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Impending cardiogenic shock due to tricyclic antidepressant toxicity, computed tomography findings; a case report

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ABSTRACT

A computed tomographic (CT) with contrast enhancement is extensively used for assessment of a wide range of thoracic and abdominal circumstances. Thus, as the use of CT in the evaluation of unstable patients increases, the chance to see the CT features of imminent cardiogenic shock and cardiac arrest also increases during scanning. The patient was a 27 year old man who brought to the emergency department by paramedics unresponsive, After physical examination and obtaining ECG due to presence of hypotension ,with suspicion to traumatic injury, Thoracoabdominal CT was done for patient which show dense opacification of the right hepatic lobe, that resembling contrast extravasations from IVC and the hepatic vein, laparatmy was done for patient which were normal .Patient Has developed cardiac arrest and died at the end of surgery. Postmortem autopsy and toxicology revealed Tricyclic antidepressant intoxication as a cause of cardiac arrest and death. TCA poisoned Patients may have normal ECG findings on arrival. In spite of infrequent reports of cardiogenic shock that occurring during CT scan, knowledge of distinctive CT findings of these patients is very essential for precise analysis of images, in addition to informing clinical physician for malpractice avoidance and immediate initiation of resuscitation.

Key word: Computed Tomography (CT); venous pooling; cardiogenic shock; TCA poisoning

INTRODUCTION

Cardiogenic shock is defined as a low systemic cardiac output because of decreased systemic cardiac output due to pump collapse. Although the occurrence of impending cardiogenic shock throughout computed tomographic (CT) examination is unidentified, indubitably it is not a frequent event. To the best of our knowledge, there are few cases in which findings of CT are reported in the literature[1-8]. Here we report a case of impending cardiogenic shock which misinterpretation of his CT scan leading to unnecessary exploratory laparotomy.

CASE

A 27 year old man was brought to the emergency department by paramedics after the patient's brother found him down stairs at home unresponsive. There was no identified history of prior disease or drug abuse .Physical examination revealed a heart rate of 132 bpm, the systolic blood pressure of 75 mmHg, respiratory rate of 21 breaths/min, temperature of 36.5c, and a pulse oximetry of 100% on a mask. Blood sugar with glucometer was 120 mg/dL. ECG revealed sinus tachycardia and otherwise 'normal' (QRS 95 ms).Neurologic examination showed reduced gag reflex, patient did not reply to auditory stimuli, withdrawal from painful stimulus in all 4 limbs, 2+ reflexes, negative Babinski's sign, and a 6 mm pupils with slow reactivity, in examination periumbilical skin bruising was seen. Intravenous access was established, blood was sent to the laboratory for analysis (complete blood count, renal panel, cardiac biomarkers, liver panel, lactate level, blood cultures, ammonia level), at once maximum naloxone dose was administered for patient but no response was seen. Patient's systolic blood pressure reached to 100 mmHg, after administration of 2 liter normal saline, patient open his eyes in response to voice and complain of

abdominal pain but level of consciousness deteriorated quickly again. Patient electively intubated for airway protection. With a suspicion for traumatic injury, FAST ultrasound was done which were negative. Brain computed tomography (CT) scan were normal. Contrast enhanced thoracic and abdominopelvic CT was ordered which shows layering of contrast medium in dependent portions of the venous system, dense opacification of the right hepatic lobe and accumulation of contrast agent in posterior segments of the right hepatic lobe and IVC that resembling contrast extravasations from IVC and the hepatic veins (Fig 1).

