Case Report

BJMP 2017;10(1):a1006

Timely intervention and effective multidisciplinary input for a woman with multi-organ failure secondary to cardiac arrest due to ruptured ectopic pregnancy

Sucheta Jindal & M Mweemba

Abstract

The clinical presentation of ectopic pregnancy is extremely variable ranging from asymptomatic to haemorrhagic shock. Unforeseen tubal rupture can be a source of substantial morbidity and mortality. There has been a worldwide decrease in the case fatality rate in women with ectopic pregnancies, suggesting it is largely due to the early detection and prompt management of ectopic pregnancies.

Here we report a case of a ruptured ectopic pregnancy of a 27-year-old woman who was unaware of her pregnancy and presented with cardiac arrest followed by multi-organ failure but showed a miraculous recovery after 72 hours, all visceral functions returning to normal within 7 days.

This case report gives insight to all medical specialities, the importance of active multi-disciplinary resuscitation measures. This will also help the medical students as part of their problem based learning to understand the basic sciences behind sharp decline and remarkable recovery in life threatening emergencies.

Précis

The significance of effective communication within multidisciplinary teams especially in emergency situations towards optimising patient care and saving lives cannot be understated.

Case Report

A 27-year-old woman who claimed to be unaware of her current pregnancy collapsed at her home. She was not known to have any co-morbidities. Paramedics were called and found her to be in cardiac arrest with pulseless electrical activity. Cardiopulmonary resuscitation (CPR) was immediately commenced. Spontaneous circulation returned after 13 minutes of CPR at home.

She was then transferred to the emergency department. On arrival to the emergency department her Glasgow Coma Scale (GCS) was 3. She had a pulse rate of 130 beats per minute; unrecordable blood pressure; haemoglobin of 55g/l; metabolic acidosis with a pH of 6.8; lactate > 15; and a potassium of 6.6 mmol/l. She was resuscitated and gradually regained consciousness with a GCS of 15.

In the midst of stabilising her condition and unaware of her pregnancy, a urine pregnancy test was obtained following siting of a urinary catheter. A positive pregnancy test prompted notification to the gynaecology team who performed ultrasonography imaging which revealed significant haemoperitoneum. An immediate decision was made to perform laparotomy in view of the most likely diagnosis of a ruptured ectopic pregnancy.

Laparotomy revealed a 3.5 litre of haemoperitoneum secondary to a ruptured right sided tubal ectopic pregnancy. A right salpingectomy was performed. The patient was subsequently transferred to the intensive care unit as her serology results were consistent with multi organ failure with a platelet count of 46 (10^9/L); creatinine of 194 mmol/L; estimated glomerular filtration rate (egfr) of 27 mls/min/ 1.73 m^2; alanine transaminase (ALT) of 441 IU/L; and alkaline phosphatase (ALP) of 49 IU/L.

She made an uneventful recovery as demonstrated in figure 1 by the improving serological parameters and was discharged home after 6 days.

Discussion

The confidential enquiry report into maternal deaths – UK has shown a decreasing trend in the case fatality rate in women with ectopic pregnancies. This has been suggested to reflect earlier detection and immediate treatment of ectopic pregnancies. However unforeseen tubal rupture with major haemorrhage continues to be a source of major morbidity and mortality. Ectopic pregnancies account for 3-4% of pregnancy related deaths. [4]

The classical triad of symptoms encountered in ectopic pregnancy includes pain, vaginal bleeding and amenorrhoea. [1]Worryingly, as illustrated by our case, rarely these women may present in a state of collapse even before the diagnosis of pregnancy is made. [4]

Figure 1: The cumulative serology- full blood count, liver function tests, urea and electrolytes, clotting profile.ankle

