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CASE REPORT

Out-of-hospital cardiac arrest from air embolism during sexual intercourse: Case report and review of the literature☆

Anatolij Truhlar[a,b,*], Vladimir Cerny[b], Pavel Dostal[b], Miroslav Solar[c], Renata Parizkova[b], Iva Hruba[c], Ladislav Zabka[a]

[a] *Helicopter Emergency Medical Service ''Christoph 6'' Hradec Kralove, Hradec Kralove Region Emergency Medical Services, Czech Republic*
[b] *Department of Anaesthesiology and Intensive Care Medicine, Charles University Prague, Faculty of Medicine Hradec Kralove, University Hospital Hradec Kralove, Czech Republic*
[c] *Department of Internal Medicine, Charles University Prague, Faculty of Medicine Hradec Kralove, University Hospital Hradec Kralove, Czech Republic*

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Summary We report the successful resuscitation of a 38-year-old woman in cardiac arrest following heterosexual intercourse 7 days after spontaneous abortion and an instrumental uterine evacuation. The collapse was thought to be due to venous air embolism (VAE). Her survival neurologically intact was attributed to appropriate first aid, pre-hospital and subsequent hospital intensive care. Neither a case of an out-of-hospital air embolism where the patient made a good recovery, nor a case of miscarriage followed by collapse from air embolism has been reported in the literature. Air embolism is a very infrequent cause of out-of-hospital cardiac arrest with a high mortality rate. Predominant causal reasons are severe penetrating neck or thoracic injuries and sexual activities in pregnancy, when air can pass into the damaged veins in the wall of the uterus and lead to total obstruction in the heart. Diagnostics and management techniques for venous air embolism are discussed. Air embolism should be included in the differential diagnosis for all young women in cardiac arrest, particularly when occuring during sexual activity. Instructions in risks of sexual intercourse during pregnancy and the puerperium should become part of pregnant women's education.

Abbreviations: CPC, cerebral performance category; ECG, electrocardiogram; EMS, emergency medical service; GPS, global positioning system; ICU, intensive care unit; ROSC, return of spontaneous circulation; VAE, venous air embolism

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* Present address: Helicopter Emergency Medical Service ''Christoph 6'' Hradec Kralove, Hradec Kralove Region Emergency Medical Services, Hradecka 1690/2A, 500 12 Hradec Kralove 12, Czech Republic. Tel.: +420 604 967 417.
E-mail address: ATruhlar@seznam.cz (A. Truhlar).

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Introduction

Life threatening air embolism has become important in anaesthesiology and intensive care medicine as invasive intravascular procedures have become widespread. Cardiovascular, thoracic, neurological and obstetric surgeries have also increased amount of documented venous air embolisms (VAE). In order to develop VAE, there must be communication between the vascular lumen and the source of air as well as a pressure gradient favouring input of the air into the vessel. Air embolism can occur during any surgical procedure where there is low venous pressure in the surgical field, especially when elevated above the level of the heart. Neurosurgical and head and neck surgery performed on patients in the sitting position are the most common clinical situations. Within obstetrics, minor VAE occurs very frequently during cesarean delivery. In the intensive care unit (ICU), air embolism is most likely to be encountered during or after insertion of a central venous catheter. The air may enter the great veins directly after the needle is inserted if the hub is not occluded.[1–4]

The clinical manifestation varies in both severity and organs affected. The signs are not specific and mostly include tachypnea, cyanosis, tachycardia, arrhythmias, hypotension and chest pain. The most severe cases can lead to unconsciousness and cardiopulmonary arrest. In humans, the fatal dose of air is uncertain, but estimated to be between 300 and 600 ml if administered in a single, rapid injection (100 ml/s). The volume required to cause death decreases as the rate of air entry increases. [1,2]

In the out-of-hospital setting, VAE is a very infrequent cause of cardiac arrest with a high mortality rate as therapeutical possibilities are very limited compared with in-hospital cases. Predominating causal reasons are severe penetrating neck or thoracic injuries and sexual activities in pregnancy when air bubbles can pass through the impaired wall of the uterus and lead to blood flow obstruction under certain circumstances.[1,5,6] The vagina is a highly distensible organ, especially in pregnancy, and has an immediate postpartum capacity up to 2000 ml.[7,8] It can easily contain a fatal amount of air under increasing pressure sufficient to dissect through the cervical canal and reach the venous drainage of the uterus. Some risk remains for 6 weeks after childbirth while the uterus is returning to normal and the placental site is healing.

