this is a very rare case of a mild hemophilia A. Finally, the correction of Factor VIII deficiency and repeated endoscopic hemostasis clip was able to stop patients bleeding and ensured a favorable clinical evolution of the patient.

Keywords: esophageal ulcer, hemophilia A, hemorrhage, endoscopy.

Introduction

Peptic ulcer disease is characterized by disruption of the inner lining of the gastrointestinal (GI) tract due to the secretion of gastric acid or pepsin [1]. Patient's peptic ulcer was located at the superior digestive mucosa, and this is found in approximatively 4% of adult population [2, 3], while the most frequent types are gastric and duodenal ulcers [4, 5], esophageal type is very rare. Esophageal ulcers occur due to mucosal structural changes which exposures the profound esophageal layers to various irritating factors [5].

There are many factors that predispose patients to esophageal ulcers namely as gastric acid [hiatal hernia and gastroesophageal reflux disease (GERD)] [6–9], medical treatment with anti-inflammatory drugs or antibiotics, ingestion of corrosive substances or alcohol, infection caused by viruses, fungus, and tuberculosis.

Diagnosis of esophageal ulcer was based on clinical symptoms and on endoscopic findings [10], other investigations, such as computed tomography (CT) using contrast substance is rarely needed. The clinical complications that can occur are esophageal wall perforation, esophageal cancer, stenosis, and bleeding [10, 11]. Severe esophageal bleeding requires emergency endoscopy, diet, antiacid, proton pump inhibitor, antibiotics and in some cases surgery [12].

Hemophilia A is an X-linked, recessive disorder due to the deficiency of functional plasma clotting Factor VIII (FVIII), which may be inherited or because of spontaneous mutation. Rarely, development of autoantibodies to FVIII results in acquired hemophilia A [13]. Depending on the level of FVIII activity, patients with hemophilia may present easy bruising, inadequate clotting in case of trauma or mild injury. It should be noted that in severe hemophilia patient may have spontaneous hemorrhage. Normal values for FVIII assays are 50–150%. Values in hemophilia A are as follows: mild: >5%, moderate: 1–5%, severe: <1% from the clotting factor level [14–16].

The association between peptic ulcer and hemophilia is extremely rare, only few clinical cases were reported

worldwide, and the most frequent association was described in case of gastric ulcer [17].

Aim

Our aim was to highlight the link between coagulation deficiency (prolonged bleeding) and esophageal ulcer bleeding. We want to emphasize the difficulty of diagnosing mild hemophilia A, as well as the effectiveness of treatment (correction of the plasma value of FVIII) associated with endoscopic hemostasis. We also reviewed the wide range of possible clinical complications due to severe hemorrhage and delayed hemostasis treatment.

Case presentation

We present a clinical case of a 68-year-old male patient, admitted at Emergency County Hospital, Arad, Romania, with the diagnosis of superior digestive tract hemorrhage. Our patient had a history of type 2 diabetes, high blood pressure and two surgical interventions (cholecystectomy, and dental extraction without prolonged postoperative bleeding).

Patient presented one first episode of bleeding at home. An hour later he was rushed to the Emergency Room (ER) where diagnostic endoscopy was carried out (Figure 1A). Endoscopic hemostasis treatment (Figure 1B) using clips was performed and the bleeding was temporary stopped (Olympus Exera III). During the first endoscopy, patient lost consciousness, which required orotracheal intubation, and subsequently he was admitted to Intensive Care Unit (ICU).

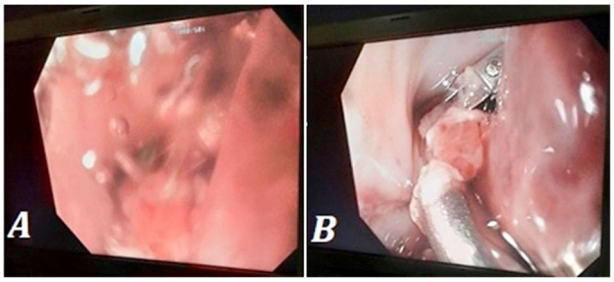

Figure 1 – ***The endoscopic images of the patient at the time of hospital admission: (A) Esophageal hemorrhagic ulcer; (B) Endoscopic hemostasis using clips.***

