DEFINITION OF SHOCK

- life-threatening medical condition

- inadequate perfusion of tissues and hypoxia of vital organs

- Inability / failure of circulation to maintain adequate perfusion of organs necessary for survival
CHARACTERISTICS OF SHOCK

- results from a critical decrease in blood flow to vital organs and/or the inability (or increased demands) of tissues to utilize oxygen and nutrients

- cardiovascular system cannot maintain adequate perfusion of organs, necessary for survival

- the common denominator of different forms of shock is the collapse of microcirculation

- fatal outcome is irreversible shock, with
  - dysfunction of cell membranes,
  - abnormal cellular metabolism and
  - death of the critical number of cells, resulting in organ failure
CHARACTERISTICS OF SHOCK

- The substantial **decrease in blood pressure**, resulting in a decreased perfusion of tissues and organs.

- The critical factor is the **time of hypoperfusion**.

- With duration of hypoperfusion, originally **positive compensatory mechanisms**, aimed at maintaining the perfusion of vital organs, turn into negative.

- The clinical picture is often dominated by one organ (*the critical organ is the one that did not function properly before the onset of shock*).

- The development of shock often depends on the **functional reserve of the malfunctioning organ** (*ischemic heart disease, cirrhosis, nephritis...*)
Shock - pathogenesis

Collaps of microcirculation - the common denominator of different forms of shock

Small vessels (100-150 μm): the perfusion of tissues during shock

distribution of blood is modified by:
- intrinsic autoregulatory mechanisms (metabolites)
- external autonomic and humoral influences

distribution of blood:
- precapillary arterioles
- precapillary and postcapillary sphincters
ETIOPATHOGENESIS

Tissue Hypoperfusion

Cells switch from aerobic to anaerobic metabolism
Increased lactate production / accumulation

Cellular dysfunction

Increased permeability of cellular membranes
Electrolytes & fluids seep in & out of cell, cells swell

Cells Die in Many Organs

Organ dysfunction and failure

Death
Effects of inadequate perfusion on cell function

**Contributors**
- Local and circulating mediators of inflammation
- Oxidative stress: ROS
Major causes of shock

- Hypovolemia
- Heart failure
- Vasodilatation

Symptoms of shock:
- Severe hypotension
- Cold, clammy skin
- Edema
- Thrombosis
- Hemorrhage
- Somnolence, coma
- Oliguria
- Dyspnea
- GI bleeding

Complications:
- Renal failure
- Gastrointestinal lesions
- Lung failure (ARDS)
- Death due to cardiorespiratory failure
COMPONENTS of Cardiovascular System

fluids

pump

pipes
Maintenance of circulation requires adequate blood pressure

Blood Pressure = Systemic Vascular Resistance (pipes) x Cardiac Output (fluids, pump)

If the Blood Pressure is low, then either:

1. Systematic Vascular Resistance is low or
2. Cardiac Output is low or
3. both are low
Factors affecting tissue/organ perfusion:
- blood pressure
- blood volume
Systemic Vascular Resistance is low

**Causes:**
- septic shock
- anaphylactic shock
- neurogenic shock

They cause **vasodilatation:**
- the patient is warm
  - pink
  - bounding pulse
  - hyperdynamic circulation

(\textit{in the initial phase of shock})
Cardiac output is low

Cardiac Output = Heart rate $\times$ Stroke Volume

**Stroke volume** depends on:
1. **Preload** (is the ventricle full?)
   - hypovolemic shock
   - obstructive shock
2. **Function of Heart** (can the ventricle contract?)
   - cardiogenic shock
Components of Blood Pressure: summary

Blood Pressure
  - Cardiac Output
    - Stroke Volume
      - Myocardial Contractility
      - Preload
      - Afterload
  - Systemic Vascular Resistance
    - Heart Rate
Cardiovascular system

Regulation of blood pressure

Mean arterial blood pressure is determined by:

1. Blood volume
   - Determined by fluid intake and fluid loss
   - May be passive or regulated at kidneys

2. Effectiveness of the heart as a pump (cardiac output)
   - Determined by heart rate and stroke volume

3. Resistance of the system to blood flow
   - Determined by diameter of the arterioles

4. Relative distribution of blood between arterial and venous blood vessels
   - Determined by diameter of the veins
The signs & symptoms of shock

