

## Ayurlog: National Journal of Research in Ayurved Science

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### Approach to disseminated intravascular coagulation

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#### Abstract:

Disseminated intravascular coagulation is a hemorrhagic disorder that occurs following the uncontrolled activation of clotting factors and fibrinolytic enzymes throughout small blood vessels, resulting in tissue necrosis and bleeding, also called consumption coagulopathy.

Disseminated intravascular coagulation is caused by a variety of underlying disorders and criteria for diagnosis are not well defined. However, the most helpful are a

#### INTRODUCTION:-

A consensus definition of disseminated intravascular coagulation [DIC] has been proposed as: - It is an acquired syndrome characterized by intravascular activation of

low platelet count, raised APTT and PTINR values. The cornerstone of therapy is prompt treatment of the underlying disease and elimination of the trigger mechanism.

Supportive treatment may include: - Plasma transfusions to replace blood clotting factors if a large amount of bleeding is occurring. Blood thinner medicine [heparin] helps to prevent blood clotting if a large amount of clotting is occurring.

**Key words:** - coagulopathy, clotting factor, platelet count, necrosis, heparin.

coagulation with loss of localization arising from different causes.

DIC is a serious disorder in which the proteins that control blood clotting become over active. It can originate from and cause damage to the microvasculature

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which if sufficiently severe can cause organ dysfunction. DIC is a pathological process characterized by the widespread activation of the clotting cascade that results in the formation of blood clots in the small blood vessels throughout the body.

The pathophysiology of DIC is complex.

### **Causes:-**

When you are injured, proteins in the blood that form blood clots travel to the injury site to help stop bleeding. If you have DIC, these proteins become abnormally active throughout the body. This may be due to inflammation, infection, or cancer. The most common causes of DIC are bacterial sepsis, malignant disorders such as solid tumors or acute promyelocytic leukemia, and obstetric causes which includes abruptio placentae, amniotic fluid embolism, dead fetus syndrome and septic abortion. Trauma, particularly to the brain, can also result in DIC. It has been shown that DIC due to obstetric disorders manifests as bleeding in almost all whereas DIC due to sepsis are mostly associated with organ dysfunction.

### **Clinical features:-**

Clinical presentation of DIC varies with its cause and severity. The major clinical features of disseminated intravascular coagulation are abrupt onset serious bleeding, shock out of proportion to blood

loss, acute renal failure and thromboembolic manifestations. Most patients have skin and mucosal bleeding from multiple sites-venepuncture sites and surgical wounds, oral, nasal, gastrointestinal or urinary tracts. Less commonly patients may have acrocyanosis, thrombosis or gangrene.

### **Pathological investigations:-**

Laboratory tests should be interpreted carefully as levels of many coagulation factors remain elevated in many of the conditions associated with DIC. Common findings include the prolongation of PT and/or APTT, or a rapid decline in platelet numbers. These tests should be repeated over a period of 6-8 hours because an initially mild abnormality can change dramatically in patients with severe DIC. Basic blood examination would show schistocytes on peripheral blood smear in ~50% of the cases which is suggestive of DIC. The most sensitive test for DIC is the FDP level. DIC is an unlikely diagnosis in the presence of normal levels of FDP.

### **Differential diagnosis:-**

The differential diagnosis between DIC and severe liver disease is challenging and requires serial measurements of the laboratory parameters of DIC. Patients with severe liver disease are at risk for bleeding and manifest laboratory features including thrombocytopenia (due to platelet

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sequestration, portal hypertension, or hypersplenism), decreased synthesis of coagulation factors and natural anticoagulants, and elevated levels of FDP due to reduced hepatic clearance. However, in contrast to DIC, these laboratory parameters in liver disease do not change rapidly. Other important differential findings include the presence of portal hypertension or other clinical or laboratory evidence of an underlying liver disease.

### Scoring system:-

The International Society for Thrombosis and Haemostasis (ISTH) DIC scoring system provides objective measurement of DIC. Where DIC is present the scoring system correlates with key clinical observations and outcomes.

Scoring system for overt DIC (should only be used if the patient has an underlying disorder known to be associated with overt DIC:

- Order global coagulation tests (prothrombin time, platelet count, fibrinogen, fibrin related marker) and score the test results:

Platelet count:-

$>100 \times 10^9/L = 0,$

$<100 \times 10^9/L = 1,$

$<50 \times 10^9/L = 2$

Elevated fibrin marker - eg, D-dimer, fibrin degradation products:-

no increase = 0,

moderate increase = 1,

strong increase = 2

Prolonged PT:-

$<3$  secs = 0,

$>3$  but  $<6$  secs = 1,

$>6$  secs = 2

Fibrinogen level:-

$>1$  g/L = 0,

$<1$  g/L = 1

Calculate score:-

$\geq 5$  - compatible with overt DIC: repeat score daily

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<5 - suggestive for non-overt DIC:  
repeat next 1-2 days

### Management:-

The morbidity and mortality associated with DIC are primarily related to the underlying disease rather than the complications of DIC. The control or elimination of the underlying cause should therefore be the primary concern. Patients with severe DIC require control of hemodynamic parameters, respiratory support, and sometimes invasive surgical procedures. Attempts to treat DIC without accompanying treatment of the causative disease are likely to fail. The control of bleeding in DIC patients with marked thrombocytopenia [platelet < 10000-20000] and low levels of coagulation factors will require replacement therapy. The PT [> 1.5 times the normal] provides a good indicator of the severity of the clotting factor consumption and replacement with FFP is indicated. The aim of replacement therapy in DIC is to replenish fibrinogen and is achieved by using cryoprecipitate which contain fibrinogen of ~250mg per bag. Heparin therapy has favorable result in patients with chronic DIC manifesting mainly with thrombosis, but is avoided in

acute DIC with active bleeding. Recombinant human activated Protein C was previously recommended in those with severe sepsis and DIC.

### Prognosis:-

Prognosis varies depending on the underlying disorders, and the extent of the intravascular thrombosis [clotting]. The prognosis for those with DIC, regardless of cause, is often grim: between 10% and 50% of patients will die. DIC with sepsis [infection] has a significantly higher rate of death than DIC associated with trauma.

### Complications:-

Bleeding,  
Lack of blood flow to the arms, legs, or vital organs,  
Stroke.

### Conclusions:-

In conclusion, DIC is categorized into bleeding, organ failure, massive bleeding, and non-symptomatic types. The diagnosis and treatment of DIC should be carried out in accordance with the type of DIC based on the guidelines on DIC.

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