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Correlationof Obstetric Complications andNeonatal Bleeding

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Abstract: *Bleeding disorders may present during the neonatal period. However,absent patient history along with unique physical signs, physiologically decreased levels of plasma proteins and laboratory variations of platelet function tests leads to difficulty in diagnosis.*
Aims and objective*: The aim of study is to know the etiology of neonatal bleeding at our centre and its relation with obstetric complications.*
Material and Methods*: This prospective study was carried out in department of paediatrics, Patna medical college and hospital, Patna from june 2012 to September 2014.One hundred ten neonates having complain of bleeding were included in this study.*
Result*: Out of 110, 43 (39.09%) neonates were preterm and 67(60.90%) were term. Most of neonates (43.63%) were low birth weight between1.5-2.499 kg. Obstetric complication was present in 36 neonates and most common complication was pregnancy induced hypertension (14.54%) followed by anaemia (10.90%) and then APH (1.81%). Intranatal complication was seen in 30 (27.27%) cases in which birth asphyxia (13.63%) was most common followed by birth injury (8.18%). In our study most common cause of neonatal bleeding was septicaemia with DIC (38.18%) followed by VKDB(18.18%), birth asphyxia(16.36%), birth injury(10.90%), malrotation with volvulus and neonatal hepatitis (3.63%). Coagulation defect was present in 2.72% of cases. Infection with klebsiella was found in 24 (53.3%) neonates. Pseudomonas was seen in 17.7% of cases while coagulase negative staphylococcus was present in 15.5% .Gastrointestinal tract(GIT) was most common site of bleeding in neonates in our study. Intracranial bleeding was present in 13.63%, skin in 11.81% and pulmonary in 9.09% of cases.*
Conclusion:*Good obstetric care andvitamin k prophylaxis at birth can prevent neonatal bleeding.*
Keywords*:Pregnancy induced hypertension, Obstetric care, Septicaemia, Birth asphyxia*

I. Introduction

Bleeding disorders may present during the neonatal period. However,absent patient history along withunique physical signs, physiologically decreased levels of plasma proteins and laboratory variations ofplatelet function tests leads to difficulty in diagnosis. Symptoms like cephalhematomas, bleeding following invasive procedures, facial purpura following birth indicate the presence of a bleeding disorder and usually associated with severe platelet dysfunction or thrombocytopenia.[1]Vitamin k deficiency bleeding VKDB was previously known as haemorrhagic disease of new born is significant bleeding due to newborns inability to sufficiently activate vitamin Kdependent coagulation factor [2].

Early VKDB is manifested in neonates born to mothers taking anticoagulantsor anticonvulsants which antagonize vitaminK. If mother is not monitored properly, umbilicus haemorrhage, cephalhaematoma, haemorrhage fromvarious systems occur at birth. Classic VKDB is manifested on the 3rd day of life, and occurs in cases mother do not provide a sufficient amount of vitamin K to the neonate.The defect is more profound in breast-fed babies.Late VKDB occurs in breast-fedneonates who have not received vitamin K at birth. Haemostasis testing reveals prolonged PT, APTT, but normal TT. The prolonged PT in neonates reflects decreased plasma concentrations of vitamin Kdependantfactors, whereas the prolonged PTT stems from decreased plasma levels of contact factors as well. [3,4]Neonatal Sepsis, birth asphyxia, birth trauma and severe RDS may causes variable degree of disseminated intravascular coagulation(DIC)leads to thrombocytopenia and neonatal bleeding [5]. Neonatal platelets were found to be hypo reactive due to decreased receptors, deficient thromboxane synthesis and impaired signal transduction [6]Pregnancy induced hypertension(PIH) affects the foetus and newborn in several ways. These effects include an increasedrisk of neonatal mortality and morbidity, IUGR, premature birth, necrotizing enterocolitis (NEC), bronchopulmonary dysplasia, and haematological abnormalities such as thrombocytopenia, polycythemia,and neutropenia. [7,8]

