CASE REPORT

Case study about interstitial pneumonitis

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ABSTRACT

This work is about constructing and writing a case report that I want to submit to medical and scientific magazines. The paper starts with an introduction section where the overall theme of discussion is clearly described. A patient with a history of mental illness who consumes 30 to 40 bottles of hair spray and 1.5 liters of hand sanitizer is unconscious at home and lying on the floor for up to a day. He lands in the ER RLS 2 and is sent to the ICU for a Dialysis indication. A chest x-ray reveals a whitish right-sided lung, likely aspiration pneumonia. Difficult to oxygenate; within a day, requires VV-ECMO. VV-ECMO from 6/10-12/10. In a medical illness known as coagulopathy, blood coagulation is disrupted, which increases the risk of bleeding from blood vessels that have been injured. The body uses coagulation as a defense mechanism to stop excessive bleeding or clotting from minor wounds. Impairment in clot formation is the usual term for it. Coagulation stops spontaneous bleeding from wounds, scrapes, burns, bruises, and even internal injuries when paired with blood platelets. The use of evidence-based medicine in developing recommendations for disease management, the booming development of lung transplants in treating severe AIP patients, the well-established lung rehabilitation, and the various molecular biomarkers of IIP used to identify the diagnosis, predict susceptibility, prognosis, and drug effectiveness, were all encouraging developments even though we were unable to do much to help patients with AIP survive.

Key Words: *Volatile substance abuse; Interstitial pneumonia; Coagulation; Sedation; Lung rehabilitation*

INTRODUCTION

The patient, in this case, was discovered unconscious at home, surrounded by empty 1- liter hand sanitizer bottles and 40 hair spray bottles. After being intubated and admitted to the ICU, the workup began. Although the patient deteriorated and required ECMO, they were admitted to the ECMO unit when the x-ray revealed a white lung (aspiration or infiltration). Unfortunately, he subsequently passed unexpectedly owing to several problems and probable illnesses.

CASE PRESENTATION

The term Volatile Substance Abuse (VSA) is most suited to describe the misuse of inhaled solvents. Other addictive ingredients, such as marijuana, cocaine, and methamphetamine, are also used for stimulating properties. For illegal purposes, fentanyl patches have also been heated and their fumes consumed. But because these medicines aren't solvents, they don't have the same chemical and physical characteristics as commonly misused inhalant substances. A patient with a history of mental illness who consumes 30 bottles to 40 bottles of hair spray and 1.5 liters of hand sanitizer is unconscious at home and lying on the floor for up to a day. He lands in the ER RLS 2 and is sent to the ICU for a Dialysis indication. A chest x-ray reveals a whitish right-sided lung, likely aspiration pneumonia. Difficult to oxygenate; within a day, requires VV-ECMO from 6/10-12/10. The patient has severe vasoplegia, multiorgan failure, liver failure, and bone marrow depression and needs massive doses of vasopressor in addition to white blood cells and platelets. Three plasmapheresis treatments will cause vasoplegia to end. In addition, have high myoglobin values after spending a lot of time lying down, remaining above 10,000, as a result of ischemic alterations in the hands and feet that are still demarking and exhibiting signs of ischemia.

DISCUSSION

The effect of his chronic hair spray use is Alveolar pneumonitis. Idiopathic fulminant respiratory failure and acute respiratory distress syndrome-related lung disease known as acute interstitial pneumonia (AIP), also known as Hamman-Rich syndrome, appears suddenly and quickly (ARDS). Clinically, it can be distinguished from other types

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of interstitial pneumonia by the rapid onset of respiratory failure in a patient without a history of lung disease. Patients who have acute interstitial pneumonia run the risk of rapidly decompensating their respiratory system. Therefore, monitoring vital signs should be done carefully. Arterial blood gases can determine the severity of hypoxemia and the requirement for mechanical ventilation. A bilateral air-space diffuse opacities pattern on a chest X-ray typically indicates ARDS. Therefore, it's crucial to rule out cardiac causes of pulmonary edema. To eliminate underlying cardiomyopathy or valve failure, echocardiography is required. In the first 12 hours, High-Resolution Computed Tomography (HRCT) is typically abnormal. It exhibits ground-glass opacities and widespread or patchy air space consolidation. Results from HRCT are correlated with DAD's various phases. Traction bronchiectasis, a sign of transition from the exudative phase to the proliferative fibrotic phase, can also be seen on a CT scan [1].

Testing for inflammatory and connective tissue disorders, such as Dermatomyositis, Sjogren Syndrome, Systemic Lupus Erythematosus (SLE), Rheumatoid Arthritis (RA), and others, should be part of the laboratory workup [2]. Blood and sputum cultures should be included in the microbiologic workup. Serology testing for fungal and atypical organisms, as well as influenza screening, are significant. A urine antigen test can identify the unusual organism Legionella. To rule out diffuse alveolar hemorrhage (DAH), eosinophilia, or malignant infiltrates, bronchoscopy with Broncho alveolar lavage (BAL) is required [3]. BAL findings for acute interstitial pneumonia frequently show neutrophil and sporadic atypical type II pneumocystis. Bronchoscopy also assists in obtaining respiratory samples for culture when there isn't sputum production. The piece must be sent for Pneumocystis jirovecii immunofluorescence, cultures, cell count, and cytology [4].

