Alotaibi Saudi J Er Med. 2022;3(2):171-175
https://doi.org/10.24911/SJEMed/72-1633272475

CASE REPORT

Recurrent cardiac arrest in emergency department secondary to abdominal compartment syndrome with a recent surgical intervention: a case report

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ABSTRACT

Background: Abdominal compartment syndrome (ACS) refers to multi-organ dysfunction caused by intra-abdominal hypertension. A devastating condition that can lead to a 100% mortality if not treated. ACS is often underrecognized in the emergency department (ED) because it mostly affects patients who are critically ill and admitted in intensive or surgical care units.

Case Report: In this paper, we report a case of a young patient with a history of recent surgical intervention but no past medical history who presented with recurrent cardiac arrest that failed to respond to aggressive management. It was initially unrecognized, but later ACS was considered in the differential and the patient was treated accordingly and eventually revived and had good neurological outcome.

Conclusion: ACS is a rare but critical diagnosis that might be under recognized in the ED. With high rates of ED turnover, it is essential for ED clinicians to be aware of this disease and have a high index of suspicion about it.

Keywords: ROSC, gastric sleeve surgery, intra-abdominal hypertension, laparotomy, post sleeve complications.

Introduction

Arrival of patients with cardiac arrest is a terrifying but familiar scene witnessed on daily bases at the Emergency Departments (ED). Patients' survival depends on immediate resuscitation, early identification of underlying pathology, and prioritizing critical interventions with survival benefits in cardiac arrest [1]. Findings on the initial rhythm are the most useful aid to identify the underlying etiology. Cardiac causes are usually suggested by ventricular tachycardia or fibrillation while asystole and pulseless electrical activity (PEA) usually indicate severe shock and constitute a wider differential [1].

History, physical examination, and bedside diagnostic testing are the main tools that help in the differential formulation [1]. History of a recent surgical procedure is a critical clue that have significant impact on recognizing specific differentials of the cardiac arrest.

Our aim in this paper is to describe a case of abdominal compartment syndrome (ACS), a rare surgical complication, that presented as a recurrent cardiac arrest in the ED, requiring special interventions, and at the same time it mimics other differentials whose management might have led to catastrophic outcome if not recognized.

Case Presentation

A 37-year-old Arabic female not known to have any medical or psychiatric illness brought by Emergency Medical Services due to sudden loss of consciousness. The patient was found unconscious while waiting for her post-operative appointment in the clinic. She had sleeve gastrectomy and nissen fundoplication 14 days prior.

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Received: 03 October 2021 | **Accepted:** 26 February 2022

Upon arrival to our ED, she looked pale, tachypneic, unresponsive, with unrecordable blood pressure, tachycardiac (HR 160), hypoxic despite supplemental oxygen applied at triage point (Saturating 80%), afebrile (T37.4), and had normal initial capillary glucose (6 mmol/l). Right after placing her on the resuscitation bed, patient was gasping for air with no palpable pulses. Cardiopulmonary resuscitation (CPR) was started according to the Advanced Cardiac Life Support Algorithms for PEA and two cubital intravenous (IV) lines were established.

A quick discussion with the mother revealed history of intermittent abdominal pain, malaise, feeling unwell, and decrease oral intake since the operation. There was no history of fever, respiratory symptoms, chest pain, overdose or pregnancy. The patient had multiple ED visits in different hospitals including our hospital. She was investigated with a Computed Tomography (CT) scan of the abdomen with IV contrast twice, one of them was at our hospital 5 days prior to the indexed visit, and she was assured after the results were negative for post-operative complications.

Quick examination during CPR cycles showed a morbidly obese female, tense abdomen, clean laparoscopic scars, and bilateral swelling of the lower limbs with extensive bluish to purplish discoloration. Given the clinical presentation, the resuscitation team considered bilateral phlegmasia cerulea dolens (PCD) and massive pulmonary embolism (PE) as the cause of cardiopulmonary arrest. After discussion with the family, the decision was made to give tissue plasminogen activator (tPA) despite the high risk of bleeding after the recent surgery. However, the patient achieved return of spontaneous circulation (ROSC) after nine cycles of CPR with persistent PEA and tPA was held. During CPR, the patient was intubated, received anti-hyperkalemic and hypoglycemia treatment when the reading of the first venous blood gas (VBG) was received as shown in Table 1. Post-ROSC, she remained persistently hypotensive (BP of 80/40 and a MAP 53 mmHg), tachycardiac (HR 120), and hypoxic (Saturating 88%) on 100% fraction of inspired oxygen. An ultrasound (US) guided central catheter was placed in the right femoral vein then a normal saline and norepinephrine infusion were started. Of notice, the swelling and discoloration on the right lower limb was more pronounced after IV fluid initiation with notable resistant to fluid flow through the cannula. During further resuscitation, patient arrested again twice, CPR was started with PEA rhythm for two cycles each until ROSC was achieved.

