Understanding disseminated intravascular coagulation

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Disseminated intravascular coagulation is associated with a high mortality rate, with patient outcomes largely dependent upon swift recognition and appropriate management of events. This article provides a basic overview of the condition, and its pathophysiology, diagnosis, treatment and nursing management, so that effective intervention may be implemented, maximising the chances of patient recovery.

Disseminated intravascular coagulation (DIC) is a haematological disorder characterised by inappropriate, accelerated, systemic activation of the clotting cascade, simultaneously causing thrombosis and haemorrhage (Levi, 2004). The condition is associated with a high rate of morbidity and mortality, but as it is always secondary to a primary disorder, which may itself be associated with a high mortality and morbidity rate, the exact figures are difficult to gauge (Levi, 2004).

The condition is often seen in intensive care units in its acute form as a complication of septic shock and shock states, as well as septicaemia, transfusion reactions and obstetric complications (Levi, 2004; Spence, 2003). The two primary problems caused by DIC are:

- Decreased tissue perfusion due to thrombi, anaemia and hypotension leading to organ ischaemia and necrosis (Adam and Osborne, 1997);
- Haemorrhage, both externally and internally into all body cavities, due to accelerated and inappropriate consumption of clotting factors (Thelan et al, 1998).

Pathophysiology and diagnosis

DIC develops when the body’s ‘normal’ clotting processes are activated systemically and uncontrollably. Coagulation is either extrinsic (caused by tissue injury, resulting in release of tissue factor, tissue thromboplastin, or other procoagulants), or intrinsic (pathway occurs as a result of blood cell injury or platelet contact with collagen in the damaged vessel endothelium – platelets adhere to the collagen surface, triggering clot formation).

Both lead to coagulation with thrombosis (micro-thrombi formation) in vessels and major organs, which may result in pulmonary embolism, thrombophlebitis, cerebrovascular accident and renal failure. Thrombosis formation can continue until the causative factor is corrected or removed. If it is not corrected, clotting continues throughout the body, leading to multi-organ ischaemia, infarction and eventual failure.

Red blood cells become damaged trying to pass through blocked capillary beds, causing excess haemolysis. The continued clot formation uses up the body’s supply of platelets, fibrinogen and other clotting factors, upsetting the balance between coagulation of circulating blood and prevention of haemorrhage.

An intense lysis (breakdown) of clots is also caused by the accelerated thrombosis formation through activation of plasminogen that converts to plasmin then destroys the clot. This leads to the production of fibrin degradation products, which are powerful anticoagulants.

Depletion of clotting products along with the release of these anticoagulants leads to the uncontrolled haemorrhage (Levi, 2004) (Fig 1).

DIC is diagnosed primarily on clinical signs and patient history, with confirmation provided by laboratory findings (Table 1, p40). No single test will confirm DIC (Levi, 2004), but Table 1 includes a guide to the expected values one might expect to see.

Medical management

The primary aims of medical management in DIC are to (Levi, 2004):

- Treat the underlying cause;
- Provide supportive management of complications;
- Support organ function;
- Stop abnormal coagulation and control bleeding.

- Hypotension, hypoxaemia, and acidosis must be corrected, and infection must be prevented and aggressively

LEARNING OBJECTIVES

- Be able to describe the pathophysiological process of disseminated intravascular coagulation (DIC)
- Become aware of the medical management of a patient with DIC and its rationale
- List the signs and symptoms of DIC
- Understand the basic nursing management, assessment and monitoring of patients with DIC and the rationale behind each action
Symptoms of DIC depend on the underlying cause and the predominance of clotting versus bleeding (Levi, 2004). Nurses should be aware of the signs listed in Box 1.

**Nursing management**

It is important to take a thorough history, especially in relation to previous bleeding disorders and any medications that induce bleeding (Murray and White, 1999).

The patient should be turned every two hours to prevent pressure ulcers and to minimise blood stasis and pooling, as this may precipitate clotting and thrombus formation (Hudak et al, 1998). Skin should be thoroughly assessed at least two-hourly for pressure areas and any signs of bleeding, such as petechiae (Woods et al, 2000). A Waterlow or other appropriate scale should be used to identify potential problems (Hudak et al, 1998).