We report a successful cardiopulmonary resuscitation (CPR) of a young woman in a full cardiac arrest following sexual intercourse where the collapse was thought to be due to VAE. Neither a case of out-of-hospital VAE where the patient made a good recovery nor a case of miscarriage followed by collapse from air embolism has been reported in the literature.

Figure 1 An emergency response vehicle (Mercedes-Benz 316 CDI Sprinter, Germany) and its medical equipment used in the town of Hradec Kralove (Czech Republic). Photo: A. Truhlar, MD.

Case report

In August 13th, 2005, a 38-year-old woman was engaged in sexual intercourse with her husband in a rear entry position. While the act was still in progress, the young woman unexpectedly developed unconsciousness, gasped and collapsed down on the bed. Seven days before she had undergone an evacuation of retained products of conception (ERPOC) after spontaneous abortion in the 12th week of gestation in the University hospital. It was her third pregnancy after she had given birth to two healthy children. Although the gynaecologist advised her of the risk of sexual activities after the surgical procedure, the advice was not respected.

An emergency call from the husband was received in the regional emergency medical dispatch centre in the town of Hradec Kralove at 1:49:08 a.m. ($T = 0$). The national emergency telephone number for ambulance service (155) was used. An emergency physician, a paramedic and a driver staffed the mobile intensive care unit (Figure 1), which was alerted at 1:51:10 a.m. (T + 2 min 2 s) and left its base immediately. After an emergency response vehicle was dispatched to the scene, the EMS dispatcher provided the husband with telephone-assisted CPR and stayed on the line until the ambulance arrived. The ambulance stopped at 1:58:57 a.m. (T + 9 min 49 s) and EMS personnel got to the patient's side in the third floor at 2:01:06 a.m. (T + 11 min 58 s).

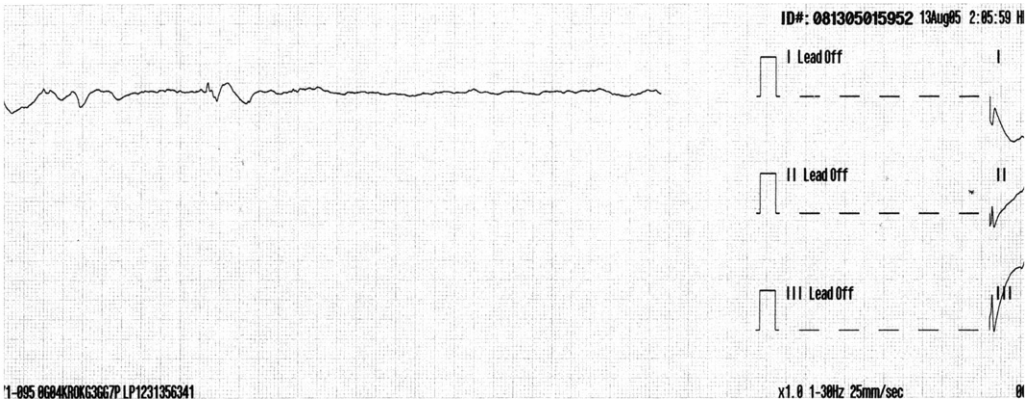

Figure 2 The first monitored rhythm (asystole) recorded shortly after the EMS personell arrival at the patient's side.

The woman was found lying naked on the floor in the bedroom in asystolic cardiac arrest. She had widely dilated, unreactive pupils. There was striking hyperaemia and cyanosis of the upper thorax, neck and face with increased jugular veins filling. She was slightly bleeding from trachea and genitals. Her husband was providing his wife with mouth-to-mouth breathing and chest compressions. The ambulance staff took over care and immediately started advanced life support (ALS) approximately 12 min after the collapse happened.