Initial bedside US was performed in effort to detect reversible causes of PEA. The left ventricle was hyperdynamic, with no signs of Right Ventricle (RV) strain or pericardial tamponade, and no signs of pneumothorax or hemothorax. The Inferior Vena Cava (IVC) was difficult to visualize. The lower limbs femoral and popliteal veins were compressible but with significant pressure. However, there was a large amount of hypoechoic free fluid in the abdomen that was difficult to detect (Figure 1). US however helped us differentiate free fluid from the solid and fixed liver by showing movement of the fluid within the peritoneal space with gentle shaking of the rigid abdomen.

Post-ROSC VBG showed pH of 7.01, HCO_3 18.8 mmol/l, PCO_2 75.8 mmHg, Lactate 10.17 mmol/l despite a high ventilatory settings of Respiratory Rate (RR) of 33 b/minute, TV 400 ml per inspiration, Positive end-expiratory pressure (PEEP) of 5 mmHg, and initial FiO_2 of 100% (Table 1). She had resistant hypoxia and hypercapnia despite a confirmed Endotracheal (ET) tube placement and the absence of pneumothorax, hemothorax or visible pneumonia. Chest X-ray showed bilateral diaphragmatic elevation (Figure 2). Initial lab results came back as shown in Table 2.

While keeping massive PE at the top of the differentials list, the team also considered intra-abdominal sepsis, small bowel obstruction, pneumonia, severe dehydration, and portal vein thrombosis. However, after gathering the clues from the initial assessment, bedside US and chest X-ray findings, a new differential came into sight and made a better and more logical explanation. The patient had tense abdomen, free fluid accumulating in all quadrants, was hypotensive with no improvement after

Table 1. *VBGs readings.*

	First sample during CPR	Post-ROSC
pH	7.012	7.017
PCO_2	75.8 mmHg	65.5 mmHg
PO_2	34.2 mmHg	35.7 mmHg
Total Hb	12.13 g/dl	11.71 g/dl
COHb	1.3%	1.5%
K^+	6.26 mmol/l	6.94 mmol/l
N^+	138.5 mmol/l	132.8 mmol/l
Ca^{2+}	1.942 mmol/l	2.049 mmol/l
Cl^-	98.9 mmol/l	99.2 mmol/l
Glu	1.58 mmol/l	13.39 mmol/l
Base excess	-13.1 mmol/l	-14.8 mmol/l
HCO_3^-	18.8 mmol/l	16.4 mmol/l
Lactate	10.96 mmol/l	10.17 mmol/l

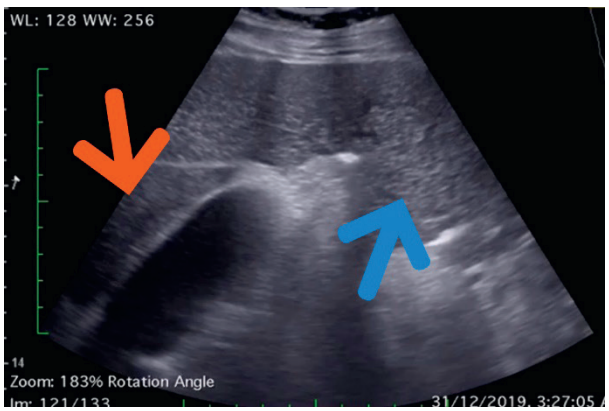

Figure 1. *A right upper quadrant US examination showing free fluid (Blue arrow) iso-dense to liver parenchyma (Red arrow).*

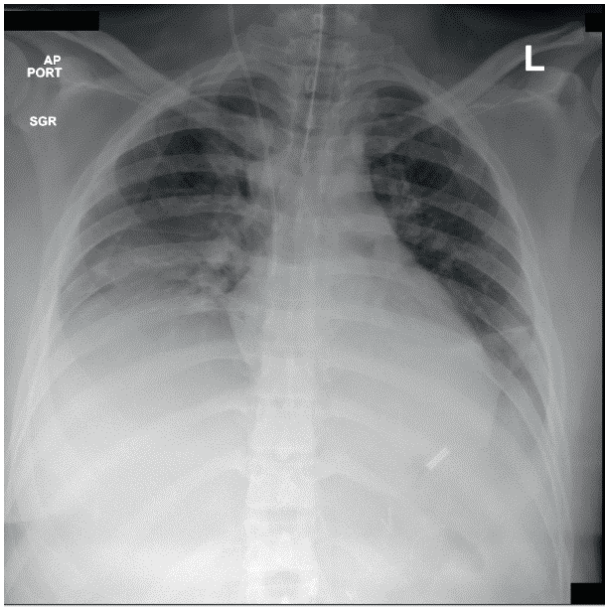

Figure 2. *A chest X-ray after intubation and Rt internal jugular vein central line showing bilateral diaphragmatic elevation.*