Investigations	Day 0	Day 0	Day 1	Day 1	Day 2	Day 3	Day 4	Day 5	Day 15
	13:46	18:30	05:53	17:27	06:49	07:00	11:14	09:37	09:50
Hb g/L (115-150)	82	100	83	73	72	88	89	93	
WCC 10^9/L (3.5-11.0)	19.8	23	18.1	13.2	11.8	11.2	9.5	9.1	
Plts 10^9/L (140-400)	46	61	49	46	48	51	76	106	
ALP IU/L (30-130)		49	42	44	51	57	73	74	100
ALT IU/L (0-40)		441	428	701	3197	2621	1807	1290	185
Bili mmol/L (0-21)		9	13	8	18	18	16	11	4
Na mmol/L (133-146)	139	142	143	141	143	142	140	141	139
K+ mmol/L (3.5-5.3)	6.8	4	4.3	4.2	3.9	3.9	3.7		4.6
Urea mmol/L (2.5-7.8)		9.6	12.4	14	14.2	11.5	7.9	7.4	8.3
Creat mmol/L (48-128)	194	174	230	279	319	269	163	137	88
egfr mls/min/ 1.73 m^2	27	30	22	18	15	19	33	40	66
INR ratio	1.4	1.4	1.5	1.6	1.4	1.1	1		
PT secs (9.7-12.3)	14.8	15.1	15.9	16.7	15.1	11.5	10.6		
Fibrinogen g/L (1.9-3.1)	1.2	1.4	1.2	1.5	2.4	3.9	>4.5		

Pathophysiology of multi-organ failure following haemorrhagic shock

Our case clearly demonstrates the detrimental multi-systemic effects and subsequent threat to life created by haemorrhage from a ruptured ectopic pregnancy. Acute haemorrhage results in decreased cardiac output and pulse pressure that is detected by baroreceptors in the aortic arch and atrium. Neural reflexes subsequently cause an increased sympathetic outflow to the heart and other vital organs resulting in vasoconstriction, and redistribution of blood flow away from non-vital organs. Neuroendocrine responses activated by neural reflexes play a major role in homeostasis during haemorrhage. Elevated aldosterone and cortisol secondary to raised adrenocorticotrophic hormone secreted by the pituitary gland leads to increased water absorption in the kidneys. The reduced tissue perfusion to non-vital organs results in insufficient delivery of oxygen and nutrients required for cellular function. [2]

The resultant hypoxia leads to anaerobic metabolism and hence lactate production and metabolic acidosis. Hyperlactaemia is defined when serum lactate is greater than 4 mmol/l. [3] A level of 15mmol/l as demonstrated by our case highlights the extent of shock the patient was in.

Endogenous heat production is restricted by anaerobic metabolism, which in turns exacerbates hypothermia that is likely to be predisposed by the administration of intravenous fluids and blood products. Hypothermia is one of the reversible causes of pulseless electrical activity and a core temperature of less than 35°C is itself an independent predictor of mortality after major haemorrhage.

Furthermore, our case revealed a severe acidosis with a pH of 6.8, which is reflective of widespread cellular anaerobic respiration secondary to hypoxia as a result of inadequate perfusion. Widespread literature has shown that a pH of less than 7.2 is associated with decreased contractility, low cardiac output, bradycardia, arrhythmias and decreased blood flow to the liver and kidneys. This can lead to multi-organ failure.[6]

Many patients with severe haemorrhage can establish coagulopathy very quickly as our case has demonstrated. At present there is nouniversally established definition of coagulopathy though many experts use prolonged prothrombin time as an indicator of coagulopathy. Our case presented with a prolonged prothrombin time of 14.8 seconds. The pathophysiology is complex and stems from immediate activation of multiple haemostatic pathways including

fibrinolysis, platelet and endothelium dysfunction. Furthermore, acute phase response after resuscitation measures can create a prothrombotic state. Sometimes, disseminated intravascular coagulation can occur in those who are insufficiently resuscitated or not resuscitated in a timely manner. [7]

Effective multi-disciplinary input

This case clearly highlights that the responsibility does not solely rely on the surgeon who is required to cease the bleeding but also on the multi-disciplinary specialists including paramedics, emergency clinicians, nursing staff, anaesthetists and haematologists. This is a vital component of resuscitation management during emergency situations.