A lung biopsy may be required if the previous workup is ineffective in producing a different diagnosis. The results of a lung biopsy may also show other etiologies, such as viral inclusion, necrosis, abscesses, non-caseation granulomas, or tuberculosis. The condition known as acute interstitial pneumonia is idiopathic. To make the diagnosis, pathologic proof of idiopathic DAD is required after ruling out other causes of ARDS [4].

Hemostasis is a cycle that forestalls and quits dying. Therefore, keeping blood inside a harmed vein is significant. The common hemostatic framework includes four sections: vasculature, platelets, coagulation factors, and fibrin lytic proteins. A deformity in these compartments can result in coagulopathy [1].

Coagulopathy can be named essential or optional. Vital hemostasis issue remembers an imperfection for veins or platelets. Though, auxiliary problems include subjective or quantitative deformities in thickening variables or their inhibitors. Inhibitors can be anticoagulants, explicit variables, direct thrombin, or vague inhibitors [2]. Then again, obtained coagulation jumble is primarily connected with persistent illnesses like liver sickness, vitamin K inadequacy, spread intravascular coagulation (DIC), and anticoagulant treatment. To evaluate these issues, research center tests like total blood count (CBC), enacted thromboplastin time (aPTT), Prothrombin Time (PT) halfway, and blending trial is requested to examine an imperfection in platelet number, factor lack, and presence of coursing coagulation factor inhibitors [5].

In a medical illness known as coagulopathy, blood coagulation is disrupted, which increases the risk of bleeding from blood vessels that have been injured. The body uses coagulation as a defense mechanism to stop excessive bleeding or clotting from minor wounds. Impairment in clot formation is the usual term for it [6].

Coagulation stops spontaneous bleeding from wounds, scrapes, burns, bruises, and even internal injuries when paired with blood platelets [7].

Coagulopathy is a term used to depict a state of disabled blood coagulation. The reason for this condition can change. However, these all share, practically speaking, exhaustion of thickening variables and an easing back of coagulating time, bringing about a body that can't quit draining accurately. More terrible, however, since coagulopathy will generally be flighty, it can cause monstrous draining that could prompt passing whenever left untreated [8].

Coagulopathy can be a hazardous issue. Albeit the seriousness of this condition differs from one individual to another, a severe ailment requires prompt consideration. It's ideal to have a drawn-out arrangement instead of controlling the side effects for the present moment or brief help. A few inhibitors typically exert inhibitory control over the coagulation interaction, limiting the coagulation development and preventing the spread of blood clots. However, this delicate equilibrium is upset when the procoagulant action of the coagulation factors is increased, or the mobility of naturally occurring inhibitors is diminished. A perioperative doctor should grasp the complexities of two frameworks (all the more so in a previous hematological issue) that go next to each other in keeping up with the flowing blood in a fluidic state.

Neurotic circumstances requiring a medical procedure, sedation, or other intrusive strategy trigger the hemostatic framework. This equilibrium is additionally upset by injury, cytokines, or irresistible specialists. The risk of hemorrhagic and prothrombotic anomalies is high throughout the perioperative period. Hypoxia, hypothermia, metabolic acidosis, and the extracorporeal course could all interfere with what happens.

Coagulation in the intensives may also be brought on by physiological aggravations, variables affecting critical hemostasis, blood or plasma anomalies, or scattered intravascular coagulation (DIC). Following vascular injury, platelet surface receptors G*pIb* and endothelium collagen are continued by vWf, which encourages platelet adherence. The platelet glycoprotein complex I serve as the primary vWF receptor (*GP-Ib*). Additional platelet accumulation is stimulated by the thromboxane A2 that triggers platelet release. This platelet total is developed by TxA2 and ADP, which causes the platelet plug to form and temporarily stop vascular damage. ADP restriction also causes the *GpIIb/IIIa* receptors on the surface of platelets to change conformation, which causes a fibrinogen statement. The elective hemostasis process, which converts this fibrinogen to fibrin by catalyzing thrombin aging, increases the platelet plug's stability [3].

Prostacyclin inhibits platelet accumulation (platelet against amassing impact), and the interaction of TxA2 and prostacyclin results in constrained platelet accumulation, which prevents the coagulation from spreading and maintains the patency of the vessel lumen. Indicators of proteolytic agents known as zymogens that flow in a lazy structure make up a more significant portion of coagulating components. The letter "A" is appended to the Roman numeral indicating each zymogen, signifying that zymogen's beginning. Except for components III, components IV, and components VIII, the liver

manufactures the majority of coagulants and anticoagulants. The post-translational change these proteins go through enables them to bind calcium and other divalent cations and participate in coagulating overflow (vitamin K Ward Y carboxylation of destructive glutamic build-ups). A deficiency brings on anticoagulation of vitamin K or the presence of vitamin K opponents (warfarin) [9,10].

CONCLUSION

Even though we could not do much to help AIP patients survive, there were some encouraging developments. These included the use of evidence-based medicine in developing recommendations for disease management, the rapid development of lung transplants in treating severe AIP patients, the well-established lung rehabilitation, and the various molecular biomarkers of IIP used to identify the diagnosis, predict susceptibility, prognosis, and drug effectiveness. Unfortunately, however, these enormous efforts in the field of AIP are insufficient and outside the scope of any one center.

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