Table 2. *Initial lab test results.*

Test	Result	Lab range (Unit)
White Blood Cell Count (WBC)	19.4	4.0-11.0 (10^9/l)
Red Blood Cell Count (RBC)	4.28	3.80-5.80 (10^{12})
Hemoglobin (HGB)	11.8	11.5-16.5 (g/dl)
Platelets (PLT)	455	150-450 (10^9/l)
Absolute neutrophils	17.0	1.8-7.5 (10^9/l)
Urea	25.1	2.8-8.1 (mmol/l)
Creatinine	616	45-84 (mmol/l)
Total bilirubin	30	2-21 (mmol/l)
Alanine transaminase	1,386	5-33 (U/l)
Aspartate aminotransferase	N/A	2-21 (U/l)
Alkaline phosphatase	131	35-104 (U/l)
Prothrombin Time (PT)	21.8	11.5-14.5 (seconds)
Activated Partial Thromboplastin Time (APTT)	62	30-41 (seconds)
International Normalized Ratio (INR)	1.8	0.9-1.3
Human Chorionic Gonadotropin (HCG)	<0.1	Non pregnant ≤1 (mIU/ml)

IV fluid and pressors initiation from the femoral line but instead the swelling and discoloration was getting worse with marked resistant of flow. She also had bilaterally elevated hemi-diaphragms on chest X-ray (Figure 2) with resistant type II respiratory failure despite all our efforts. These could be explained by surgical site leakage causing ACS.

After considering ACS, the surgical team were called immediately, and her primary surgeon was at the bedside. Foley catheter and nasogastric tube were inserted to decompress the abdomen however, she had no urine output. A right internal jugular catheter was inserted to bypass the resistance of venous return passing through the abdomen. After instant discussion with the primary surgeon, a decision to decompress the abdomen immediately in our ED resuscitation room was reached. A small incision to open one of the laparoscopic ports in the left upper quadrant was made to allow the passage of a blunt Foley catheter into the abdominal cavity. After the tip was introduced, a gush of dense brownish fluid backflow came promptly draining more than 3,500 ml within 30 minutes. Shortly after decompression, the abdomen became soft, the blood pressure improved (110/69 mmHg) and MAP of 82 mmHg was achieved, saturation improved to 90%, and the bilateral lower limbs venous engorgement and discoloration diminished remarkably. After stabilization, the patient was shifted to the operating room for exploration and abdominal washout. Intraoperative finding confirmed free fluid in the abdomen with small perforation lateral to the sleeve stapler line. An abdominal lavage was done, and the perforation was sutured. Afterword, an intraoperative gastrografin study showed no leak and the abdomen was closed, with multiple drains left.

The patient was shifted to intensive care unit (ICU) and had a lengthy stay where she recovered from renal failure requiring temporary renal replacement therapy, treated with antibiotics and antifungals for intra-abdominal infection after the drained fluid resulted in a positive candida culture, and experienced an episode of bradycardia requiring temporary cardiac pacing. After 30 days of ICU stay, the patient was successfully extubated and transferred to a regular room. One month later, she was discharged on room air with Glasgow Coma Scale Score of 15/15. A fallow up with the patient after 6 months showed that she lives with good neurological outcome and functioning normally with no disabilities.

Discussion

In this paper, we report a unique and rare case of recurrent cardiac arrest due to ACS that emergency physicians should consider when caring for a critically ill patient with surgical procedure history. A great emphasis should be placed to explore all the possible causes to patients' deterioration with the best utilization of available tools before making life-saving diagnostic and therapeutic decisions.

ACS is defined as a "sustained intra-abdominal pressure (IAP) >20 mmHg that is associated with new organ dysfunction/failure" [2]. The mechanism underlying the development of multisystem dysfunction in ACS may be related to several factors. The accumulation of fluid in the abdominal cavity in addition to the pre-existing layers of fatty tissue from the morbid obesity contributed to the rise in the IAP [2,3]. Such high pressure would cause damage to multiple organs by compressing intra-abdominal vessels including renal, hepatic and the IVC. Subsequently, it will cause reduction in preload and interruption of venous return due to fluid sequestration in the lower limbs, leading to hypovolemic shock and cardiovascular collapse [2,3]. Furthermore, the increase in the IAP and subsequent decline in cardiac output will lead to a reduction in hepatic and renal blood flow and decrease urine output as well as impermeant

of respiratory and central nervous system [2-4]. Of note, the increasing pressure on the diaphragm will decrease pulmonary compliance and restricted lung expansion, causing respiratory failure [2,3]. Evidence also suggest a close relationship between the IAP and intracranial pressure, with some investigators reporting that increased IAP would translate into increased central venous pressure that can subsequently cause elevated intracranial pressure [5]. ACS effect on renal, hepatic, cardiopulmonary, and central nervous system will lead to organ dysfunction and death with a mortality of 100% if not treated [3]. ACS was first recognized by Kron et al. [4] and has been gaining attention in ICU and after damage control surgeries. A previous study indicated 50% accuracy of ICU physicians' identification of increased intraabdominal hypertension (NOT compartment) by examination alone [3].