Pressure relieving mattresses reduce the amount of manual handling required and the risk of skin trauma (Adam and Osborne, 1997), while the use of glide sheets reduces the risk of skin shearing.

Skin should be kept clean and moisturised, as dry skin is more easily damaged (Skewes, 1996) and poor hygiene can lead to further infections. Safety devices such as padded side rails should be used where necessary and patients should be assisted out of bed where appropriate (Hudak et al, 1998).

To further reduce the risk of skin damage, sticky tapes should be avoided, and electric rather than blade razors should be used (Woods, et al, 2000). Soft swabs and mild saline solution or water should be used for mouth care, as toothbrushes can damage mucous membranes and small capillaries and alcohol-based mouthwash can cause irritation (Swearingen and Keen, 2001).

Temperature should be monitored and recorded at least four-hourly. It should not be measured rectally due to risk of trauma (Swearingen and Keen, 2001). Excessive pyrexia should be treated with appropriate medications. Light bed clothing and avoidance of an overheated environment are common sense measures. However use of fans, cool compresses and tepid sponges is recommended due to the increased risk of triggering heat-conserving-and-producing mechanisms in the patient.

**REFERENCES**


**CASE STUDY**

George Brown is 64 years old and was admitted to your unit following bowel surgery three days ago. Apart from a mild pyrexia and slightly raised white cell count he had made an apparently good recovery on the ward but deteriorated suddenly overnight, with an increased temperature, cool clammy skin, tachycardia, reduced blood pressure, and cold hands and feet. He also has numerous small bruises and petechiae on his body, which were previously unreported, and there is blood oozing from his abdominal wound, around his peripheral line, and from a small cut on his face, which happened two days ago while he was shaving. Routine blood tests reveal a decreased platelet count, raised PT and APTT, and lowered Hb and haematocrit levels. A provisional diagnosis of DIC secondary to septic shock is made, and further investigations are carried out.

**KEYWORDS**

- Medicine
- Coagulopathy
- Shock
such as vasoconstriction, shivering and goosebumps.

Intravenous access should be maintained at all times for fluid replacement therapy, emergency infusion of blood products and medications (Hudak et al, 1998). Insertion of invasive lines can cause haemorrhage, but is often preferable to numerous venepuncture stabs (Swearingen and Keen, 2001).

If injections are necessary, the smallest gauge needle should be used. Injection sites should be rotated, direct pressure applied for 3–5 minutes (Swearingen and Keen, 2001) and a pressure dressing used. The site should be observed regularly for haemorrhage. If bleeding from cannulation sites continues, haemostatic dressings may be applied (Adam and Osborne, 1997).

An accurate fluid balance record is essential due to the risk of a volume deficit related to bleeding or haemorrhage (Swearingen and Keen, 2001). Wound dressings and linen can be weighed for accurate measurement of blood loss (Woods et al, 2000). Blood pressure should be monitored via an arterial line, as this is less traumatic than frequent cuff inflations, which can lead to further tissue trauma and haemorrhage (Woods et al, 2000).

If the patient is on inotropic support or other medication, the nurse must be aware of the specific observations, assessment, and care appropriate to each drug. Be aware of administering drugs that may precipitate bleeding (Woods et al, 2000) such as aspirin, warfarin and NSAIDs.

### Cardiovacular

Continuous ECG monitoring is necessary to detect dysrhythmias and ST and T-wave changes brought on by decreased myocardial or pulmonary perfusion (Swearingen and Keen, 2001). A 12-lead ECG should be carried out (Adam and Osborne, 1997) for reference, and repeated if wave or rhythm changes are suspected.

Blood pressure, pulse and respiratory rate should be observed at least every 15 minutes to rapidly identify deterioration, and arterial pressure should be observed and kept above 90mmHg systolic to ensure adequate tissue perfusion (Swearingen and Keen, 2001). Fibrin degradation products have a myocardial depressant action and may therefore lead to exacerbated hypotension disproportionate to blood loss (Murphy, 1999).

Tissue perfusion and cardiac output can be assessed through monitoring vital signs, haemodynamic parameters, mental status, urine output and cardiac rhythm (Swearingen and Keen, 2001). Haemoglobin (Hb) should be regularly monitored as haemorrhage may lead to anaemia (Swearingen and Keen, 2001).