Appropriate initial fluid management

The management with intravenous fluid resuscitation remains challenging as some evidence suggests that aggressive fluid resuscitation can be detrimental because it can lead to dislodging of clots and dilutional coagulopathy leading to increased risk of haemorrhage. [5]

Clinicians supporting this hypothesis suggest to cautiously administer fluid resuscitation with the aim of maintaining a subnormal blood pressure (systolic of 70-90 mmHg), whilst allowing sufficient oxygen delivery. The very early use of crystalloids and blood products is paramount to help treat acute coagulopathy. [7]

Immediate surgical treatment

Recourse to immediate surgical cessation of bleeding is a vital part of the resuscitation process, and must not be delayed. [7]The presence of free fluid in the abdomen and a positive pregnancy test immediately alerted an ectopic pregnancy as the most likely diagnosis. The majority of women of reproductive age are free of comorbidities with a greater ability to adapt to resuscitative measures and hence showing quicker recovery.

Conclusion

Teaching Points

1. Despite advances in the management of ectopic pregnancies an emphasis must be given on improving the understanding of the women and the healthcare professionals of the pathophysiology of haemorrhagic shock.
2. Educating the public and all health care professionals about the phrase “Think Ectopic” as a main differential in any women of childbearing age with atypical signs and symptoms of general ill health is paramount.

Despite advances in the management of ectopic pregnancies an emphasis must be given on improving the understanding of the women and the healthcare professionals of the pathophysiology of haemorrhagic shock. Educating the public and all health care professionals about the phrase “Think Ectopic” as a main differential in any women of childbearing age with atypical signs and symptoms of general ill health is paramount.

Furthermore, the significance of effective communication within multidisciplinary teams towards optimising patient care and saving lives cannot be understated.

Competing Interests

None declared

Author Details

SUCHETA JINDAL, Consultant Obstetrician & Gynaecologist, Pilgrim Hospital, United Lincolnshire Hospitals NHS Trust, UK, PE21 9QS. M MWEEMBA, ST4 Obstetrics and Gynaecology, Calderdale & Huddersfield Hospitals NHS Trust, Halifax, UK, HX3 0PW.

CORRESPONDENCE: SUCHETA JINDAL, Consultant Obstetrician & Gynaecologist, United Lincolnshire Hospitals NHS Trust, Pilgrim Hospital, Sibsey Road, Boston, Lincs, PE21 9QS.

Email: drsucheta@yahoo.com

References

1. Alsuleiman SA, Grimes EM. Ectopic pregnancy: a review of 147 cases. J Reprod Med. 1982 Feb. 27(2):101-6.
2. H.Haljame, The pathophysiology of shock. Acta Anaesthesiol Scand 1993: 37, Supplementum 98: 3-6
3. Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med. 2008;36(1):296–327.
4. Centre for Maternal and Child Enquiries (CMACE). Saving Mothers’ Lives: reviewing maternal deaths to make motherhood safer: 2006–08. The Eighth Report on Confidential Enquiries into Maternal Deaths in the United Kingdom. BJOG 2011;118(Suppl. 1):1–203
5. Cherkas D. Traumatic hemorrhagic shock: Advances in fluid management. Emerg Med Pract. 2011;13:1–19.
6. Chatrath V, Khetarpal R, Ahuja J. Fluid management in patients with trauma: Restrictive versus liberal approach. Journal of Anaesthesiology, Clinical Pharmacology. 2015;31(3):308-316. doi:10.4103/0970-9185.161664.
7. Cap A, Hunt BJ. The Pathogenesis of Traumatic Coagulopathy. Anaesthesia 2015, 70 (Suppl. 1), 96–101.