ALS was performed according to the International Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (International Liasion Commitee on Resuscitation). The patient's airway was secured by a tracheal tube (internal diameter 7.5 mm) without any complications. Intermittent positive-pressure ventilation (IPPV) was initiated with a transport ventilator Oxylog 1000 (Dräger Medical, Germany). Ventilatory variables were: FIO_2 1,0, respiratory rate 12/min, tidal volume 10 ml/kgBW, I:E ratio 1:2, positive end-expiratory pressure (PEEP) level of 3 cm H_2O. The patient was given adrenaline [epinephrine] (1:1000) 1.0 mg by the tracheal route. After venous access via an external jugular vein was obtained and secured, 1.0 mg of adrenaline IV was given every 3 min (up to total dose of 8.0 mg). Atropine 1.0 mg was given as asystole was the first monitored rhythm (Figure 2). Ventricular fibrillation (VF) developed after 5 min. Biphasic defibrillation with Lifepak 12 device (Medtronic Physio-Control, USA) was performed five times (initial sequence of 200, 300 and 360 J external shocks was delivered). Amiodarone in a single dose of 300 mg IV was given for refractory VF after the first three defibrillation efforts failed. The clinical symptoms suspicious of acute pulmonary embolism made an emergency physician give heparin IV in a dose of 5000 units as an antithrombotic agent. No alkaline buffer was

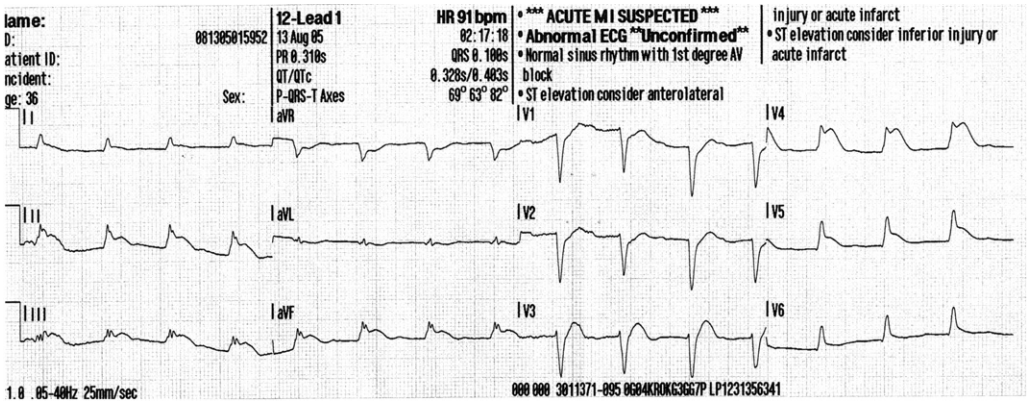

Figure 3 A 12-lead electrocardiogram (Lifepak 12, Medtronic, USA) obtained approximately 4 min after the return of spontaneous circulation showed sinus rhythm with first degree atrio-ventricular block and ST-segment elevations (II, III, aVF, V_3—V_5) suggesting inferior and anterolateral injury or acute myocardial infarction. The right coronary artery originates above the right cusp of the aortic valve on the anterior wall of ascendent aorta and thus can be predominantly blocked by air in a patient who is lying down.

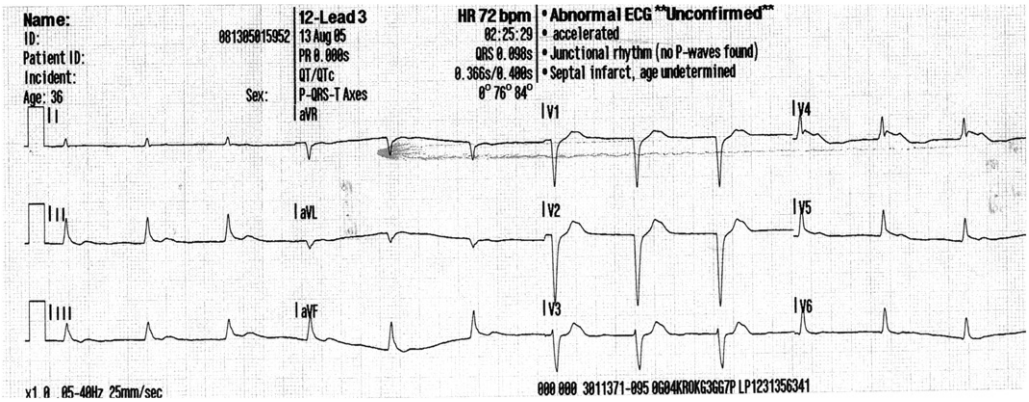

Figure 4 In order to decide if primary transport to the coronary intervention laboratory was required, the prehospital electrocardiogram was repeated. The second record showed junctional rhytm and non-specific ST-segment abnormalities. At this moment myocardial infarction was not considered to be the probable cause of cardiac arrest in the differential diagnosis.