<table>
<thead>
<tr>
<th>ORGAN SYSTEM</th>
<th>SYMPTOM OR SIGN</th>
<th>CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Mental status changes</td>
<td>Decreased Cerebral perfusion</td>
</tr>
<tr>
<td></td>
<td>Pinpoint pupils</td>
<td>Narcotic overdose</td>
</tr>
<tr>
<td>Circulatory Heart</td>
<td>Tachycardia</td>
<td>Adrenergic stimulation, depressed contractility</td>
</tr>
<tr>
<td></td>
<td>Other dysrhythmias</td>
<td>Coronary ischemia</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
<td>Depressed contractility secondary to ischemia or MDFs, right ventricular failure</td>
</tr>
<tr>
<td>Systemic</td>
<td>New murmurs</td>
<td>Valvular dysfunction, VSD Decreased SVR, decreased venous return</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
<td>Hypovolemia, decreased venous return</td>
</tr>
<tr>
<td></td>
<td>Decreased JVP</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increased JVP</td>
<td>Right heart failure</td>
</tr>
<tr>
<td></td>
<td>Disparate peripheral pulses</td>
<td>Aortic dissection</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Tachypnea</td>
<td>Pulmonary edema, respiratory muscle fatigue, sepsis, acidosis</td>
</tr>
<tr>
<td></td>
<td>Cyanosis</td>
<td>Hypoxemia</td>
</tr>
<tr>
<td>Renal</td>
<td>Oliguria</td>
<td>Decreased perfusion, afferent arteriolar vasoconstriction</td>
</tr>
<tr>
<td>Skin</td>
<td>Cool, clammy</td>
<td>Vasoconstriction, sympathetic stimulation</td>
</tr>
<tr>
<td>Other</td>
<td>Lactic acidosis</td>
<td>Anaerobic metabolism, hepatic dysfunction</td>
</tr>
<tr>
<td></td>
<td>Fever</td>
<td>Infection</td>
</tr>
</tbody>
</table>
SHOCK: clinical signs

- pale skin
- cold sweat
- quick, shallow breathing
- cyanosis
- alterations of mental status, anxiety, restlessness
- tachycardia, palpitations
  
  *(from hypoperfusion of CNS and activation of the sympathetic nervous system)*

- hypotension
- oliguria (less than 20 ml of urine / hour)

- thirst
- breathing difficulties
- coldness
- diziness
Acute congestion of liver due to shock
Acute tubular necrosis of the kidney in shock
Intestinal mucosal hemorrhages in shock
Adrenal gland hemorrhage in shock
Compensatory mechanisms aim at improving oxygenation of vital organs:

- the maintenance of blood pressure
- maximization of cardiac function
- redistribution of circulating blood towards vital organs
- optimal utilization of oxygen in tissues
Compensatory mechanisms in shock

**SNS activation**
- Decreased baroreceptor stimulation
- Sympathetic nerves and adrenal medulla stimulated
- Norepinephrine and epinephrine released into sympathetic synapses and into bloodstream
- Systemic arteriolar and venous vasoconstriction, increased myocardial contractility, and increased HR
- Increased CO and increased blood pressure

**Endocrine response**
- Decreased arterial pressure
- Stimulates posterior pituitary to secrete ADH
- Vasoconstriction leading to increased SVR and blood pressure and increased venous return to the heart (preload) and increased CO

**Renin-Angiotensin activation**
- Decreased renal perfusion and increased sympathetic stimulation
- Releases renin to stimulate angiotensin I
- Angiotensin I is converted to angiotensin II by ACE
- Arteriolar constriction and stimulates adrenal cortex to release aldosterone
- Kidney conserves sodium and water to increase preload

---

Figure 54-2 Compensatory mechanisms in shock. ACE, angiotensin-converting enzyme; ADH, antidiuretic hormone; CO, cardiac output; HR, heart rate; SNS, sympathetic nervous system.

