CASE REPORT

Successful emergency splenectomy during cardiac arrest due to cytomegalovirus-induced atraumatic splenic rupture

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SUMMARY

A 27-year-old woman was admitted to the emergency department with fever and a petechial rash on suspicion of meningitis. Shortly after arriving she developed cardiac arrest. Blood work up showed severe lactate acidosis, anaemia and thrombocytopenia. A focused assessment with sonography in trauma scan showed free intraperitoneal fluid and an emergency laparotomy revealed massive bleeding from a ruptured spleen. The patient was successfully resuscitated. She proved to be infected with cytomegalovirus causing idiopathic thrombocytopenic purpura, splenomegaly and splenic rupture. She was treated for 14 days with ganciclovir and meropenem and discharged on recovery. Atraumatic splenic rupture caused by viral infection is a rare condition although well described. In the case of our patient, thrombocytopenia added to the severity of the splenic rupture. A multidisciplinary team approach was essential for the management and the eventual recovery of the patient.

BACKGROUND

The case shows the importance of a broad range of differential diagnoses when receiving critically ill patients in the emergency department (ED). In addition, it underlines the importance of accessibility of doctors from different disciplines, and the value of teamwork and full exploitation of resources.

CASE PRESENTATION

A 27-year-old woman presented with fever at a local hospital on a remote Danish island with only one hospital doctor on call. As a young child, she had undergone a successful surgery for Tetralogy of Fallot with complete recovery and normal cardiac function. She was otherwise healthy and without medication.

One week prior to admission, the patient started to feel tired; she had a slight fever and nausea as well as diffuse muscle and abdominal pain. The day before admission she developed an itchy rash throughout the arms and legs, and started to bleed from her gingivae and her nose, and developed a severe headache and hypersensitivity to bright light.

When seen at the local hospital, the patient was described as pale, with a universal petechial rash. She was febrile with a temperature of 38.7°C. She had neck tenderness but no stiffness. Her blood

pressure was low (71/35 mm Hg) and an arterial puncture showed raised serum lactate at 3.4 mmol/L (figure 1). Suspecting meningitis caused by *Neisseria meningitidis*, the patient was transferred to our University Hospital by helicopter. Prior to transport, blood samples including blood cultures were drawn, but there was no time for lumbar puncture. Treatment was started with intravenous penicillin G and ceftriaxone preceded by infusion of methylprednisolone as the standard recommended, dexamethasone, was not available.

On arrival in the ED, the patient was restless and confused. A medical trigger team tried to calm her, but she reported of not being able to feel her legs and of abdominal pain, and was trying to flee the stretcher. It was impossible to measure her blood pressure or heart rate or to perform arterial blood gas analysis due to her agitated state. Considering her petechial rash and increasing confusion, the suspicion of meningitis was sustained.

Although the patient had already received antibiotics, it was considered important to sample spinal fluid for microbiological investigation, and a lumbar puncture was prepared with assistance from anaesthesiology. Prior to sedation, however, the patient started to vomit and lose consciousness and finally went into cardiac arrest with pulseless electrical activity. Heart and lung resuscitation (HLR) was started immediately and a cardiologist was called. While performing HLR the patient was intubated and continuously ventilated. Several intravenous and intraosseous cannulations were secured and rapid infusion of normal saline was initiated.

An arterial blood sample was drawn, and surprisingly showed haemoglobin (Hb) of 2.7 mmol/L (4.35 g/dL), lactate 14.8 mmol/L and pH 6.86. The cardiologist performed a bedside echocardiography and found severe hypovolaemia and ceased systolic function. Suspecting hypovolaemia originating from bleeding to be the cause of the cardiac arrest, a FAST (focused assessment with sonography for trauma) scan of the abdomen was performed.

The FAST revealed free intra-abdominal fluid and a diagnostic puncture confirmed the suspicion of intraperitoneal bleeding. A new arterial puncture showed a lactate of 8.2 mmol/L and Hb was 1.1 mmol/L (1.76 g/dL). The patient's pH had risen to 7.04. HLR was continuously performed by the team. The surgeons who were called to the scene decided to perform an acute laparotomy in the

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Analysis	Reference	1:03 p.m.	2:48 p.m.	2:55 p.m.	3:36 p.m.
pH	7,35-7,45	7,42	6,86	7,04	6,88
pO ₂	11,1-14,4 kPa	11,1	<2,7	3,9	39,6
pCO ₂	4,3-6,0 mmol/L	4	9,6	5,3	10,2
HCO ₃	21,0-28,0 mmol/L	21	12	10,3	13,5
Lactate	0,5-1,6 mmol/L	3,4	14,8	8,2	23
Base exch	-6	-4	-19	-17,7	-18,8
Hemoglot	7,3-9,5 mmol/L	7,2	2,7	1,1	4,7

Figure 1 Arterial blood analysis at different times. 13:05 at the local hospital, 14:48 and 14:55 in the emergency department during cardiac arrest. 15:36 after successful resuscitation. The condition continues to be critical although it is slowly stabilising.

emergency room, as stopping the bleeding seemed to be the only possible way to save the young patient's life.