used during CPR. The fifth defibrillation attempt (360 J) was successful at 2:13:10 a.m. (T + 24 min 2 s) and a spontaneous circulation was restored (ROSC). In order to maintain MAP above 70 mmHg, an infusion of noradrenaline [norepinephrine] was started at an approximate rate of 0.9 μg/kg/min. 12-lead electrocardiography (ECG) was completed at the scene at 2:17:18 a.m. (Figure 3) and at 2:25:29 a.m. repeated (Figure 4) as the ECG pattern promptly changed after ROSC. The first ECG showed sinus rhythm with first degree atrio-ventricular block and ST-segment elevation suggesting anterolateral and inferior injury or acute myocardial infarction. The second record showed junctional rhythm and non-specific abnormalities of the ST-segment. The woman was transferred directly to the University hospital department of anaesthesiology and intensive care medicine. Mechanical ventilation had to be enabled by sufentanil 10 μg IV and pipecuronium 4.0 mg IV during transportation.

The patient was admitted to ICU at 2:50 a.m. (T + 61 min). The blood pressure at the time of admission was 107/75 mmHg, the heart rate was 94/min, the oxygen saturation was 98% while the patient was being ventilated with FIO_2 0.6, and the temperature was 34.7 °C. After connection to the ventilator, an arterial catheter, central venous catheter, nasogastric tube, Foley catheter and oesophageal temperature probe were placed. At 3:00 a.m., shortly after the patient's arrival at ICU, moderate therapeutic hypothermia was started with target of core body temperature 33 °C to prevent ischaemic brain damage. Metabolic acidosis was corrected with a bicarbonate infusion. There was haemodynamic instability and a mean arterial pressure above 65 mmHg was maintained by noradrenaline infusion. Analgesia and sedation was provided by midazolam and sufentanil. Continuous infusion of furosemide 1 mg/h was started to keep the urine output above 100 ml/h. During the first hour of admission an ECG was recorded and blood samples were taken. The ECG revealed sinus rhythm with a negative T-wave in $V_1 - V_6$ with no significant changes over the ensuing days. Selected biochemistry values were: creatinkinase, 5.64 μkat/l; MB fraction of creatinkinase, 2.37 μkat/l; troponin T, 0.626 μg/l; D dimers, 19.4 mg/l; arterial lactate, 9.6 mmol/l. The arterial blood gases were pH, 7.1; p_{O_2}, 80 mmHg; p_{CO_2}, 40.5 mmHg; BE −15. The cardiology service was consulted immediately but cardiac catheterisation was not advised. Echocardiography was not performed due to the bedside unavailability. After 24 h, therapeutic hypothermia was completed and controlled rewarming started at 0.5 °C/h. Simultaneously sedation was tapered and the dose of noradrenaline decreased. On day 4 major organ functions were stable, she started to obey commands and was extubated after a trial of spontaneous breathing lasting 10 min. On day 5 the patient was discharged from ICU with no apparent neurological deficit.

After ICU discharge, the patient underwent cardiac catheterisation, lower-extremity venous testing (duplex ultrasonography), thoracic and pelvic ultrasound, transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), telemetry heart monitoring and ventilation—perfusion scanning to exclude possible causes of out-of-hospital cardiac arrest. Brain and heart magnetic resonance (MR) imaging and brain MR angiography (MRA) were performed in August, 26th with no abnormalities detected. No signs suspicious of arrhythmogenic right ventricle dysplasia were found. The left ventricle ejection fraction was cal-

culated to be 67%. An electrophysiological study (EP study) was performed on August, 29th and did not detect abnormalities in the cardiac electrical conduction system. Gynaecological, neurological and haematological examinations were done with no pathological findings. Prophylactic anticoagulation therapy with low-molecular-weight heparin (LMWH) was terminated and the woman was discharged from hospital on September, 1st (on day 20) with a cerebral performance category (CPC) of 1 (conscious, no neurological disability). At 6-month follow-up she was in good health with a CPC of 1.

Discussion

The differential diagnosis in a young healthy patient such as this woman, who presented with an out-of-hospital cardiac arrest, is broad and includes cardiac and non-cardiac disease. An extensive diagnostic process was performed to find the primary cause of her sudden collapse.