II. Material and methods

The study to evaluate impact of obstetrics on neonatal bleeding is a prospective study done in department of pediatrics Patna medical college and hospital, Patna during period of October 2012 to September 2014. The aim of study is to know the etiology of neonatal bleeding at our centre and its relation with obstetric complications.Neonates admitted with bleeding manifestation or those who developed bleeding during their neonatal intensive care unit(NICU) stay were included in this study. Neonates admitted in NICU due to septicemia irrespective of their gestational age,place of birth, sex andbirth weight were control.Family history of bleeding disorder either in the sibling or in the relatives was noted. Detailed antenatal history of maternal age, parity, blood group, maternal medication, maternal SLE/ ITP was taken. Obstetric complicationlike pregnancy induced hypertension, diabetes,ante parturmhemorrhage, intrauterine growth restriction and premature rupture of membrane was noted. Intranatal history regarding gestational age, place of delivery, mode of delivery, prolonged or difficult vaginal delivery, instrumental delivery and meconium stained liquor was taken. History regarding delayed cry or birth asphyxia was taken. Birth weight and site of bleeding was noted.

After history thorough clinical assessment was done to exclude any congenital abnormality, hemangioma and vascular malformation. Pallor, icterus, petechiae and purpura was noted. Systemic examination was done. Routine blood investigation like CBC, RBS, peripheral blood and blood culture was done in every patients. Bleeding time(BT), clotting time(CT), prothrombin time (PT), activated partial thromboplastin time(aPTT) werecalculatedin every neonates. TORCH screening and fibrin degradation product FDP was done in selective cases.In case of hematemesis in newborn, theApt- Downey test was performed to differentiate between maternal and fetal blood in gastric aspirate. Positive test indicates maternal blood and neonates with positive test were excluded from study.Treatment was started with antibiotic and vitamin k from begning. Cases were individualized and blood, fresh frozen plasmaFFP, and platelets was transfused according to need. Follow upwas done at 1month, 3month and 6month.

III. Result

A total of 110 neonates were included in studygroup and30 were in control during study periods. Out of 110, 43 (39.09%) neonates were preterm and 67(60.90%) were term. Most of neonates (43.63%) were lowbirth weight between1.5-2.499 kg.Obstetric complication was present in 36 neonates and most common complicationwas pregnancy induced hypertension (14.54%) followed by anaemia (10.90%) and then APH (1.81%). Intranatal complication was seen in 30 (27.27%) cases in which birth asphyxia (13.63%) was most common followed by birth injury (8.18%).In our study most common cause of neonatal bleeding was septicaemia with DIC (38.18%) followed by VKDB(18.18%), birth asphyxia(16.36%), birth injury(10.90%), malrotation with volvulus and neonatal hepatitis (3.63%). Coagulation defect was present in 2.72% of cases. Blood culture was done in all patients. Infection with klebsiella was found in 24 (53.3%) neonates. Pseudomonas was seen in 17.7% of cases while coagulase negative staphylococcus was present in 15.5% .Gastrointestinal tract(GIT) was most common site of bleeding in neonates in our study. Intracranial bleeding was present in 13.63% , skin in 11.81%and pulmonary in 9.09% of cases.

Table 1: Bleeding neonates and birth weight

Weight(kg)	Number	percentage
< 1.499	22	20%
1.5-2.499	48	43.63%
> 2.5	40	36.36

Table2: Gestational age distribution

Gestational age	Number	percentage
Preterm	43	39.09%
Term	67	60.90%

Table3: Antenatal factor

Antenatal factor	Preterm	Term	Number	Percentage
PIH	12	04	16	14.54%
APH	03	00	03	2.72%
DM	00	02	02	1.81%
IUGR	00	01	02	1.81%
Epilepsy	00	02	01	0.90%
Anemia	06	06	12	10.90%

Table4: Intranatal risk factor

Intranatal factor	Preterm	Term	Number	Percentage
Birth injury	02	07	09	8.18%
Birth asphyxia	01	14	15	13.63%
PROM	04	02	06	5.45%

Table 5: Etiology

Etiology	preterm	Term	Number	Percentage
VKDB	4	16	20	18.18%
Septicemia with DIC	32	10	42	38.18%
Birth asphyxia	03	15	18	16.36%
Birth injury	03	09	12	10.90%
GI perforation	01	04	05	4.54%
Malrotationwith volvulus	00	04	04	3.63%
Coagulation defect	00	03	03	2.72%
NAIT	00	02	02	1.81%
Neonatal hepatitis	00	04	04	3.63%