Primary ACS is referred to direct injury to the abdominopelvic organs such as in trauma casing splenic rupture, rapture of abdominal aortic aneurysm, acute pancreatitis leading to third space expansion and acute rise in abdominal pressure similar to our case with rapid accumulation of leaking fluid that wasn't evidence in her recent CT prior to ED presentation, not to mention her preexisting morbid obesity causing additional external pressure on the abdomen exceeding her body's physiologic ability to compensate [1]. Additionally, Massive ascites has been reported although many can tolerate large volumes if accumulation is gradual [6]. Secondary ACS is linked to excessive fluid resuscitation and subsequent sequestration in the intestinal space even without abdominopelvic pathology. It usually originates in the ED with massive resuscitation of trauma, critically ill, and burn patients but later discovered at ICU after patients' deterioration [1].

Prevention is the mainstay of treatment of ACS with early anticipation in high-risk patients. If it cannot be prevented, early recognition and abdominal decompression using medical, conservative and surgical options are vital [1].

The incidence of ACS in ED is unknown, and the clinical picture of patients presenting with ACS to ED is still not well defined [6]. Data about ED staff awareness of ACS is absent which makes it difficult to recognize and might delay its management [6]. Patients presenting with late-stage ACS, like in our case with cardiac arrest, might be mistaken for other diagnosis in a situation where time is critical and rapid intervention is needed. We presumed a diagnosis of pulmonary embolism due to patient history of recent surgery with lower limbs findings resembling bilateral PCD. We intended to give tPA but, mercifully, the patient achieved ROSC and we had time to consider ACS as a differential diagnosis and act accordingly although we did not have the equipment and the time to measure the IAP. We advocated for ACS diagnosis in our patient from all the clues gathered that were supported by the dramatic improvement of her condition after our ACS targeted interventions.

Conclusion

We reported a case of ACS which is a rare, life-threatening presentation in ED. It should be considered in any critically ill patients with a possibility of increase IAP with acute organ dysfunction. It also highlighted the importance of increasing the awareness of ACS among ED physicians and having a high index of suspicion with the utilization of available diagnostic tools to promptly recognize it, especially in critical cases where quick interventions are lifesaving.

List of Abbreviations

ACS	Abdominal compartment syndrome
CPR	Cardiopulmonary resuscitation
ED	Emergency department
IAP	Intra-abdominal pressure
ICU	Intensive care unit
IV	Intravenous
PCD	Phlegmasia cerulea dolens
PEA	Pulseless electrical activity
ROSC	Return of spontaneous circulation
tPA	Tissue plasminogen activator
US	Ultrasound

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Funding

None.

Consent for publication

Written informed consent was obtained from the patient after her discharge from the hospital by her primary ER physician to publish this case report and any accompanying images.

Ethical approval

Ethical Approval is not required in our institution for an anonymous case report.

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References

1. Littmann L, Bustin DJ, Haley MW. A simplified and structured teaching tool for the evaluation and management of pulseless electrical activity. Med Princ Pract. 2014;23(1):1–6. https://doi.org/10.1159/000354195
2. Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med. 2013;39(7):1190–206. https://doi.org/10.1007/s00134-013-2906-z
3. Hecker A, Hecker B, Hecker M, Riedel JG, Weigand MA, Padberg W. Acute abdominal compartment syndrome:

current diagnostic and therapeutic options. Langenbecks Arch Surg. 2016;401(1):15–24. https://doi.org/10.1007/s00423-015-1353-4

4. Harman PK, Kron IL, McLachlan HD, Freedlender AE, Nolan SP. Elevated intra-abdominal pressure and renal function. Ann Surg. 1982;196(5):594–7. https://doi.org/10.1097/00000658-198211000-00015

5. Lauerman MH, Stein DM. Multicompartment management of patients with severe traumatic brain injury. Curr Opin Anesthesiol. 2014;27:219–24. https://doi.org/10.1097/ACO.0000000000000044.

6. Harrisson SE, Smith JE, Lambert AW, Midwinter MJ. Abdominal compartment syndrome: an emergency department perspective. Emerg Med J. 2008;25(3):128–32. https://doi.org/10.1136/emj.2007.050344