Ischaemic heart disease is the leading cause of death in the world.[9] The patient's initial ECG was suggestive of acute myocardial infarction, but subsequent ECG records showed only non-specific variations. No characteristic symptoms of myocardial ischaemia (chest pain, dyspnoea, epigastric discomfort) were present in the patient, although some patients present asymptomatically. Coronary artery disease was excluded at coronary angiography. The initial ECG changes and the transient elevation of cardiac markers can be attributed to the cardiac arrest and prolonged course of chest compressions. Further samples showed borderline or normal values.

Cardiac causes of obstruction to blood flow include cardiac tamponade, aortic stenosis and hypertrophic obstructive cardiomyopathy. There was no known history of trauma to explain cardiac arrest due to the rapid accumulation of blood in the pericardial sac. Non-traumatic tamponade caused by aortic dissection or myocardial rupture could not be resuscitated without rapid surgical intervention. Aortic stenosis and obstructive cardiomyopathy usually cause symptoms including dyspnoea and chest pain. In hospital, all these causes were definitely excluded by MR scan and echocardiography.

Patients with congenital anatomical heart disease may also be at increased risk for sudden death from cardiac causes even if the defect has been repaired.[10] No signs of structural heart disease were found both on echocardiography and MR imaging. The patient had no history of congenital heart disease.

Primary dysrhythmic causes of cardiac arrest may or may not be associated with structural heart disease. Coronary artery disease, dilated and hypertrophic cardiomyopathy were excluded. Telemetry heart monitoring and an electrophysiological study did not detect any signs of dysrhythmias or structural abnormalities in the electrical conduction system of the heart. There were no cardiac dysrhythmias present in the patient's history. Although we are not able to exclude a dysrhythmic cause for sure, it seems to be unlikely.

Sudden cardiac arrest can also happen in patients with viral myocarditis where ST-segment elevation and increased enzyme levels are described despite normal coronary arteries.[10] However, this patient neither had preceding signs of infection nor chest pain.

Most frequent non-cardiac internal causes of out-of-hospital cardiac arrest include lung disease, cerebrovascular disease, epilepsy, metabolic disorders (diabetes mellitus, renal disease) and pulmonary embolism.[9] Respiratory causes, including bronchospasm, aspiration, or both, should be considered but seem to be unlikely in a patient with no history of dyspnoea. The previous medical history together with the complete recovery and the normal result of the neurological examination (including brain MR imaging, brain MR angiography and carotid ultrasound) makes a neurovascular origin of cardiac arrest very improbable. There was also no supporting historical or physical evidence of seizures. Metabolic disorders and intoxication were excluded by laboratory examination.

Non-cardiac conditions that obstruct blood flow, such as spontaneous tension pneumothorax, can also lead to cardiac arrest but not without preceding symptoms (chest pain and dyspnoea), as it was in our case. Moreover, this condition is unlikely to be successfully resuscitated without treating the precipitating cause. Pneumothorax was exluded by a normal chest X-ray performed immediately after ICU admission.

As the patient presented with hyperaemia and cyanosis of the upper thorax, increased jugular veins filling and elevated D-dimer level, pulmonary embolism was initially considered to be the most likely diagnosis. However, because elevations of D-dimer are non-specific, an abnormal result has a low positive predictive value. The positive D-dimer level after previous cardiac arrest, but negative lower-extremity venous testing (duplex ultrasonography), thoracic and pelvic ultrasounds reduced the likelihood of deep venous thrombosis and from that resulting thromboembolism. Ventilation—perfusion

Table 1 Clinical details of the 22 reported cases of severe out-of-hospital air embolisms associated with sexual activities