His clinical status on admission to ICU was life threatening because he presented altered mental status (Glasgow Coma Scale – 12) and was placed on ventilator support (synchronized intermittent mandatory ventilation – Dräger Savina 300). Patient's hemodynamic state was stable, with blood pressure value of 100/60 mmHg. On admission, laboratory results were the following: hemoglobin 6.3 g/dL, hematocrit 22%, blood urea 94 mg/dL, blood creatinine 0.7 mg/dL, lactic acid 4.5 mmol/l, glycemia 350 mg/dL, activated partial thromboplastin time (APTT) 38 seconds, international normalized ratio (INR) 0.8, transaminases 9.9 U/L, direct bilirubin 0.13 mg/dL, total bilirubin 0.3 mg/dL, serum total protein 5 g/dL, serum albumin 28 g/L.

The patient was transfused with whole blood and fresh frozen plasma and was also maintained on continuous saline and colloid infusion. Hemostatic and prokinetic agents were also administered along with proton pump inhibitor (Pantoprazole) continuous infusion. Osmotic diuretic therapy was used to reduce pulmonary congestion (Figure 2A) and possible cerebral edema.

His clinical condition improved (Figure 2B) and he was weaned from ventilator two days after the admission to ICU. Radiographies shown below revealed the difference in patient's pulmonary congestion.

After the first days on ICU, four more episodes of bleeding happened even though endoscopic hemostasis was attempted. To exclude a possible esophageal fistula, chest CT angiography was also performed (Figure 3, A and B).

Due to prolonged prothrombin time, which could not be corrected by transfusion with fresh frozen plasma and cryoprecipitate, more clinical investigations to rule out hemophilia few days after admission were warranted. This type of analysis could not be performed in our Medical Center. It was necessary to send the blood sample to an external laboratory and the result was released after 3–4 days. The low plasma value of FVIII was established through coagulometer test (12% of the normal value) thus sustaining the diagnosis of hemophilia, which was immediately corrected by infusion of this factor (3000 IU every 12 hours for three days). The transfusion of FVIII concentrate increased the plasma value from 12% to 50%. After this correction together with the 5th hemostasis endoscopic attempt, patient's clinical status significantly improved. The last endoscopic image, after the complete treatment, shows cicatricial ulcer with clips. Peripheral blood smear analysis was also performed and revealed a slightly hypochromic, microcytic red blood cells (Figure 4, A and B).

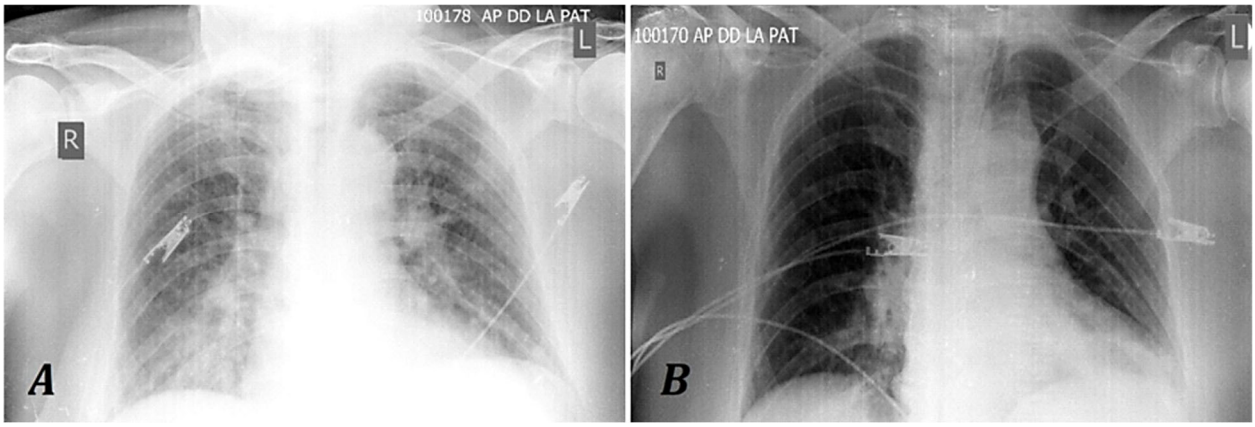

Figure 2 – ***The radiographic images of the patient: (A) The radiographic image on admission time shows bilateral alveolar condensation and hilar stasis; (B) The radiographic image after two days of intensive treatment shows only hilar stasis.***