STAGES of shock

- **Compensated / early shock**
  - Vasoconstriction (RAAS & carotid sinus baroreceptor)
  - Increase in HR (sympathetic activation)
  - Patients usually normotensive (aldosterone/ADH activation; Na+/H$_2$0 retention)

- **Decompensated / late shock**
  - Cool, clammy skin, hypotension
  - Vital organ preservation
  - Continued increase in HR (Chemoreceptor response to metabolic acidosis)

- **Irreversible shock**
  - HR drop – Multi-Organ Failure (Impending death)
Multiple Organ Dysfunction Syndrome (MODS)

<table>
<thead>
<tr>
<th>Number of Organs</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.8</td>
</tr>
<tr>
<td>1</td>
<td>6.8</td>
</tr>
<tr>
<td>2</td>
<td>26.2</td>
</tr>
<tr>
<td>3</td>
<td>48.5</td>
</tr>
<tr>
<td>4</td>
<td>68.8</td>
</tr>
<tr>
<td>5</td>
<td>83.3</td>
</tr>
</tbody>
</table>
SHOCK: DIAGNOSIS

- Mean Arterial Pressure < 60 mmHg or a decrease of 20 mmHg from baseline

- systolic Blood Pressure ≤ 90mmHg OR

- drop in systolic Blood Pressure > 40mm Hg from the patient’s normal BP

- Shock index (Heart Rate > Systolic Blood Pressure)

- Clinical signs of hypoperfusion of vital organs
<table>
<thead>
<tr>
<th>Table - 4: General Approach to Shock: Initial Diagnosis and Evaluation⁵</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical</strong></td>
</tr>
<tr>
<td><strong>Laboratory</strong></td>
</tr>
<tr>
<td><strong>Monitoring</strong></td>
</tr>
<tr>
<td><strong>Imaging</strong></td>
</tr>
</tbody>
</table>
Circulating lactate as a predictor of shock severity: A peak blood lactate level of >4.0 mmol/L - a strong independent predictor of mortality and morbidity and suggests tissue hypoperfusion.
Sublingual O₂

- Decreased gut perfusion
  - Stomach = esophagus = sublingual tissue
- Non-invasive, hand-held monitor
- Rapid measurement
- Sensitive marker of decreased blood flow
MANAGEMENT of shock

Goals: 1. restore Blood Pressure
2. normalize systemic perfusion
3. preserve organ function

Parameters of adequate resuscitation:

1. Urine output (0.5 - 1.0 ml/kg/h)
   (adequate renal perfusion)
2. Reversal of lactic acidosis (nl. pH)
   (improved perfusion)
3. Normal mental status
   (adequate cerebral perfusion)
COMPONENTS of Cardiovascular System: the classification of shock

- fluids
- pump
- pipes
Adequate circulating blood volume depends on 3 components.

A minor impairment in one can be compensated for by the other 2 for a limited time.

Prolonged or severe impairments will lead to SHOCK.
TYPES of shock

1. Hypovolemic (fluids)
2. Cardiogenic (pump)
3. Distributive (pipes)
1. HYPOVOLEMIC SHOCK - definition

- blood loss that cannot be managed
- development of shock depends on the velocity of blood loss

quick loss of blood - shock
slow loss of blood - asymptomatic

Figure 15-3 Pathophysiologic sequence of events in hypovolemic shock.
1. HYPOVOLEMIC SHOCK - causes

- hemorrhage
- vomiting
- diarrhea
- dehydration
- third-space loss
- burns

**loss of blood** (external or internal bleeding or blood donation)

**loss of plasma** (severe burns and lesions discharging fluid)

**loss of body sodium and consequent intravascular water**
1. HYPOVOLEMIC SHOCK: clinical signs

<table>
<thead>
<tr>
<th>% Blood loss</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 15</td>
<td>Slightly increased heart rate</td>
</tr>
<tr>
<td>15-30</td>
<td>Increased HR, increased DBP (narrow pp), prolonged capillary refill, flat neck veins</td>
</tr>
<tr>
<td>30-50</td>
<td>Above findings plus: hypotension, confusion, acidosis, decreased urine output</td>
</tr>
<tr>
<td>&gt; 50</td>
<td>Refractory hypotension, refractory acidosis, death</td>
</tr>
</tbody>
</table>
1. HYPOVOLEMIC SHOCK: diagnosis

- ↓ cardiac output
- ↓ Central Venous Pressure
- ↑ Systematic Vascular Resistance
2. CARDIOGENIC SHOCK - definition

- results from damage to the myocardium, or inhibition of myocardial contractility
- the most common cause is myocardial infarction
- is characterized by
  - an increase in central venous pressure,
  - an increase in peripheral resistance and
  - a decrease in cardiac output
Figure 15-5 Pathophysiologic sequence of events in cardiogenic shock.
2. CARDIOGENIC SHOCK - causes

