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Pulmonary emboli cardiac arrest with CPR complication: Liver laceration and massive abdominal bleed, a case report

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ABSTRACT

INTRODUCTION: Massive pulmonary emboli may cause right ventricular failure and backward stasis with parenchymal organ swelling thus increasing the risk for laceration, e.g. if CPR is needed.
PRESENTATION OF CASE: A 28-year-old Colombian female with no medical history but taking contraceptive pills and having had a recent longer flight was admitted to Danderyds hospital Emergency Department because of respiratory failure. She developed cardiac arrest in the emergence department following the emergent diagnosis of pulmonary emboli. Cardio-pulmonary resuscitation was initiated, initially with manual and subsequent mechanical compressions with a so called LUCAS device. Patients did not respond properly to the CPR and showed signs of hypovolemia. Emergent ultrasound raised suspicion of abdominal bleed. Emergent laparotomy confirmed liver laceration and massive haemorrhage.
CONCLUSION: Pulmonary emboli with subsequent right ventricular failure may cause backwards stasis, and parenchymal organ e.g. liver enlargement. The risk for laceration injuries and internal bleed must be acknowledged when applying external forces as in case of cardiac arrest and need for resuscitation. Frequent and vigilant control of positioning of manual as well as mechanical compressions is of importance.

1. Introduction

Contraceptive-pills and immobilisation such as during long air flight are known risk factors for the development of pulmonary emboli. Initial thrombotic symptoms may be discrete and thus cause patient delay. We describe a case of pulmonary emboli with a complicated course and fatal outcome.

2. Case presentation

A previously healthy 28-year-old Colombian female taking contraceptive pills with a recent longer flight was admitted to the Emergency Department because respiratory failure. Her medical history involved six days of progressive tachypnea, diffuse chest and back pain, escalating into severe respiratory failure. There was no history of trauma. At the arrival by ambulance in the emergency room she had a saturation of 80% breathing room air, a breathing rate of 50 breaths per minute and pulse hard to palpable. She was awake but unable to speak in full sentence due to respiratory distress. An electrocardiogram (ECG) that was sent in to the emergency department (ED) from the ambulance showed sinus tachycardia at a rate of 147 per minute, and T-wave inversion in anterior precordial leads and incomplete right bundle-branch block. A focus assessed transthoracic echo (FATE) and ultrasound abdomen showed liver venous stasis but no other sign of right ventricular failure and no signs of fluid in the right upper quadrant. The patient did receive a bolus of i.v. 5000 E Heparin on the preliminary diagnosis of pulmonary emboli. The patient suddenly went into cardiopulmonary arrest with pulse-less electrical activity (PEA) shortly after arrival in the emergency room. Cardiopulmonary resuscitation (CPR) was immediately instituted according to advanced life support guidelines. Manual chest compression and artificial ventilation were performed but to relieve the emergency department staff from manual compressions the LUCAS chest compression system was applied and initiated. The LUCAS device was placed and strapped in accordance to the instructions for use, but the neck strap was loosened in order to gain a central venous access, an internal jugular vein puncture. The CPR was ineffective and in order to gain further information around the cause for the cardiac arrest and PEA and a second FATE and ultrasound abdomen was performed that couldn't reveal sign of right ventricular strain but were positive for fluid in the tight and left upper quadrant and also in the lower quadrant assessed as abdominal bleed. This finding initiated massive transfusion but still with no return of spontaneous circulation. A decision to perform an exploratory laparotomy was made and the patient was brought to the operating theatre under CPR white LUCAS. A massive bleeding due to a liver laceration was found. A 4-quadrant

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Table 1
Blood laboratory findings.

	At arrival	Postoperative	4 h after operation
CRP	105	11	13
Leucocytes	21	7	6
Procalcitonine		0,03	1,4
Hb	125		115
Platelets	218	79	79
Creatinine	115	115	140
Amylase	0,3	3	
INR	1,2	1,5	1,3
APTT	26	204	88
ASAT	0,5	41	55
ALAT	0,2	31	27
ALP	1	1	
Bilirubine	17	14	23
Fibrinogen		0,6	0,7
D-dimer			>10,5
LD		65	52
Myoglobine		6200	19000
CK		17	
Troponine	52	382	
NT-PRP BNP		9000	1100
NSE		56	
S-100B		5,3	13

packing and manual sub diaphragmatic aortic compression were performed.

The ECMO-team was called based on the inability to restore circulation. The ECMO-team arrived at the time for the explorative laparotomy and her right femoral vein and artery were surgically cannulated and connected to the ECMO-circuit. No additional bleeding or cardiac tamponade was found. The ECMO had adequate flow and the patients was transported to the ECMO-centre. Time from cardiac arrest to adequate ECMO flow was 1 h and 35 min. The blood tests at arrival and during the first 4 h are presented in Table 1.