Patient details	VAE circumstances	Clinical manifestation	Outcome	Ref.
38-year-old woman, 7 days after abortion (12th week)	Heterosexual intercourse with rear entry position	Collapse, gasping, cardiac arrest	Survival, full recovery	Reported case
36-year-old woman, pregnancy (30th week)	Orogenital sex (air insufflation)	Abdominal tightness, seizures, unconsciousness (without cardiac arrest)	Survival, neurologic dysfunction	Bray et al.[8]
19-year-old woman, pregnancy (27th week)	Orogenital sex (air insufflation)	Collapse, dyspnea, somnolency (without cardiac arrest)	Survival, infant delivered and survived	Fyke et al.[5]
Pregnancy (38th week)	Orogenital sex (air insufflation)	Unknown	Survival, infant delivered and survived	Hill and Jones[21]
Pregnancy	Orogenital sex (air insufflation)	Cardiac arrest	Death	Kaiser[26]
28-year-old woman, pregnancy (30th week)	Orogenital sex (air insufflation)	Abdominal pain, convulsions, cardiac arrest	Death	Aronson and Nelson[7]
20-year-old woman, pregnancy (38th week)	Orogenital sex (air insufflation)	Gasping, convulsions, cardiac arrest	Death	Aronson and Nelson[7]
16-year-old woman, pregnancy (20th week)	Orogenital sex (air insufflation)	Dyspnea, collapse, death	Death	Heid[27]
17-year-old woman, pregnancy (20th week)	Orogenital sex (air insufflation)	Sudden death	Death	Benjamin[28]
16-year-old woman, pregnancy (24th week)	Orogenital sex (air insufflation)	Sudden death	Death	Wuermeling[29]
17-year-old woman, pregnancy	Orogenital sex (air insufflation)	Sudden death	Death	Allen[30]
17-year-old woman, pregnancy (20th week)	Orogenital sex (air insufflation)	Sudden death	Death	Hendry[31]
26-year-old woman, pregnancy	Orogenital sex (air insufflation)	Sudden death, strong vaginal bleeding	Death	Soska et al.[32]
16-year-old woman, pregnancy (full term)	Orogenital sex (air insufflation)	Collapse, cardiac arrest (death in 35 min)	Death	Fatteh et al.[33]
19-year-old woman, pregnancy	Orogenital sex	Not stated	Death	Herzig and Mojola[34]
Pregnancy	Vaginal ''fisting'' during foreplay	Unknown	Death, infant delivered brain dead	Eckert et al.[17]
Pregnancy	Heterosexual intercourse with rear entry position	Unknown	Death	Lifschultz and Donoghue[18]
22-year-old woman in puerperium (8 days after delivery)	Heterosexual intercourse with rear entry position	Sudden collapse, vaginal bleeding, cardiac arrest	Death	Batman et al.[13]
29-year-old woman in puerperium (5 days after delivery)	Heterosexual intercourse in the missionary position	Sudden death	Death	Batman et al.[13]
Non-pregnant woman	Vaginal ''fisting''	Unknown	Death	Fain and McCormick[14]
40-year-old non-pregnant woman	Autoerotic practice (carrot insertion)	Unknown	Death	Marc et al.[16]
17-year-old non-pregnant woman	Heterosexual intercourse with rear entry position	Sudden death (probable vaginal laceration during digital foreplay)	Death	Sadler and Pounder[15]

scanning is an essential diagnostic study and a normal scan essentially excluded diagnosis of pulmonary thromboembolism.[11,12]

Not only thromboemboli released into the venous circulation can induce acute pulmonary vascular obstruction, but other embolic sources such as fat, tumor cells, amniotic fluid, foreign bodies and air. Based on the patient's history (collapse during sexual intercourse after recent instrumental evacuation of retained products of conception), clinical presentation and an absence of evidence of any alternative explanation, we assume that the VAE was very likely to be responsible for cardiac arrest.

Out-of-hospital air embolism leading to acute right-heart failure or cardiac arrest is very rare. In the literature, it is described more commonly in pregnant women (Table 1). In the pregnant female, the air is forced into the cervical canal by insufflation or the piston effect of the penis or finger in the vagina and could dissect the placental membranes and enter the non-collapsible veins at the placental edge. The key factor is the distensibility of the vagina in pregnancy during which up to 2000 ml of air under pressure may be introduced.[8]

No case of severe air embolism following miscarriage has been reported yet. The mechanism seems to be the same as in reported deaths due to sexual intercourse in puerperium. Uterine spiral arteries are converted by invading trophoblasts into distended uteroplacental arteries in the early pregnancy. The sequence of events by which these arteries thrombose and the endometrium regenerates after parturition is not clearly understood but involution of the placental site is normally complete within about 3 weeks. Delay in the physiological obliteration of the large vessels underlying the placental site is not uncommon and typically presents as a haemorrhage in the first few weeks of the puerperium.[13] In that period, the wall of the uterus where the placenta was attached during pregnancy is raw with gaping blood vessels. If air is introduced into the uterus under pressure the air bubbles tend to pass into the circulation. The potential hazard of sexual intercourse in pregnancy, puerperium and after miscariage should be more widely appreciated by physicians and all pregnant women should be informed that having sex within 6 weeks after delivery or abortion carries a risk that can even be fatal.