- Myocardial infarction
- Heart failure
- Severe arrhythmia
- Severe valvular dysfunction
2. CARDIOGENIC SHOCK – clinical signs

- **Skin**: progressive peripheral vasoconstriction results in cool, moist / clammy, pale skin with mottling
- **Heart**: murmurs
- **Pulse**: rapid, thready/weak pulse, due to the reduced circulation combined with tachycardia
- **Blood Pressure**: decreased Blood Pressure and Mean Arterial Pressure
- Distended jugular veins due to increased jugular venous pressure
- **Kidneys**: Decreased renal perfusion
- **CNS**: Anxiety, restlessness, altered mental state due to decreased cerebral perfusion and subsequent tissue hypoxia
- **Lungs**: Pulmonary edema involving fluid back-up in the lungs due to insufficient pumping of the heart
2. CARDIOGENIC SHOCK - diagnosis

- ↓ cardiac output
- ↑ Central Venous Pressure
- ↑ Systematic Vascular Resistance
- ↓ left ventricular stroke work (LVSW)
3. DISTRIBUTIVE SHOCK

Types
- Septic
- Anaphylactic
- Acute adrenal insufficiency
- Neurogenic

Signs / diagnosis
± cardiac output
+/− Central Venous Pressure or Pulmonary wedge pressure; heart rate
Reduced systemic vascular resistance/SVR
an abnormal distribution of blood flow in the smallest blood vessels results in an inadequate supply of blood to the body's tissues and organs

**Types of distributive shock:**

- Septic shock
- Anaphylactic shock
- Neurogenic shock
ANAPHYLACTIC shock - characteristics

- Rapid onset
- Diffuse systemic vasodilation (histamine & bradykinin release)
- Edema from increased capillary permeability
- Bronchoconstriction
ANAPHYLACTIC shock: causes

- reaction to insect sting
- application of contrast substances
- vaccination
- pharmacotherapy
- milder forms can occur after specific food
ANAPHYLACTIC shock: clinical signs

- Cutaneous manifestations
  urticaria, erythema, pruritus, angioedema

- Respiratory compromise
  stridor, wheezing, bronchorrhea, respiratory distress

- Circulatory collapse
  tachycardia, vasodilation, hypotension

- CNS
  altered consciousness, coma
ANAPHYLACTIC shock - treatment

- Remove the antigen
- Restore / maintain **Acid-base balance**
- **IV Fluids, O₂, ECG, BP** monitoring, pulse oxymetry

**First line: Epinephrine**

For severe bronchospasm, laryngeal edema, signs of upper airway obstruction, respiratory arrest or shock: IV epinephrine

100 micrograms of 1:100,000 (place 0.1 mL of 1:1000 in 10 mL of NS, give over 5-10 min)

If less severe, can give 0.3-0.5 mL 1:1000 SC

- **2nd line:**
  - H1 blocker: Diphenhydramine 25-50 mg IV
  - H2 blocker: Ranitidine 50 mg or Famotidine 20 mg IV
  - Steroids (Methylprednisolone 125 mg IV or Prednisone 40-60 mg po)
  - Albuterol (*bronchodilation*)

For patients taking Beta-blockers with refractory hypotension, think about glucagon (*cardiovascular effects, positive chrono and inotropic effect*)
SEPTIC shock: characteristics

SIRS – Sepsis - Severe Sepsis - Septic Shock

Sepsis = Systemic Inflammatory Response Syndrome (SIRS) + confirmed or presumed infectious etiology

Severe Sepsis: SIRS criteria + source of infection and infection-induced organ dysfunction or hypoperfusion abnormalities (sepsis + lactic acidosis/oliguria/etc.)

Septic Shock: SIRS criteria + source of infection + and hypotension not reversed with fluid resuscitation and associated with organ dysfunction or hypoperfusion abnormalities

Systemic Inflammatory Response (SIRS) manifested by two or > of following:

- Temp > 38 or < 36 °C
- HR > 90 beats per minute
- PaCO₂ < 32
- WBC > 12,000/mm³ or > 10% Bands (immature neutrophils)
Excessive inflammatory mediator production during sepsis.