Postoperative CT-scan of thorax showed a massive central PE that had almost totally occluded both pulmonary arteries. CT-scan of the brain 9 h after the cardiac arrest showed significant sign general brain swelling and life-threatening cerebral herniation. ECMO- and ICU-treatment were withdrawn 12 h after the cardiac arrest and the patient died shortly thereafter.

3. Discussion

Our patient had right after arrival in the emergency department cardiac arrest most certainly caused by massive PE. She received emergent dose of heparin directly, before the cardiac arrest. Cardiopulmonary resuscitation in accordance to the AHA Guidelines was promptly initiated. Compressions were initially done manual but subsequently with a LUCAS device. She develop massive bleeding from liver laceration. Our patient had extensive resuscitation efforts including, CPR, laparotomy to stop the liver bleeding, massive transfusion and extra-corporeal circulatory assist she still developed signs of severe brain ischemia and therapy was withdrawn 12 h later.

The suspicion of PE was raised early but initial emergent bedside ultra-sound wasn't fully confirmatory. According to European guidelines key-factors for suspecting PE are predisposing factors, symptoms and objective sign [1]. Our patient had predisposing factors for PE including contraceptive-pills and immobility due to sitting during longer flight. Her symptoms with dyspnoea and pleuritic chest pain are symptoms correlated with PE, syncope is infrequent seen and does not correlate with PE and none of these symptoms are specific for the PE diagnosis [2]. Our patient developed arterial hypotension and shock which is seen in the most severe cases and indicates central PE and/or a severely reduced hemodynamic reserve and a high PE-related early mortality risk [1].

Our patient showed signs of massive PE with an increase in pulmonary artery pressure and subsequent acute right ventricular failure (RVF). RVF also contributes to backward failure transmitted directly to the hepatic veins and sinusoids and leads to a passive congestion of the liver. In long term the stasis will also cause failure of the liver [3]. Liver enlargement is well-known in chronic RVF. There are no explicit studies, evidence that patient with liver enlargement, e.g. caused by backward failure of the right heart have a higher risk for liver laceration during CPR but the connection is theoretically appealing. There are case-reports of liver injury after CPR due to PE [4]. Joseph et al. described a liver bleed flowing prolonged resuscitation of a PE induced circulatory collapse [5]. CPR per see may in a low incidence cause abdominal bleed caused by laceration injury to the liver [6].

Liver injury associated to CPR, sternal compression is rare, occurring at a rate of 0.6%–2% [6–8]. In a multicentre study D, Smekal et al. did not see any difference in incidence of liver injuries between LUCAS compared to manual compression but they conclude that mechanical chest compressions adds 14–15% more patients with rib fractures [8]. Our patient had normal liver enzymes at admission and there was no obvious finding suggesting liver stasis, however the ultra-sound did show slightly except distended liver veins possibly related to right ventricular strain. We cannot explicitly state to what extent the backward failure and increased venous pressure and liver stasis could have contributed to the liver laceration and the massive bleeding. The imported factors for developing intra-abdominal complication during CPR seem to be the placement of the hands or the piston rather the aetiology to the cardiac arrest [8]. It should also be acknowledged that our patient did receive a loading dose of heparin right before going into cardiac arrest.

We cannot state whether the lacerations occurred during the manual or the mechanical compressions and we are unable to provide any firm suggestion how to avoid the risk for liver laceration during CPR in patients having a cardiac arrest cause by massive pulmonary emboli. Optimising the positioning of hands and any mechanical chest compression device is however reasonably of importance.

4. Conclusion

Pulmonary emboli compromising cardiac function with subsequent forward as well as backward failure creates parenchymal organ stasis increases the risk for laceration and internal bleeding caused by external forces. The risk for bleeding must not be forgotten taking care of massive pulmonary emboli. CPR during cardiac arrest caused by PE should be performed with vigilant positioning of the hand or the mechanical compression device.

Conflicts of interest

The authors have no conflict of interest in relation to the present Case Report.

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Ethical approval

The event had a fatal outcome and we have discussed the ethics in publishing with the head of the Institution for Clinical Science, head of Institution supports it publication without further consent.

The event has been reported to the Swedish Medicinal Product agency as a potential device incident.

Consent

Not applicable.

Authors' contribution

John Lundqvist**;** Data collection, revision, approval of final manuscript.

Jan Jakobsson; Manuscript preparation, revision, approval of final manuscript.

Guarantor

Jan Jakobsson.

Trial registry number

Not applicable.

This Case report has been written in accordance to the SCARE Guidelines [9].

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