Pulmonary artery pressures and central venous pressures should be measured at least hourly as they reflect a patient’s fluid status (Swearingen and Keen, 2001). Hypovolaemia should be rectified promptly to avoid organ damage. Cardiac output, cardiac index, systemic vascular resistance, oxygen delivery and oxygen consumption should be measured every 6–12 hours if a pulmonary artery catheter is in place (Hudak et al, 1998).

Pedal and peripheral pulses, capillary refill, skin temperature and colour should be checked at least every two hours to assess peripheral perfusion, as this can identify the formation of microclots in the peripheral vascular system (Swearingen and Keen, 2001).

### Monitoring respiratory function

Respiratory function requires close continual monitoring as blood loss and pulmonary microemboli may result in reduced oxygenation (Swearingen and Keen, 2001). Pulse oximetry and arterial blood gases should also be monitored regularly, as should respiratory rate, rhythm and effort, the patient’s colour and evidence of cyanosis or shortness of breath (Adam and Osborne, 1997).

Respiratory failure caused by pulmonary shunting, pulmonary haemorrhage, haemothorax, pulmonary embolus, or acute respiratory distress syndrome (ARDS) can occur, as oxygen demand and cellular uptake can be greatly altered (Thelan et al, 1998).

To maintain adequate organ perfusion, respiratory support in the form of supplemental oxygen may be necessary. If this is insufficient to maintain acceptable arterial oxygen levels, artificial ventilation may be required, although this should be used with caution due to the increased risk of haemorrhage (Adam and Osborne, 1997).

Chest physiotherapy may be required to maintain optimal lung function. This may involve regular turning and repositioning, deep breathing and coughing, percussion and vibration, and regular tracheal and oropharyngeal suctioning as indicated by assessment (Wood, 1998). All

### Table 1. Test values expected in DIC diagnosis

<table>
<thead>
<tr>
<th>TEST</th>
<th>NORMAL VALUE</th>
<th>VALUE IN DIC</th>
</tr>
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<tbody>
<tr>
<td>Prothrombin time</td>
<td>11–15 sec</td>
<td>Prolonged</td>
</tr>
<tr>
<td>Activated partial</td>
<td>39–48 sec</td>
<td>Prolonged</td>
</tr>
<tr>
<td>Thrombin time</td>
<td>10–13 sec</td>
<td>Usually prolonged</td>
</tr>
<tr>
<td>Fibrinogen level</td>
<td>200–400mg/</td>
<td>Decreased</td>
</tr>
<tr>
<td></td>
<td>100ml</td>
<td></td>
</tr>
<tr>
<td>Platelet count</td>
<td>150,000–400,000/mm³</td>
<td>Decreased</td>
</tr>
<tr>
<td>Fibrinogen degradation</td>
<td>&lt;10µg/ml</td>
<td>Increased</td>
</tr>
<tr>
<td>products</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasminogen levels</td>
<td></td>
<td>Decreased</td>
</tr>
<tr>
<td>D-dimer assay</td>
<td></td>
<td>Increased</td>
</tr>
<tr>
<td>Antithrombin III</td>
<td></td>
<td>Decreased</td>
</tr>
</tbody>
</table>

Adapted from: Hudak, C. et al, 1998
these procedures must be performed gently to avoid further trauma and haemorrhage.

Suctioning should be carried out with great care and at the lowest possible pressure – research has shown an optimum range of 80–150mmHg (Griggs, 1998) to avoid further haemorrhage. To avoid hypoxia, suction should last no longer than 10–15 seconds (Griggs, 1998).

Monitoring renal function

Fluid intake and output must be recorded accurately at least every 2–4 hours, increasing to every 30–60 minutes if the patient is actively bleeding (Swearingen and Keen, 2001). Oliguria resulting from hypoperfusion of the kidneys may occur due to hypovolaemia. It is therefore essential to maintain an adequate blood pressure.

Renal support such as filtration or dialysis may be required if urine output drops and remains less than 0.5 ml/kg/hour in an adult despite other interventions (Adam and Osborne, 1997).

