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STUDY OF DIC AS AN OBSTETRIC COMPLICATION AND FETO-MATERNAL OUTCOME

Obstetric and Gynaecology

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KEYWORDS

INTRODUCTION

Disseminated intravascular coagulation (DIC) occurs when the finely controlled process of haemostasis becomes disrupted. As a result, coagulant responses can change from being naturally protective to the host into a maladaptive response with pathological consequences. Clinically, this is reflected in the increased morbidity and mortality that is associated with DIC. The recognition that DIC arises as a complication of different disease states reflects the variety of ways in which clinical events can uncouple normal haemostasis.

In pregnancy however, the rheostat for coagulation is already adjusted to a higher level in order that any excess risk of bleeding is diminished at the mother–baby interface and at parturition.3 The inherent risk of such a physiological response is the increased susceptibility to dysregulation in the event of intercurrent obstetric pathology

The causes of DIC in obstetrics

Table 1

Commonly described causes of DIC in obstetrics

Amniotic fluid embolism
Intrauterine fetal demise
HELLP syndrome
Pre-eclampsia/Eclampsia
Placental abruption
Septicemia
Postpartum haemorrhage
Acute fatty liver

1] Intrauterine foetal demise (IUD) has been classically described as frequently associated with DIC. However, the incidence of undiagnosed IUD is likely to be rare in the well-resourced obstetric environment. This is because spontaneous delivery would usually ensue within the first two weeks whilst marked coagulation disturbances only tend to occur after a month of foetal demise.6 If the foetus were retained longer, up to 25% of cases develop a coagulopathy that is mediated by release of thromboplastin-like material from dead products of conception. These defects may correct spontaneously before evacuation although this can be a slow process.

2] Pre-eclampsia is a significant contributor to maternal as well as neonatal mortality and morbidity. Occurring in 3–5% of all pregnancies, it is considered to be a consequence of an abnormal maternal response to placentation. As such, its clinical severity depends on the extent to which abnormal placental sensing provokes inflammatory signals. The abnormal placentation could be caused by a form of maternal–paternal immune maladaptation that is initiated at the time of semen deposition in the female genital tract. This provokes a cascade of cellular and molecular events resembling a classic inflammatory response. Subsequent increase in syncytiotrophoblast shedding would further augment this inflammatory response. Endothelial dysfunction secondary to an exaggerated maternal inflammatory response towards the trophoblast would result in decreased vasodilator prostaglandins, especially prostacyclin and nitric oxide. This could potentiate platelet aggregation and utero-placental ischaemia to cause preeclampsia.

3] Abruptio placenta also demonstrates the important role played by thrombin, which has potent uterotonic properties in addition to its pivotal role in coagulation. Placentae from women with pre-term labour often demonstrate evidence of old placental bleeding to support the concept of thrombin production in potentiating placental abruption and spontaneous pre-term birth. The consequent haemorrhage upon premature placental separation can also allow entry of placental tissue factor into the circulation to promote thrombin generation and coagulation. The degree of placental separation has been shown to correlate with the extent of fibrin formation and thrombocytopenia, suggesting that coagulation is initiated from the placenta bed.

4] Post-partum haemorrhage (PPH) is another cause of bleeding associated DIC. The coagulopathy arises mainly from the excessive blood loss, consumption of clotting factors and the further effects of massive transfusion in the setting of acidosis and hypothermia.

5] The syndrome with haemolysis, elevated liver enzymes and low platelets (HELLP) is characterised by prominent endothelial cell damage within the liver. It may be considered as a placenta-mediated and liver-targeted acute inflammatory condition whereby Fas-dependent apoptosis of hepatocytes has been observed. Endothelial dysfunction, platelet and complement activation with release of inflammatory mediators are the different factors that predispose towards DIC in this condition.

6] Another liver-specific but mechanistically distinct condition is acute fatty liver of pregnancy (AFLP). This is usually seen in the third trimester of pregnancy and is often fatal. A genetic deficiency of fatty acid beta oxidation has been described in its pathogenesis and the coagulopathy is mainly caused by severe hepatic dysfunction. Notably, there is marked antithrombin deficiency in AFLP to further drive procoagulant changes and DIC.

Pathophysiology of DIC

It is important to understand the normal coagulation process in order to characterise the abnormalities observed during DIC. The coagulant response begins with exposure of tissue factor (TF) and binding of factor VIIa to activate factor X for conversion of prothrombin to thrombin (Fig. 1). Thrombin generation is further propagated through the intrinsic pathway and the explosive burst of thrombin results in cleavage of fibrinogen into fibrin. Clot formation is homeostatically regulated to achieve the desired haemostatic effect and this involves a number of regulatory responses that are spatially and temporally differentiated. Normal endothelium at the margins of injury switches from thrombin procoagulant to anticoagulant activity via its binding to the endothelial receptor, thrombomodulin (TM). The thrombin–TM complex activates protein C bound to the endothelial protein C receptor (EPCR). The generated activated protein C (APC) degrades activated factor V and VIII with co-factor support from protein S to inhibit further clot formation. Other important anticoagulants involved are antithrombin and tissue factor pathway inhibitor (TFPI). The former inactivates thrombin and factor Xa whilst TFPI forms a quaternary complex with tissue factor, factor VIIa and Xa to inhibit the cascading effect towards thrombin generation. Normal clot formation is followed by its own regulated dissolution. This process of fibrinolysis involves thrombin-induced tissue plasminogen activator (tPA) dependent generation of plasmin from plasminogen. Regulation of fibrinolysis is mainly through plasminogen activator inhibitor (PAI)-1 and thrombin activatable fibrinolysis inhibitor (TAFI). PAI-2 is also involved physiologically in pregnancy.