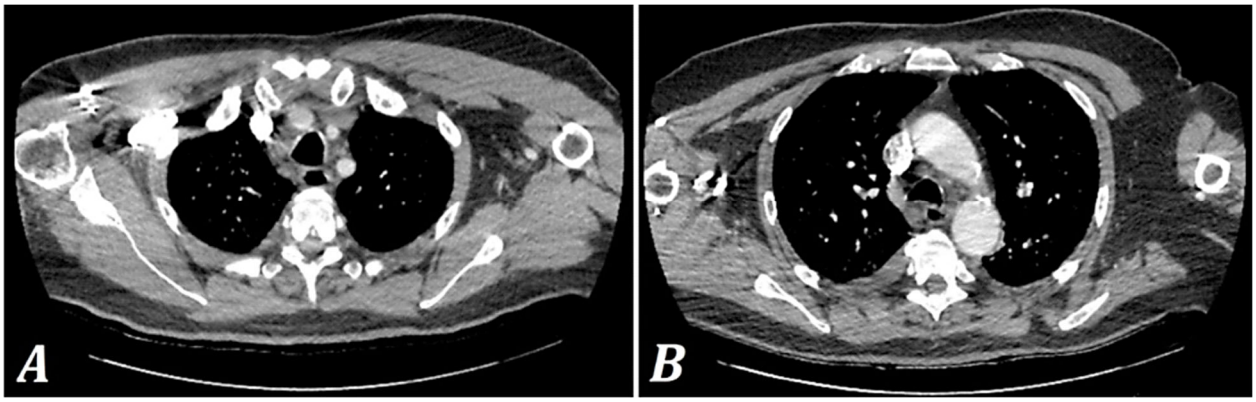

Figure 3 – ***CT images after few days of intensive treatment: (A) CT angiography image of proximal portion of esophagus; the normal structure of esophageal and tracheal wall; lack of contrast-media extravasation from vascular structure; (B) CT angiography image of distal portion of esophagus; the image also shows normal structure of esophageal and tracheal wall; lack of contrast-media extravasation from vascular structure. CT: Computed tomography.***

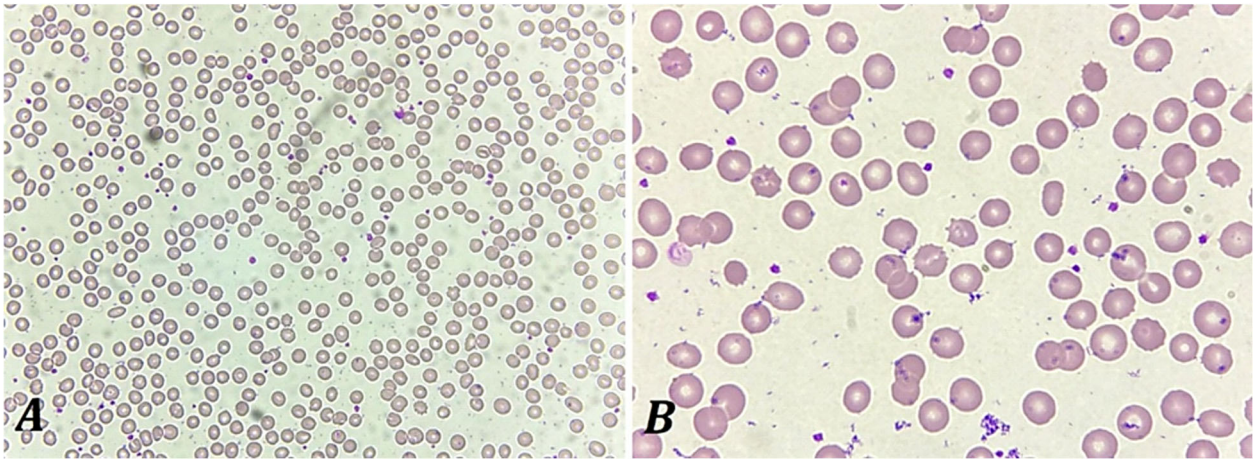

Figure 4 – ***(A) Peripheral blood smear showing microcytic, hypochromic red blood cells; (B) Microcytic, hypochromic red blood cells and normal platelets. May-Grünwald–Giemsa (MGG) staining: (A) ×400; (B) ×1000.***

We also must mention patient's hospital acquired infection while on ICU. This complication may be related to the numerous digestive bleeding episodes, required broad-spectrum antibiotic therapy, and prolonged his stay on ICU.