In non-pregnant females, fatal air embolism following repeated insertion of the fist into the vagina and following penile-vaginal intercourse have also been reported.[14,15] An exceptional case was reported in a woman using a carrot in the vagina during an autoerotic practice.[16] The mechanism of a very rare VAE in a non-pregnant female is thought to be through vaginal laceration and rupture of vaginal wall veins into which a large volume of air is introduced by the piston effect of either the fist, penis or foreign body in the vagina.[14–17]

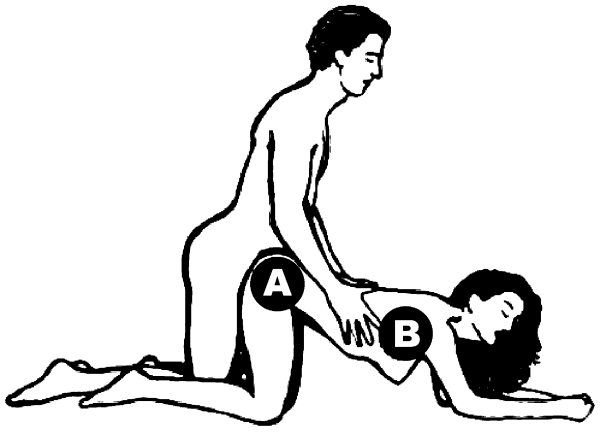

Figure 5 Sexual rear entry position during an intercourse seems to increase the danger of air embolism. Pelvic elevation with the uterus (A) raised above the right ventricle (B) produces pressure gradient favouring air passage to the heart.

Although the effect of coital posture has not been studied, three cases of VAE during heterosexual intercourse were reported with a rear entry position.[13,15,18] We suppose that the sexual position, particularly if the uterus is raised above the right ventricle during intercourse (Figure 5), can significantly increase the danger of air embolism. Pelvic elevation produces a gravitational pressure gradient favouring passage of air along the inferior vena cava.[15] Also in the reported case, a rear entry position prior to cardiac arrest was confirmed by questioning the male partner.

Patients with VAE may present with a wide variety of mainly non-specific symptoms. Pulmonary or cardiovascular function compromise often dominates the clinical picture. The most serious clinical manifestation of VAE leading to out-of-hospital cardiac arrest should be differentiated from aortic dissection, pericardial tamponade, myocardial infarction, ventricular dysrhythmia, stroke, intracranial bleeding, pulmonary thrombembolism, tension pneumothorax and acute pulmonary oedema. Some are potentially reversible causes for which specific treatment exists and must be considered during any cardiac arrest, particularly if non-shockable rhythms are being monitored [1,19].

Laboratory abnormalities vary with the severity of the embolism but lack specificity for this process as well. Reports of VAE in humans describe sinus tachycardia, non-specific ST-segment and T-

wave changes and evidence of acute ventricular strain. Different atrial and ventricular dysrhythmias may be noted. Chest radiographs are usually normal but later may show evidence of pulmonary oedema. Arterial blood gases reflect abnormalities in the matching of ventilation and perfusion. Pulmonary artery pressure may remain normal, with an increased central venous pressure, due to a large air bubble present in the outflow tract of the right ventricle. Only three findings are relatively specific for VAE: X-ray visualization of air in the right heart or pulmonary arteries, air bubbles in the retinal vessels and the characteristic ''mill wheel'' murmur.[1,5] If the air blocking the right ventricle is mechanically separated into smaller bubbles with a closed-chest cardiac massage, detection of VAE is very difficult. In cases such as ours, in which specific findings were absent, the diagnosis of air embolism was mainly based on the patient's history and exclusion of another possible causes explaining out-of-hospital cardiac arrest.

In anaesthesiology, monitoring methods (e.g. precordial and/or transoesophageal echocardiography, intra-arterial blood pressure and/or pulmonary artery pressure monitoring, capnography) are used for early recognition of clinically important VAE and they became standard in high-risk surgical procedures. After VAE detection immediate measures should follow: (1) rapid identification and obturation of the air entry, (2) 100% oxygen delivery, (3) patient positioning (head down on the left side position may result in reduction of the right ventricular outflow obstruction), (4) air retrieval using a central venous catheter or direct needle puncture of the right heart in the case of cardiac arrest and (5) other measures to stabilise the circulation (e.g. vasoactive drugs, chest compressions, cardiopulmonary bypass). Closed-chest compressions have been recommended as a means of forcing air from the right ventricle into the small pulmonary vessels. Hyperbaric oxygen therapy may also be useful in patients with severe central nervous system or cardiac manifestations.[1,4,8,20]