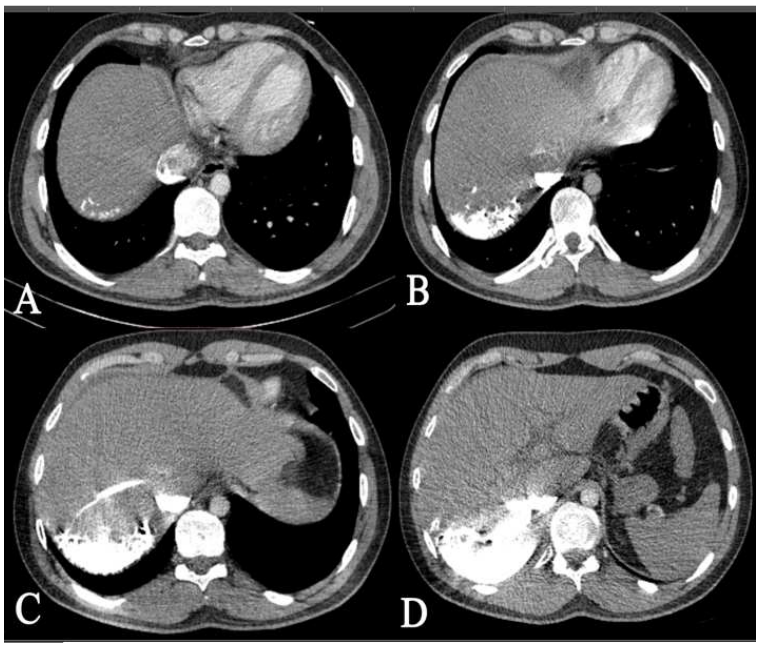

Figure1. Enhanced CT scan shows venous layering of contrast medium in dependant portions of venous system and Regurgitation of contrast medium into IVC and hepatic veins(A,B) also noted densely opacified parenchyma in posterior segment of the right hepatic lobe (C, D)

Patient transferred to the operating room with an impression of hepatic and IVC trauma(Rupture); exploratory laparotomy performed but IVC, hepatic vein and other abdominal organ were normal, at the end of the surgery sudden cardiac arrest occurs; Cardiopulmonary resuscitation performed immediately for 1hour but patient didn't respond to CPR and unfortunately the patient died. Postmortem autopsy and toxicology revealed Tricyclic antidepressant intoxication as a cause of cardiac arrest and death.

DISCUSSION AND CONCLUSION

TCA medications are administered for the treatment of many psychiatric diseases and remain a frequent reason of fatal drug poisoning. Knowledge of the clinical features of TCA overdose is essential to accelerate diagnosis and start the proper treatment. The toxic effects of TCAs are caused by four major pharmacological mechanisms, including: direct alpha-adrenergic Blockage, inhibition of norepinephrine reuptake at nerve terminals, quinidine like effect on the myocardium and anticholinergic effect. Incidence of arrhythmia and hypotension are evidence of cardiovascular toxicity. It frequently causes R in aVR of 3 mm or more, prolongation of QT interval and Torsadede pointes, tachycardia or bradycardia. Hypotension possibly caused by decreased myocardial contractility and reduced systemic vascular resistance may be caused by alpha-adrenergic blockade [10,11].In this case despite the TCA poisoning, the primary QT interval and R in aVR was normal.

Imaging a patient during Hypotension and imminent cardiogenic shock is not a regular event, but as the use of CT in the evaluation of hemodynamically unstable patients increases, the opportunity to observe the CT features of these conditions also increases.

Contrast agent, is heavier than blood, and when cardiac pump failure happens accumulate in dependent part of the venous system, CT scan findings of dependent venous pooling of contrast material after death or in cases of acute cardiac arrest in the CT scanner have been described previously in a few case reports in the English literature [1-4]. CTfindings during impending cardiac arrest or hypotensive cardiogenic shock include: dependent venous pooling of contrast material, layering of contrast medium in dependent portions of venous system, dense opacification of the right lobe of the liver, Regurgitation of contrast medium into IVC, hepatic veins, Renal vein and, decreased

enhancement of the abdominal organ, contrast pooling in dependent lungs, and contrast stasis in pulmonary veins seen in contrast-enhanced CT imaging [1-8].

Our case was alive during the study but misinterpretation of his CT scan leading to unnecessary exploratory laparatomy. The contrast layering in IVC and hepatic veins along with dense opacification of posterior segments of right liver lobe was a marker of imminent hypotensive cardiogenic shock in this case. Similar cases have been reported previously in the literature[5-8],but normal ECG finding and lack of familiarity of emergency team physicians with CT findings causes misdiagnosis and unnecessary exploratory laparatomy.

These CT findings hold a poor prognosis for the patient [7,8]. Although the entire reported cases in literature, died shortly after this CT scan findings [1-8], the managing physicians must be notified quickly to establish the rapid treatment since these findings indicate cardiac collapse and impending severe cardiogenic shock and cardiopulmonary resuscitation be supposed to be prepared rapidly.

Conflict of interest

The authors declare that they have no conflict of interest

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