After controlling the bleeding, patient was transferred to Department of Gastroenterology for further treatment where he was later discharged in a good clinical condition after seven days of hospitalization.

Discussions

We present a very rare case of patient with digestive bleeding from esophageal ulcer and an associated coagulopathy with unknown origin, which was later proved to be mild hemophilia A.

Esophageal ulcer is a relatively rare cause of bleeding among GI diseases. The most common cause of esophageal ulcer is duodenogastric reflux (DGR) disease [18]. GERD is present in about 13% of the world's population [19, 20]. A study conducted by Higuchi *et al.* (2003) showed that DGR disease is responsible for about 66% of esophageal ulcers [21].

Another important cause in the pathogenesis of esophageal ulcers is represented by the ingestion of chemical substances, and especially drugs (antibiotics, antihypertensive, non-steroidal anti-inflammatory drugs, etc.). The incidence is estimated at 3.9 per 100 000 individuals per year [22]. Some studies claim that more than 100 drugs can cause damage to the esophagus [22, 23]. Unfortunately, in our patient, the cause of the esophageal ulcer was not identified.

Hemophilia A is a genetic condition that predominantly affects men, producing bleeding that causes problems in hemostasis, having as the cause the deficiency of the synthesis of coagulation FVIII, being an X-linked recessive inherited disease.

The severity of this case with altered mental status and cardiac failure on admission to ICU should be noted. Patient's altered mental status could be caused by possible cerebral edema [24], edema that can be explained by tissue hypoxia due to severe anemia [25] and also by the low level of albumin [26, 27]. Clinical research shows that hypoalbuminemia is frequently determined in case of critical patient with systemic inflammatory syndrome [28, 29]. The risk factors, such as diabetes mellitus, high blood pressure and the age of the patient [30], can determine cardiac clinical complication. The known cardiac pathology of the patient, in the setting of the severe anemia, explains the occurrence of heart failure.

The presence of sepsis secondary to soft tissue infection could be regarded because of multiple bleeding episodes and secondary anemia. It was clearly demonstrated that the stress response, mediated by cytokines [31] and hormones [32], can be triggered by any episode of severe bleeding. This human response to stress has an immunosuppressant effect [33].

An element that could have hindered the rapid establishment of the diagnosis in this case was severe bleeding in a patient with a coagulopathy (a prolonged APTT and normal INR) and a history of major surgery without severe bleeding [34]. In our case, each bleeding episode was severe, resulting in severe anemia [35], but without hypovolemic shock, which contrasts with data obtained from other studies [36, 37].

Coagulation tests, performed few days after admission, found a low level of FVIII, and a normal level of Factor IX. It is worth to be mentioned that the coagulometry test which demonstrated the plasma deficiency of FVIII could not be performed in our Laboratory Unit, and the results were obtained in a period of few days. This delay in getting the results led to an aggravated clinical status.

We could not determine the presence of FVIII inhibitors to exclude an acquired hemophilia. Later, we found out the patient has two nephews with mild hemophilia A and this data also sustained the diagnosis of mild hemophilia A [38, 39].

Conclusions

More attention should be focused on severe digestive hemorrhage that does not respond to endoscopic treatment. It is always a good idea to take in account a possible coagulopathy. In some circumstances, coagulation times routinely determined, cannot accurately predict hemostasis disturbance but this fact should not be an impediment for performing additional coagulation tests. All these investigations are necessary to make the treatment more efficient. In this particular case, the efficient treatment of hemorrhagic esophageal ulcer was possible only after the diagnosis and treatment of mild hemophilia A, which required teamwork, a good collaboration between many specialists from different medical fields.