There was no time to secure a sterile environment or to summon experienced nurses to assist the surgeons. While the team of ED doctors and anaesthesiologists were running the HLR and trying to replace the lost blood with massive transfusions of RhO-ve blood and fresh frozen plasma, the surgeons opened up the abdomen with a long vertical incision. Immediately, 3–4 L of blood with only a few clots came out. While compressing the aorta just below the diaphragm, it became possible to transfuse the patient up to a sufficient blood pressure, and after 25 min of cardiac arrest with primarily pulseless electric activity and only one episode of shockable rhythm treated with direct-current conversion, spontaneous circulation returned. During the HLR a total of 10 mg of intravenous adrenalin was administered.

The surgeons discovered a ruptured spleen to be the cause of the bleeding. The spleen was enlarged, porous and with three large lacerations. An immediate splenectomy was performed and the bleeding was stopped. While removing the spleen, the tail of the pancreas and the ventricular wall were accidentally damaged with minor lesions. They were quickly sutured and haemostasis was established. A surgical drain was put in the splenic bed and a KCI ABThera vacuum-assisted closure (VAC) system with continuous suction of 125 mm Hg was connected.

The condition of the patient was now stabilising though still very critical. A total of 18 portions of O-ve red cell transfusion, 6 portions of fresh frozen plasma and 6 portions of thrombocytes had been administered during HLR and surgery. On arterial blood gas analysis at the end of the surgery Hb had increased to 4.7 mmol/L (7.5 g/dL). Throughout the process, the anaesthesiologist reported normally sized pupils with a normal reaction to light and the patient had reacted with gasping after the HLR. The blood pressure and pulse were stable, and the patient was transported from the ED to the intensive care unit. Because of the non-sterile conditions during surgery, single doses of meropenem and metronidazole were added to the antibiotic regimen.

During the surgery, the ED team leader received a message from the primary hospital stating the initial blood samples showed haemoglobin of 5.7 mmol/L and a thrombocyte count of 0! Disseminated intravascular coagulation (DIC) secondary to meningococcal bacteraemia was suspected, but the coagulation factors II and V were 0.57, only slightly under the normal range (0.70–1.30), and the D-dimer was 1.76 mg/L and only mildly elevated (<0.50 mg/L), which did not add up to the DIC being

severe enough to completely eliminate the thrombocytes from the circulation. An acute haematological condition, such as thrombotic thrombocytopenic purpura (TTP), was suspected. Since almost the entire blood-pool of the patient had been replaced by transfusion, it was pointless to perform diagnostic procedures on current blood samples. Fortunately, the primary hospital had saved blood samples, and these were transported to the University Hospital and immediately examined by a haematologist and a pathologist. The blood was without any schistocytes but with many neutrophil granulocytes with an overweight of virocytes. TTP and acute leucaemia were ruled out and a viral infection was suggested to have caused the idiopathic thrombocytopenic purpura (ITP) and thereby the thrombocytopenia. Serological testing for Epstein-Barr virus (EBV), cytomegalovirus (CMV), parvovirus, and hepatitis A, B and C, were performed together with PCR for EBV and CMV, and antibody testing for HIV. Treatment with ganciclovir 5 mg/kg was initiated and the treatment with penicillin G, ceftriaxone and dexamethasone was continued as meningitis was not definitively disproved.

During the following days, the patient continued to be intubated and lightly sedated. She seemed to be cerebrally intact with relevant responses to questions when awake, and she was able to move her arms and legs.

The patient was reoperated twice to stop bleeding from the abdominal muscles, to change the VAC system and to ensure that no surgical equipment was left behind in the abdomen during the primary emergency surgery. The postoperative period was further complicated by development of non-oliguric acute kidney failure.

Results from the initial blood samples confirmed the suspicion of a viral infection being the trigger of the thrombocytopenia, the splenomegalia and the splenic rupture. CMV DNA was found in both, the blood from the local hospital and in the samples taken after surgery, respectively, 15 000 and 80 000 copies/mL. The treatment for meningitis was stopped and changed to meropenem, ganciclovir and prednisolone. The ladder was to avoid renewed destruction of the thrombocytes.

In the following weeks, the patient continued to recover. She was extubated 3 days after admission, her kidney function gradually normalised and apart from a minor pneumonia, there were no infectious complications. After the first few days, there was no need for further surgery and the patient gradually started to get out of bed. Treatment with ganciclovir and meropenem was continued for a total of 14 days with decreasing amounts of CMV DNA in the blood.

DIFFERENTIAL DIAGNOSIS

- Meningitis caused by *N. meningitidis* because of the petechial rash
- TTP because of the thrombocytopenia
- DIC caused by agents other than those mentioned above

TREATMENT

The hypovolaemia caused by splenic rupture was treated by splenectomy and massive transfusion.