Various stimuli can cause activation of different cell types and serum proteins, as well as the coagulation and complement systems, leading to excessive production of pro-inflammatory cytokines and chemokines and upregulation of adhesion molecules on endothelial cells and polymorphonuclear leukocytes (PMNs). Monocytes, PMNs and other phagocytes release large amounts of granular enzymes and generate ROS in response to the original stimulus in the early (hyperreactive) phase of sepsis. As result of excessive pro-inflammatory mediator production, vascular permeability increases, tissue damage and organ failure occur and crucial innate immune functions become defective, resulting in increased susceptibility toward infection in the later / hyporeactive phase of the immune response, often along with immune paralysis.
Pathogenetic networks in shock. Lipopolysaccharide (LPS) and other microbial components simultaneously activate multiple parallel cascades that contribute to the pathophysiology of adult respiratory distress syndrome (ARDS) and shock. The combination of poor myocardial contractility, impaired peripheral vascular tone and microvascular occlusion leads to tissue hypoperfusion and inadequate oxygenation, and thus to organ failure.
SEPTIC shock: risks factors

- Age: <1 year and >65 years
- Surgical / invasive procedures
- Malnutrition
- Chronic illness
  DM, CRF, Hepatitis, CVD…
- Compromised immune status
  AIDS, immunosuppressives, EtOH, malignancies…
- Pathogens resistant to pharmacoth
SEPTIC shock: clinical signs

circulation

early phase, vasodilation:
- vasodilation and loss of intravascular volume
- warm extremities
- lower systemic resistance
- increased or normal cardiac output

late phase, vasoconstriction:
- lower myocardial contractility, loss of intravascular fluid, periferal vasoconstriction
- cold extremities
- hypotension
- reduced cardiac output
- normal or increased systemic resistance
NEUROGENIC shock: characteristics

- Essential derangement: paralysis of the sympathetic nervous system which controls vascular tone, caused by thoracic or cervical level spinal cord injury.

- Produces decreased systemic vascular resistance from a loss of vascular tone and bradycardia from unopposed parasympathetic input.
NEUROGENIC shock: causes

- **Neurogenic shock** is a distributive type of shock resulting in hypotension, occasionally with bradycardia, that is attributed to the disruption of the autonomic pathways within the spinal cord. Hypotension occurs due to decreased systemic vascular resistance resulting in pooling of blood within the extremities lacking sympathetic tone.

- Neurogenic shock can result also from a severe central nervous system damage (brain injury, cervical or high thoracic spinal cord)
NEUROGENIC shock: clinical signs

- Hypotension without tachycardia
- **Warm pink skin** from cutaneous vasodilation
- Low BP with minimal response to fluids
- Accompanying neurologic deficit
Obstructive Shock

**Causes**
- Cardiac Tamponade
- Tension Pneumothorax
- Massive Pulmonary Embolism

**Signs**
- ↓ cardiac output
- ↑ CVP
- ↑ SVR

**Treatment**
- Needle decompression
- Embolectomy
Cases
A case report

• a 48-year-old woman developed a high temperature, was diagnosed as having a respiratory tract infection.

• The next day, she became unconscious and was transferred to the intensive care unit for monitoring and treatment.

• She complained of increasing lower back pain as consciousness improved.

• MRI of the lumbar spine showed an abscess shadow between the vertebral body and dura mater at the level of L3–S1.

• Surgical drainage of the epidural abscess was performed as an emergency procedure, and the patient recovered immediately.

• The primary source of the epidural abscess was probably the respiratory tract infection, which spread to the epidural space through hematogenous dissemination.

• The initial treatment of the epidural abscess was rest and antibiotics, but surgical treatment was needed to save the patient.

A lumbar epidural abscess caused septic shock

Yayama et al, 2003
Case report

Case presentation
After a **minor surgical intervention**, a 32-year-old Caucasian woman with no significant medical history went into sudden hemodynamic deterioration due to acute heart failure.

An urgent echocardiogram showed severe biventricular dysfunction and an estimated left ventricular ejection fraction of 20%. Extracorporeal life support and mechanical ventilation were required.

Five days later her ventricular function had fully recovered, which allowed the progressive withdrawal of medical treatment.

Prior to her hospital discharge, cardiac MRI showed neither edema nor pathological deposits on the delayed contrast enhancement sequences. At her six-month follow-up examination, the patient was asymptomatic and did not require treatment.

Conclusion
Although there are many causes of **cardiogenic shock**, the presence of abrupt hemodynamic deterioration and the absence of a clear cause could be related to the use of **propofol and fentanyl**.