Conflict of interests

The authors declare that they have no conflict of interests. The authors are responsible of the content and writing of the paper.

Authors' contribution

Cristian Mircea Nicolescu and Alexandru Neşiu have equally contributed to this manuscript.

References

[1] Narayanan M, Reddy KM, Marsicano E. Peptic ulcer disease and *Helicobacter pylori* infection. Mo Med, 2018, 115(3):219–224. PMID: 30228726 PMCID: PMC6140150

[2] Wang AY, Peura DA. The prevalence and incidence of *Helicobacter pylori*-associated peptic ulcer disease and upper gastrointestinal bleeding throughout the world. Gastrointest Endosc Clin N Am, 2011, 21(4):613–635. https://doi.org/10.1016/j.giec.2011.07.011 PMID: 21944414

[3] GBD 2013 Mortality and Causes of Death Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet, 2015, 385(9963):117–171. https://doi.org/10.1016/S0140-6736(14)61682-2 PMID: 25530442 PMCID: PMC4340604

[4] Menon S, Trudgill N. Risk factors in the aetiology of hiatus hernia: a meta-analysis. Eur J Gastroenterol Hepatol, 2011, 23(2):133–138. https://doi.org/10.1097/MEG.0b013e3283426f57 PMID: 21178776

[5] Gordon C, Kang JY, Neild PJ, Maxwell JD. The role of the hiatus hernia in gastro-oesophageal reflux disease. Aliment Pharmacol Ther, 2004, 20(7):719–732. https://doi.org/10.1111/j.1365-2036.2004.02149.x PMID: 15379832

[6] Karamanolis G, Polymeros D, Triantafyllou K, Adamopoulos A, Barbatzas C, Vafiadis I, Ladas SD. Hiatal hernia predisposes to nocturnal gastro-oesophageal reflux. United European Gastroenterol J, 2013, 1(3):169–174. https://doi.org/10.1177/2050640613490295 PMID: 24917956 PMCID: PMC4040758

[7] Lee YY, Wirz AA, Whiting JGH, Robertson EV, Smith D, Weir A, Kelman AW, Derakhshan MH, McColl KEL. Waist belt and central obesity cause partial hiatus hernia and short-segment acid reflux in asymptomatic volunteers. Gut, 2014, 63(7):1053–1060. https://doi.org/10.1136/gutjnl-2013-305803 PMID: 24064007

[8] Lam S, Hart AR. Does physical activity protect against the development of gastroesophageal reflux disease, Barrett's esophagus, and esophageal adenocarcinoma? A review of the literature with a meta-analysis. Dis Esophagus, 2017, 30(11):1–10. https://doi.org/10.1093/dote/dox099 PMID: 28881908

[9] Swanson JM. The UK Biobank and selection bias. Lancet, 2012, 380(9837):110. https://doi.org/10.1016/S0140-6736(12)61179-9 PMID: 22794246

[10] Proper KI, Singh AS, van Mechelen W, Chinapaw MJM. Sedentary behaviors and health outcomes among adults: a systematic review of prospective studies. Am J Prev Med, 2011, 40(2):174–182. https://doi.org/10.1016/j.amepre.2010.10.015 PMID: 21238866

[11] ASGE Technology Committee; Parsi MA, Trindade AJ, Bhutani MS, Melson J, Navaneethan U, Thosani N, Trikudanathan G, Watson RR, Maple JT. Cryotherapy in gastrointestinal endoscopy. VideoGIE, 2017, 2(5):89–95. https://doi.org/10.1016/j.vgie.2017.01.021 PMID: 29905303 PMCID: PMC5991494

[12] Mastracci L, Grillo F, Parente P, Unti E, Battista S, Spaggiari P, Campora M, Valle L, Fassan M, Fiocca R. Non gastro-esophageal reflux disease related esophagitis: an overview with a histologic diagnostic approach. Pathologica, 2020, 112(3):128–137. https://doi.org/10.32074/1591-951X-156 PMID: 33179617 PMCID: PMC7931579

[13] Benson G, Auerswald G, Dolan G, Duffy A, Hermans C, Ljung R, Morfini M, Šalek SZ. Diagnosis and care of patients with mild haemophilia: practical recommendations for clinical management. Blood Transfus, 2018, 16(6):535–544. https://doi.org/10.2450/2017.0150-17 PMID: 29328905 PMCID: PMC6214819