Regular testing for protein and blood in the urine is important. The kidneys are common sites for microemboli so urea, creatinine and electrolyte levels should also be monitored. Acute renal failure is common due to acute tubular necrosis (Adam and Osborne, 1997).

Daily weights are often useful as an indicator of fluid overload, although other signs such as generalised oedema, periorbital and facial oedema, excessive frothy saliva, rapid dyspnoea, moist rales and cough, or rapid pulse (Hudak et al, 1998) may be more practical in intensive care units.
Monitoring neurological function

Full neurological assessment should be undertaken at least two hourly to check for intracranial haemorrhage, ischaemia and cerebral infarction (Baird, 1997). Pupillary reaction should be recorded in sedated patients (Swearingen and Keen, 2001). When assessing consciousness levels in patients who are unresponsive to verbal stimuli, the use of painful stimuli such as pinching or pressure should be avoided or minimised as it may lead to further haemorrhage. Restlessness, agitation, visual disturbances, headaches and sensory or motor dysfunction can indicate a change in consciousness levels, which may point to intracranial haemorrhage or cerebral ischaemia (Hudak et al, 1998).

Gastrointestinal tract and nutrition

Assess for gastrointestinal bleeding by checking emesis, nasogastric aspirate and stools for blood. Stool softeners may be required to help maintain bowel function. Rectal procedures should be avoided as they may cause unnecessary trauma (Swearingen and Keen, 2001).

If the patient is unable to take an oral diet, then an artificial feeding programme should be started in consultation with the dietitian or nutritional support service (Kennedy, 1997). Enteral feeding via a nasogastric or orogastric tube is usually preferred over parenteral feeding as it helps to maintain mucosal integrity, reduces translocation of gut bacteria, increases blood flow to the gut, and reduces the incidence of septic complications (Thelan et al, 1998). Feeding tubes must be inserted with great care to avoid unnecessary trauma and further haemorrhage.

Infection control

Infection is one of the most common causes of DIC (McCance and Huether, 1998) and is often already present, requiring treatment with appropriate antibiotics. Strict infection control measures and effective handwashing are essential to avoid infection (Thelan et al, 1998). Regular monitoring for infection markers will be necessary, as will extreme care with all invasive equipment and strict aseptic techniques for all wounds (Thelan et al, 1998).

Comfort and pain control

Pain not only brings suffering, but also causes physiological complications such as vasoconstriction and tachycardia (Hudak et al, 1998). Ischaemic pain can occur throughout the body and is caused by obstruction of the microvasculature. Severe abdominal pain is common and may be due to an embolus-induced small bowel infarction, ischaemia or necrosis of the gut, or from haemorrhaging into the retroperitoneal space, which can also cause tingling and numbness due to nerve compression (Hudak et al, 1998).

Joint pain is also due to the formation of thrombi within the joint. A pain scale should be used to assess the patient’s comfort, and appropriate analgesia or sedation should be provided. Measures such as distraction, imagery or relaxation may also be of help (Hudak et al, 1998).

Psychological and social care

The nurse should ensure that rest and sleep are adequate, and that sufficient meaningful sensory stimulation, such as familiar voices, TV and music, is provided, thereby avoiding sensory overload or deprivation (Hudak et al, 1998). The patient should be allowed uninterrupted periods of no less than 90 minutes (an average sleep cycle), with as little disturbance as possible during the night so that normal diurnal patterns may be maintained (Hudak et al, 1998).

It is the nurse’s responsibility to provide reassurance and explanations to the patient and their loved ones on the disease process, diagnostic procedures and planned therapy (Hudak et al, 1998). A trusting, open, honest relationship with good communication will help to allay anxiety. Social services, clergy, interpreters and other community services should be consulted as appropriate for provision of specialist needs (Hudak et al, 1998).

Conclusion

The nurse’s role in caring for the patient with DIC is broad and requires constant assessment and evaluation of physical and mental status, interpretation of all available data, planning, implementation and constant evaluation of appropriate individualised holistic care, as well as an understanding of the pathophysiology of the condition, the signs and symptoms and the various forms of treatment available. The patient and their family will need much support and understanding, as well as education and the encouragement to take an active role in their own treatment and management decisions.