Gonzales et al, 2011
Case report

- Cardiogenic shock is very uncommon in healthy people.

- The differential diagnosis for patients with acute heart failure in previously healthy hearts includes acute myocardial infarction and myocarditis.

- However, many drugs can also depress myocardial function. Propofol and fentanyl are frequently used during different medical procedures. The cardiovascular depressive effect of both drugs has been well established, but the development of cardiogenic shock is very rare when these agents are used.

Gonzales et al, 2011
Case report
An association between influenza A viruses and myocarditis was noted during the 1918 influenza pandemic. Since then, the link between the influenza B virus and fulminant myocarditis or cardiogenic shock has been rarely reported.

Case presentation
a 50 year-old-woman without known heart disease presented in profound cardiogenic shock with a left ventricular ejection fraction of 10%.
This was preceded by six days of fever, chills, myalgia and fatigue.
She had a junctional tachycardia, a troponin I of 12.6 ng/ml and her coronary angiography demonstrated normal coronary arteries.
Percutaneous extracorporeal membrane oxygenation was required.
An endotracheal aspirate at admission was positive for influenza B.
All other respiratory, blood and urine cultures were negative.
On day 7, a repeat echocardiography demonstrated significant recovery of left ventricular function with an ejection fraction of 50%. She was later discharged home in good condition.

Conclusions
Influenza B infection can be complicated by fulminant myocarditis leading to cardiogenic shock in adults without preexisting cardiac disease.

Taremi et al, 2013
Case report

- Ranitidine hydrochloride (Zantac®), a histamine-2-receptor antagonist, is a widely used medication with an excellent safety record. Anaphylactic reaction to ranitidine is an extremely rare event and a related death has never been described in the literature.

Case presentation

- a 51-year-old man with negative anamnesis for allergic events, died suddenly after the intravenous administration of one phial of Zantac® 50 mg prescribed as a routine post-surgical prophylaxis for stress ulcer.

Conclusion

- Although the incidence of anaphylactic reactions related to ranitidine is low, caution needs to be exercised on administration of this drug. In addition, further study is needed to define strategies for the prevention of adverse drug reactions in hospitalized patients.

Oliva et al, 2008
Case report

- A 31-year-old woman, gravida-1 (23 weeks' gestation), presented with acute severe pain in her left buttock region radiating to the leg and increasing with ambulation.

- No underlying pathologies or drug abuse were present and no systemic symptoms were encountered. Backache was initially attributed to nerve compression. Nonsteroidal anti-inflammatory drugs and rest were prescribed.

- After 4 days, the pain became worse. Temperature: 39.2 °C, pulse rate: 111 beats/minute, respiratory rate: 43 breaths/minute, blood pressure of 100/50mmHg.

- The laboratory test results were significant for leukocytes of 5400/mm3 with left shift (92%), haematocrit of 24%, D-Dimer of 946.8μg/L and platelet count of 85,000/mm3. Chest X-ray showed images of bilateral pulmonary condensation.
The patient was admitted to the intensive care unit with a diagnosis of septic shock and acute respiratory distress. Doppler ultrasound examination of both legs and pulmonary arteriography disproved the diagnosis of pulmonary embolism. An echocardiogram did not find any evidence of endocarditis.

Treatment with broad-spectrum antibiotics (gentamicin and ceftriaxone), inotropic drugs and ventilatory support was prescribed.

Her general status improved throughout the following days. Nevertheless, her back pain became worse.

MRI scan revealed left-sided sacroiliitis with a small abscess at the lower joint margin extending into the iliac notch. A CT-guided aspiration of the abscess was performed and the patient reported partial relief of her symptoms.

Sacroiliac aspiration yielded a small amount of fluid. Although blood cultures were positive for Staphylococcus aureus, culture of the material from the sacroiliac aspiration failed to yield positive results. Intravenous cloxacillin was added to the antibiotic therapy and a rehabilitation programme was initiated so that the patient might recover her strength and mobility.

A new MRI performed 6 weeks later showed progression of sacroiliac joint destruction and focal osteomyelitis (Figure 1). A cesarean section was performed under general anaesthesia at 34 weeks' gestation and a 2570g male neonate was delivered.

The patient is doing well with normal ambulation, although she continues to experience mild discomfort in her left buttock.
Thank you for your attention