[14] Makris M, Oldenburg J, Mauser-Bunschoten EP, Peerlinck K, Castaman G, Fijnvandraat K; Subcommittee on Factor VIII, Factor IX and Rare Bleeding Disorders. The definition, diagnosis and management of mild hemophilia A: communication from the SSC of the ISTH. J Thromb Haemost, 2018, 16(12):2530–2533. https://doi.org/10.1111/jth.14315 PMID: 30430726

[15] Pizarro TT, Cominelli F. Cloning IL-1 and the birth of a new era in cytokine biology. J Immunol, 2007, 178(9):5411–5412. https://doi.org/10.4049/jimmunol.178.9.5411 PMID: 17442919

[16] Roy AM, Siddiqui A, Venkata A. Undiagnosed acquired hemophilia A: presenting as recurrent gastrointestinal bleeding. Cureus, 2020, 12(9):e10188. https://doi.org/10.7759/cureus.10188 PMID: 33029467 PMCID: PMC7529483

[17] Damek DM. Cerebral edema altered mental status, seizures, acute stroke, leptomeningeal metastases, and paraneoplastic syndrome. Hematol Oncol Clin North Am, 2010, 24(3):515–535. https://doi.org/10.1016/j.hoc.2010.03.010 PMID: 20488351

[18] Cohen DL, Bermont A, Richter V, Shirin H. Real world management of esophageal ulcers: analysis of their presentation, etiology, and outcomes. Acta Gastroenterol Belg, 2021, 84(3):417–422. https://doi.org/10.51821/84.3.004 PMID: 34599565

[19] Richter JE, Rubenstein JH. Presentation and epidemiology of gastroesophageal reflux disease. Gastroenterology, 2018, 154(2):267–276. https://doi.org/10.1053/j.gastro.2017.07.045 PMID: 28780072 PMCID: PMC5797499

[20] Okamoto T, Suzuki H, Fukuda K. Clinical and endoscopic characteristics of acute esophageal necrosis and severe reflux esophagitis. Medicine (Baltimore), 2021, 100(44):e27672. https://doi.org/10.1097/MD.0000000000027672 PMID: 34871245 PMCID: PMC8568454

[21] Higuchi D, Sugawa C, Shah SH, Tokioka S, Lucas CE. Etiology, treatment, and outcome of esophageal ulcers: a 10-year experience in an urban emergency hospital. J Gastrointest Surg, 2003, 7(7):836–842. https://doi.org/10.1007/s11605-003-0027-7 PMID: 14592655

