Short Communication
Volume 11:6, 2022

Journal of Trauma & Treatment

ISSN: 2167-1222 Open Access

An Injured 15-year-old Patient's Chest Pain and Syncope

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Introduction

After an inner-tubing accident, fifteen-year-old Drek arrived at the emergency room at around 1:50 PM. While sledding down a steep slope, Drek lost control of his inner tube, and his back collided with a tree. He did not experience vomiting or loss of consciousness, but he did experience pain in his neck and right arm. He arrived with a cervical collar, spinal immobilisation, and a full-length vacuum splint for his right arm. His right upper arm deformity was revealed to be a fracture of the right clavicle by x-ray films. A neck injury was ruled out after the cervical collar was removed and he showed no signs of soreness or pain when moving his neck. Upon urinalysis, no blood was found. Initial vital signs were as follows: 37.1°C for the body temperature; 119 for the heartbeat; 26 for the respiratory rate; and 120 for blood pressure [1,2].

He received 4 mg of morphine through an IV line placed in his left hand at 2:20 PM, roughly one and a half hours after his admission. A pressure balloon was utilised to quicken the IV infusion of lactated Ringer's solution about 2 hours after arrival, at 4:10 PM, so Drek could leave. After receiving the lactated Ringer's solution infusion around 3 hours later, at 5:15 PM, Drek reported experiencing chest pain, turned pale and dehydrated, and experienced a syncopal episode [3]. His vital signs were as follows: 109 beats per minute for his pulse, 110/80 for blood pressure, and 26 breaths per minute for his respiratory rate. After receiving oxygen through mask, the symptoms appeared to go away. What has become of Drek? We soon thought of five options.

Description

Hyperventilation may trigger syncope. However, according to Drek's mothis, he wasn't hyperventilating before to the syncopal episode, and no nurses saw Drek do so before the onset of symptoms. The syncopal episode can be a sign of anaphylaxis-related blood pressure drop. An allergic reaction, however, typically happens more quickly, and symptoms last even after oxygen treatment [4]. None of the typical signs of allergic reactions, such as a rash, wheezing, or shortness of breath, were evident [3].

Drek's chest pain can be a sign of a developing cardiac contusion. The fact that his symptoms disappeared after receiving oxygen is incompatible with heart contusion. Since Drek's damage was to his back rathis than his chest, a cardiac contusion was improbable. A cardiac contusion typically comes from forceful chest trauma [5].

A pneumothorax may be the cause of Drek's chest pain, diaphoresis, and pallor. He had no tracheal deviation, no diminihed breath sounds, and his symptoms went away after receiving oxygen. Pneumothorax was thisefore unlikely. Drek's chest discomfort, pallor, diaphoresis, and syncope were all signs of an air embolus. Furthis examination revealed that Drek had an air embolus due to his abrupt onset chest pain, pallor, diaphoresis, and still-attached pressure bag in his IV bag. Thise was also air in the line leading to the hub.

For an air embolus to be lethal in an adult, it needs about 100 ml of air, although even less air is needed to start showing symptoms. This appears to be a significant amount of air. However, when a pressure bag is utilised, a 14-gauge IV line will deliver 100 cc of air per second. 1 Even less air would be needed in the paediatric or baby population to produce symptoms and result in mortality.

An estimated 50% of those who experience venous air embolism die. The "aggregation of platelets, red blood cells, and fat globules" that occurs when air enters the venous circulation effectively starts the clotting cascade. I The air itself has the potential to block blood flow in the pulmonary arteries. Air moves to the pulmonary valve while the patient is seated or standing because air rises [6]. The blood may not be able to reach the lungs as a result, returning to the periphisal circulation with less oxygen. Additionally, this makes the right side of the heart work harder and causes blood to back up in circulation, all of which can result in congestive heart failure. Pulmonary edoema is brought on by the pulmonary obstruction, which increases pulmonary vascular resistance.

The majority of venous air embolism cases in the literature research were linked to eithis surgical procedures or central venous access devices. However, thise is a small but present danger of venous air embolism whenever a vein is accessed. Normal venous pressure typically prevents air from IV tubing from entering the veins, but when a pressure bag is utilised, the outside pressure is highis than the venous pressure.

The danger of venous air embolism exists for any patient who has a periphisal line implanted, as Drek did. Utilizing a pressure bag may make this danger worse. If a central line is thought to be the source of the embolus, the nurse needs to look for any breaks or cracks in the line and clamp it close to the break. As previously mentioned, the symptoms should then be addressed until the patient's condition is stable. As soon as feasible, the incidence should be reported to the surgeon to let him or his know about it.

Conclusion

Our emergency department establihed a protocol on the use of IV pressure bags as a result of Drek's experience. After receiving oxygen, Drek's symptoms appeared to improve. Despite the fact that doing so could have been beneficial, he was not positioned in the Trendelenburg or left side-lying positions. But Drek's nausea and pallor when standing persisted as he got ready to be discharged. He was admitted, and during the night, his health was checked for any recurrence of dyspnea, pallor, reduced blood pressure, dizziness, or continuing nausea. The following day, he was released with no furthis issues.

Acknowledgements

Not applicable.

Conflicts of interest

The authors declare that they have no competing interests.

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Date of Submission: 04 June, 2022, Manuscript No. JTM-22-70116; **Editor assigned:** 06 June, 2022, PreQC No. P-70116; **Reviewed:** 18 June, 2022, QC No. Q-70116; **Revised:** 23 June, 2022, Manuscript No. R-70116; **Published:** 29 June, 2022, DOI: 10.37421/2167-1222.2022.11.516

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How to cite this article: Anderson, Jessica. “An Injured 15-year-old Patient's Chest Pain and Syncope.” *J Trauma Treat* 11 (2022): 516.