In the pre-hospital setting, the majority of diagnostical procedures widely used in the hospital environment are not applicable. The patient's history, clinical manifestation and a few monitoring methods (e.g. ECG, oxygen saturation, capnometry) are the only features leading to a working differential diagnosis out of hospital. The possibilities of therapeutic interventions are also limited. The extent of these measures depends on emergency ambulance or helicopter equipment and the structure of the EMS team. The European EMS model may profit from physician-staffed emergency response vehicles in certain conditions. Above all, the therapeutic and decision-making competences of an emergency physician are more extensive compared with those in a paramedic system.

Only three cases of non-fatal air embolism associated with sexual activity have been reported in medical literature, but none had documented cardiac arrest and successful cardiopulmonary resuscitation.[5,8,21] In our case, the patient's survival without remaining neurological sequalae was attributed to the sequence of high quality bystander CPR, emergency physician guided prehospital care and hospital intensive care. Cardiac arrest outcome depends on immediate resuscitative efforts. Any delay before treatment, either basic or advanced life support, determines both the immediate and overall prognosis.[22] Dispatcher-assisted CPR may also contribute to a higher rate of survival from out-of-hospital cardiac arrest.[23]

The most important determinant of ROSC is the interval from collapse to initiation of CPR. However important the collapse time identification is for making therapeutic decisions, the EMS personnel are usually not able to obtain this information with the exception of witnessed cardiac arrests. In the reported case, we suppose basic life support was started within 2 or 3 min after the collapse occurred. The time the call was received was recorded by EMS dispatch centre software (Dispecer, Profia, Czech Republic) as well as all the core time events associated with emergency response vehicle movements (the time the first emergency response vehicle is mobile and the time the vehicle stops). There is a difference between the call—response interval (period from call receipt by EMS dispatcher to the moment the emergency response vehicle stops moving) 9 min 49 s (time points extracted from EMS dispatch and ambulance GPS navigation softwares), and the real collapse-to-CPR by EMS personnel interval that took more than 11 min 58 s definitely. The exact time points were extracted from the relevant emergency call record as an EMS dispatcher provided the husband with telephone instruction in CPR until the EMS personnel arrival. A short delay was registered in the call-processing time which exceeded the 1-min standard as the excited husband was neither able to describe the main symptoms nor location of the incident. In most cases of data collection it is difficult to document the exact time of arrival at the patient's side, taking into account the interval from leaving the emergency response vehicle to beginning the advanced life support. This previously established interval is therefore no longer recommended to be recorded for resuscitation registries.[24,25]

Pre-hospital ROSC is just the first step toward the survival to hospital discharge. Interventions in the post-resuscitation period are likely to influence the final outcome significantly. One of them could be mild therapeutic hypothermia that is thought to suppress many of the chemical reactions associated with reperfusion brain injury and optimise neurological recovery.[19] Immediate temperature control during early post-resuscitation care might have contributed to the good clinical outcome. The woman was discharged from hospital 3 weeks after the cardiac arrest with a CPC of 1 and 6 months later passed a foreign language exam successfully.

Conclusion

We report a case of out-of hospital cardiac arrest in a young woman having sexual intercourse shortly after abortion and instrumental uterine evacuation. The most probably cause of cardiac arrest was air embolism. A retrospective diagnosis of air embolism related to sexual intercourse was based on the patient's history and negative information about other possible causes explaining cardiac arrest. This case appears to be the first reported instance of successfully resuscitated air embolism associated with sexual intercourse in the out-of-hospital setting. The patient's survival without lasting morbidity was attributed to the sequence of immediate bystander CPR and highly skilled pre-hospital and subsequent hospital intensive care.

The potential risk of sexual intercourse during the first few weeks after delivery or abortion should be more emphasised by physicians. Instructions in risks of sexual intercourse during pregnancy and the puerperium should become an inseparable part of pregnant women's education. Infrequent air embolism should be included in the differential diagnosis for all young women in cardiac arrest, particularly when it happens during sexual activity.

Conflict of interest

There is no financial and personal relationship with other people or organizations that could inappropriately influence our work, all within 3 years of beginning the work submitted.

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