[22] Hu SW, Chen AC, Wu SF. Drug-induced esophageal ulcer in adolescent population: experience at a single medical center in Central Taiwan. Medicina (Kaunas), 2021, 57(12):1286. https://doi.org/10.3390/medicina57121286 PMID: 34946231 PMCID: PMC8708022
[23] Kim SH, Jeong JB, Kim JW, Koh SJ, Kim BG, Lee KL, Chang MS, Im JP, Kang HW, Shin CM. Clinical and endoscopic characteristics of drug-induced esophagitis. World J Gastroenterol, 2014, 20(31):10994–10999. https://doi.org/10.3748/wjg.v20.i31.10994 PMID: 25152603 PMCID: PMC4138480
[24] LeRoux P. Haemoglobin management in acute brain injury. Curr Opin Crit Care, 2013, 19(2):83–91. https://doi.org/10.1097/MCC.0b013e32835eba43 PMID: 23385374
[25] Lupu A, Miron IC, Cianga AL, Cernomaz AT, Lupu VV, Munteanu D, Ghica DC, Fotea S. The relationship between anemia and *Helicobacter pylori* infection in children. Children (Basel), 2022, 9(9):1324. https://doi.org/10.3390/children9091324 PMID: 36138631 PMCID: PMC9497312
[26] Nicolescu CM, Bedreag O, Osakwe H, Pop A, Nicolescu LC. Evaluation of plasma albumin as a potential prognostic biomarker in patients with traumatic SIRS. Rev Chim (Bucharest), 2017, 68(9):2181–2188. https://doi.org/10.37358/RC.17.9.5852 https://revistadechimie.ro/Articles.asp?ID=5852
[27] Gatta A, Verardo A, Bolognesi M. Hypoalbuminemia. Intern Emerg Med, 2012, 7(Suppl 3):S193–S199. https://doi.org/10.1007/s11739-012-0802-0 PMID: 23073857
[28] Soeters PB, Wolfe RR, Shenkin A. Hypoalbuminemia: pathogenesis and clinical significance. JPEN J Parenter Enteral Nutr, 2019, 43(2):181–193. https://doi.org/10.1002/jpen.1451 PMID: 30288759 PMCID: PMC7379941
[29] Kim S, McClave SA, Martindale RG, Miller KR, Hurt RT. Hypoalbuminemia and clinical outcomes: what is the mechanism behind the relationship? Am Surg, 2017, 83(11):1220–1227. https://doi.org/10.1177/000313481708301123 PMID: 29183523
[30] Sharma A, Christodorescu R, Agbariah A, Duda-Seiman D, Dahdal D, Man D, Kundnani NR, Cretu OM, Dragan S. Cardiovascular risk prediction parameters for better management in rheumatic diseases. Healthcare (Basel), 2022, 10(2):312. https://doi.org/10.3390/healthcare10020312 PMID: 35206926 PMCID: PMC8872463
[31] Shimba A, Ikuta K. Immune-enhancing effects of glucocorticoids in response to day–night cycles and stress. Int Immunol, 2020, 32(11):703–708. https://doi.org/10.1093/intimm/dxaa048 PMID: 32710629
[32] Weber A, Wasiliew P, Kracht M. Interleukin-1 (IL-1) pathway. Sci Signal, 2010, 3(105):cm1. https://doi.org/10.1126/scisignal.3105cm1 PMID: 20086235
[33] Gatti G, Cavallo R, Sartori ML, del Ponte D, Masera R, Salvadori A, Carignola R, Angeli A. Inhibition by cortisol of human natural killer (NK) cell activity. J Steroid Biochem, 1987, 26(1):49–58. https://doi.org/10.1016/0022-4731(87)90030-6 PMID: 2434732
[34] Hunt BJ. Bleeding and coagulopathies in critical care. N Engl J Med, 2014, 370(9):847–859. https://doi.org/10.1056/NEJMra1208626 PMID: 24571757
[35] Ierardi AM, Coppola A, Tortora S, Valconi E, Piacentino F, Fontana F, Stellato E, Cogliati CB, Torzillo D, Giampalma E, Renzulli M, Bargellini I, Cioni R, Scandiffio R, Spinazzola A, Foà RA, Del Giudice C, Venturini M, Carrafiello G. Gastrointestinal bleeding in patients with SARS-CoV-2 infection managed by interventional radiology. J Clin Med, 2021, 10(20):4758. https://doi.org/10.3390/jcm10204758 PMID: 34682879 PMCID: PMC8541615
[36] Lier H, Bernhard M, Hossfeld B. Hypovolämisch-hämorrhagischer Schock [Hypovolemic and hemorrhagic shock]. Anaesthesist, 2018, 67(3):225–244. https://doi.org/10.1007/s00101-018-0411-z PMID: 29404656
[37] Kelley DM. Hypovolemic shock: an overview. Crit Care Nurs Q, 2005, 28(1):2–19; quiz 20–21. https://doi.org/10.1097/00002727-200501000-00002 PMID: 15732421
[38] Lemoine C, Giacobbe AG, Bonifacino E, Karapetyan L, Seaman C. A case of acquired haemophilia A in a 70-year-old post COVID-19 vaccine. Haemophilia, 2022, 28(1):e15–e17. https://doi.org/10.1111/hae.14442 PMID: 34708898 PMCID: PMC8652744
[39] Cameron H, Perez Botero J. Recognition of the unique bleeding pattern and laboratory findings in acquired haemophilia A facilitates prompt treatment of a life-threatening disorder. BMJ Case Rep, 2021, 14(8):e244238. https://doi.org/10.1136/bcr-2021-244238 PMID: 34344656 PMCID: PMC8336148

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