The suspected meningitis was treated with intravenous penicillin G, ceftriaxone and dexamethasone, following the recommended standard treatment of our hospital.

The infection with CMV was treated with intravenous ganciclovir for 14 days. The treatment was supplemented with meropenem because of the unsterile environment during surgery and complicating pneumonia.

OUTCOME AND FOLLOW-UP

Five months after admission, the patient is in full recovery and is expected to start working again within the next couple of months. She will now be evaluated by immunologists to find out if she suffers from any immunological defects that can explain the strong reaction to an otherwise quite harmless viral infection.

DISCUSSION

CMV is a member of the herpes virus family and normally causes mild or even asymptomatic infection in immunocompetent individuals. It establishes a latent infection but CMV-related disease is mostly related to primary infection. The infection may present as infectious mononucleosis with fatigue, fever and lymphadenopathy as seen in patients infected with EBV, but CMV is also known to cause pneumonia, colitis and hepatitis. In immunocompromised patients, such as transplant recipients or patients infected with HIV, CMV can cause severe life-threatening infections. Infection with CMV is common, and the seroprevalence rate ranges from 40% to 100% in the adult population with the highest rates found in developing countries.

Splenomegaly is well described in patients with infectious mononucleosis, although it is most often associated with EBV. The infection causes the spleen to enlarge, but it is generally a self-limiting condition as the size of the spleen usually decreases and normalises 1–3 months after symptomatic illness. The splenomegaly is usually asymptomatic, but the patient might experience pain and discomfort in the upper left quadrant of the abdomen. Atraumatic splenic rupture is a rare but severe complication, with increasing abdominal pain, haemodynamic instability and anaemia as red flag symptoms. The patient often needs urgent surgical treatment, and a splenectomy can be required to stop the bleeding, as seen in our case. In a review of 613 cases of splenic ruptures without risk factors, Aubrey-Bassler and Sowers¹ found infection to be the most frequent cause of rupture, followed by haematological and neoplastic diseases. In a review by Renzulli *et al*,² the overall mortality related to atraumatic splenic rupture was 12.2%. Risk factors for death were splenomegaly, neoplastic disorders and age over 40 years.

Immune thrombocytopenic purpura (ITP) caused by viral infection—in this case CMV—was suggested to be causing the severe depletion of platelets in our patient. In recent years, several cases of apparently ITP have been found to be secondary to CMV infection.³ Even though the infection itself is mild and transient, the virus might trigger an autoimmune process against the platelets. The mechanism is not yet fully understood, but the antibodies are believed to destroy the platelets peripherally and to directly infect the megakaryocytes, thereby inhibiting platelet production in the bone marrow.³

Even though atraumatic splenic rupture and thrombocytopenia secondary to CMV infection is a rare condition, it is

already described in several case reports and reviews. What makes the history of our patient unique is the surprising development from suspected meningitis into cardiac arrest and successful resuscitation during emergency laparotomy. Considering the massive blood loss, the 25 min of HLR, and a blood pH with a minimum of 6.6, it is even more surprising that the patient recovered completely. She was discharged 29 days after the dramatic admission. She walked out of the hospital without any help and is expected to be able to return to her job. She will now be evaluated by immunologists to investigate if she has any immunological conditions that can explain the unusual reaction to the CMV infection.

If the patient had not been transferred to a University Hospital immediately, the outcome might not have been as positive, and we believe that the multidisciplinary team approach with doctors from the ED, internal medicine, surgery, anaesthesiology, cardiology and haematology, was essential for the outcome.

Learning points

- ▶ Infectious mononucleosis caused by Epstein-Barr virus or cytomegalovirus can result in splenomegaly and severe thrombocytopenia in otherwise healthy adults.
- ▶ Splenomegaly is a relatively harmless condition when caused by viral infection, but it may result in atraumatic splenic rupture and require immediate surgery.
- ▶ A multidisciplinary approach is essential when receiving diagnostically unsorted patients *in extremis*.

Contributors SP, JMRN and KRM managed the portion of the text concerning their specialty and department, and contributed to the overall reviewing of the text. MKG is the leading and corresponding author, and put the text together and managed the portion pertaining to CMV infection. SP was the team leader, receiving the patient with assistance from MKG. JMRN was leading surgeon and KRM was responsible for the patient in the intensive care unit. When debriefing the episode all the authors identified the case as suitable for publication.

Competing interests None.

Patient consent Obtained.

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REFERENCES

- 1 Aubrey-Bassler FK, Sowers N. 613 Cases of splenic rupture without risk factors or previously diagnosed disease: a systematic review. *BMC Emerg Med* 2012;12:11.
- 2 Renzulli P, Hostettler A, Schoepfer A, *et al*. Systemic review of atraumatic splenic rupture. *Br J Surg* 2009;96:1114–21.
- 3 DiMaggio D, Anderson A, Bussell JB. Cytomegalovirus can make immune thrombocytopenic purpura refractory. *Br J Hematol* 2009;146:104–12.

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