Central to the development of DIC is the excessive generation of thrombin in vivo. Whilst thrombin generation is generally dependent on prothrombinase complex assembly on platelet surfaces.cell free phospholipid can also support such reactions in vivo. These form as a result apoptosis or damaged cell membranes to externalise the inner

leaflet and expose PS. Microparticles which carry the externalised PS are generally procoagulant and their circulating levels increase in pregnancy.

DIAG.

AIMS AND OBJECTIVES

To study the antecedent factors, morbidity,mortality associated with disseminated intravascular coagulation.

MATERIALS AND METHODS

The study was conducted in the department of Obstetrics and Gynaecology, Civil hospital, B.j medical college, Ahmedabad.

Study Design : it was retrospective observational study of 20 cases of DIC carried out between period of June 2020 to December 2020 which includes all postpartum women who gave birth during study period at Civil hospital Ahmedabad.

The presence of overt DIC was determined using two scoring system 1] International society of thrombosis and haemostasis scoring system (ISTH)

Which assigns points on basis of decrease platelet counts, prolonged Prothrombin time, elevated fibrin related markers and fibrinogen level 2] obstetrical DIC severity staging system.

Maternal outcomes included massive blood transfusion(>5 units), hysterectomy, admission to ICU, acute tubular necrosis requiring dialysis and death.

Neonatal outcome included birth weight, NICU admission, and death. Treatment of DIC was assessed by blood products administered, postpartum haemorrhage management and laboratory massurements.

International society on thrombosis and haemostasis diagnostic scoring system for overt DIC

1. Risk assessment: Does the patient have an underlying disorder known to be associated with overt DIC?
2. If yes: proceed
3. If no: do not use this algorithm

2. Order global coagulation tests (prothrombin time, platelet count, fibrinogen, fibrin related marker)

3. Score the test results

Platelet count- >1,00,000 = 0
< 1,00,000 =1
< 50,000 =2

Elevated fibrin marker (fibrin degradation product)

No increase =0
Moderate increase =2
Strong increase =3
Prolonged prothrombin time
<3 second = 0
>3 second but < 6 second =1
> 6 second =2

Serum fibrinogen level
>1 g/L =0
<1g/L = 1

Calculate score:

>5 compatible with overt DIC
<5 suggestive of non overt DIC

DIC severity staging in obstetrics

Severity of DIC	findings
Stage-1: low grade DIC	Increase FDP, decrease platelet
Stage-2: uncompensated but no haemostatic failure	Marked decrease in platelet count, and decrease in fibrinogen
Stage-3: uncompensated DIC + haemostatic failure	Marked decrease in platelet and coagulation factor and increase level of FDP

RESULT:

Conditions associated with DIC

Condition	Cases with DIC	Percentage
Abruption	4	20
Pre-eclampsia	5	25
PPH	4	20
Septicemia	2	15
Jaundice	1	5
IUD	2	10
HELPP	2	10

In this study, it was observed that pre-eclampsia had the highest association with the incidence of DIC i.e. 25% which accounts of 5 cases out of 20 cases of DIC. Other causes showing a significant association with DIC are abruption (20%), PPH (20%) and septicemia (15%).

Mode of Delivery:

Mode of delivery	Cases	Percentage
Normal Delivery	6	30
Assisted Delivery	2	10
Cesaerian section	12	60

On retrograde study it was found that out of 20 cases of DIC , highest association was seen in operative deliveries of upto 60% (12/20 cases).

Maternal outcomes:

Morbidity	Cases	Percentage
ICU admission	17	85
Ventilatory support	12	60
AKI, MODS and dialysis	7	35
Massive blood transfusion	14	70
Hysterectomy	3	15
Maternal death	8	40

Majority of the females suffering from DIC, undergo ICU admission (85%) and require ventilator support (60%) and massive blood transfusion (>5 units) (70% cases).

Neonatal outcomes:

Outcome	Cases	Percentage
IUD	2	10
NICU admission	5	25
Low birth weight	4	20
Total neonatal mortality	3	15

It was observed that, 25% of cases were associated with NICU admission and a total mortality of 15% which is suggestive that along with a poor maternal outcome, DIC also increases morbidity of the newborn.

Severity of DIC by score in obstetric cases:

(according to ISTH system)

Severity score	Cases	Percentage
<5(Non-overt)	11	55
>5(overt)	9	45

DISCUSSION:

A retrospective database study of 6 months identified 20 cases of DIC according to which the incidence rate was 0.6%. When examined with context of various obstetrical condition, highest association was seen with pre-eclampsia, placental abruption and PPH.

DIC was diagnosed by using DIC scoring system proposed by ISTH. It is mainly based on PT-INR, D- dimer, fibrinogen and platelet count. The other approach to determining the severity of DIC is to use marker of maternal morbidity such as massive blood transfusion, hysterectomy, ICU admission and MODS.

Our study highlights that high level of maternal perinatal morbidty and mortality is associated with DIC.

CONCLUSION:

DIC, as a marker of severe obstetrical complications, is associated with

high levels of mortality and morbidity. Recognition of the antecedent causes and early investigation for and active management of DIC may help lower this morbidity. Despite its rarity, systematically searching for DIC should be added to treatment algorithms in the management of known obstetrical antecedents, because treatment delays may significantly worsen prognosis. In particular, the prompt management and arrest of postpartum hemorrhage before the need for massive transfusion and its attendant coagulopathy is one of the lessons from this study.