Table 6: Blood culture in septicemic neonates

Organism	Number	Percentage
Klebsiella	24	53.3%
Pseudomons	08	17.7%
E.coli	03	6.6%
Fungus	03	6.6%
Coagulase-negative Staphylococcus	07	15.5%

Table 7: Site of bleeding in different etiology

Site of bleeding	VKDB	Sep DIC	BA DIC	Coag.defect	Birth injury	GI perforation	Malrotation	NAIT	Neo hepatitis	Number	Percentage
GIT	12	25	12			5	4	2	1	61	55.45%
Umbilicus	1			3						4	3.63%
Skin	3	8			1				1	13	11.81%
Intracranial	3		10						2	15	13.63%
Subgaleal					6					6	5.45%
Pulmonary		10								10	9.09%
Genitourinary		1								1	.90%
Intra abdominal					1					1	.90%

Table 8: Mortality in relation to etiology

Etiology	Number	Death	Percentage
Septicemia with DIC	42	25	59.52%
Birth asphyxia with DIC	18	05	27.77%
VKDB	20	02	1%
Birth injury	12	02	16.66%
Neonatal hepatitis	04	01	25%

Table 9: Follow up at 6 month

Outcome	number	Percentage
Immediate Death	35	31.81%
Lost to follow up	21	19.09'%
Developmental delay	07	6.36%
Coagulation defect	03	2.72%
Normal	44	40%

IV. Discussion

The neonate is born with a combined deficiency in plasma coagulation factors, natural and inhibitors of haemostasis and components of the fibrinolytic system. In the healthy neonate, there is a balance between haemostatic systems, therefore under normal circumstances, the healthy neonate does not present haemorrhage or thrombosis. However several coagulation disorders, congenital or acquired, may be expressed during the neonatalperiod in a healthy or diseased neonate.In our study approx 60% neonates were term. Most of them were low birth weight(43.63%) which was also found in some study.[2]In our study most common obstetric complication associated with neonatal bleeding was PIH. The pathogenesis of thrombocytopenia among infants born to mothers with PIH is presently unknown and a topic of current research.[9,10]. The severity of neonatal thrombocytopenia is highly variable, some neonates develop severe or clinically significant thrombocytopenia (<50,000/μL) and persistent thrombocytopenia which result in bleeding. Thrombocytopenia is generally identified at birth orwithin the first 2 to 3 days following delivery, with resolutionby 7 to 10 days of life in most

cases.[11]. A recent prospective study of neonates with severe thrombocytopenia found that 91% of neonateswhose platelet counts 20 $x10^9$/L did not developmajor haemorrhage, suggesting that this is a reasonably safethreshold for platelet transfusion for most neonates and exchange transfusion is necessary ifplatelet count is less than 100,000/µL.,[12]

Birth asphyxia and birth injuries were common intranatal cause of neonatal bleeding in our study like some other studies [13, 14].. In our study most common cause of neonatal bleeding was septicaemia with DIC (38.18%) followed by VKDB(18.18%), then neonatal hepatitis and NAIT.Septicaemia causes DIC which leads to haematological abnormality like thrombocytopenia [5,15].Vitamin K deficiency bleeding occurs due to endogenous and exogenous deficiency of vitamin k [2]. There are three mode of vitamin k administration ,intramuscular, oral and intravenous. Intramuscular route is preferred [16].Oral route may be alternative where parents refuse intramuscular route [17].So, to prevent early VKDB administer vitamink to pregnant women who is on anticonvulsantor anticoagulant.Also administer vitamin k1 within 6 hours of birth prophylacticaly as a routine to prevent classic and late VKDB.In our study blood culture wasdone in all neonates. Culture was positive in 50% of cases in which klebsiella was found in 24 (53.3%) neonates. Pseudomonas was seen in 17.7% of cases while coagulase negative staphylococcus was present in 15.5% cases.This finding is similar to other study which showed Klebsiella pneumoniacommonest organism followed by Staphylococcus aureusand Pseudomonasin India[18,15].NAIT is the common cause of intracranial haemorrhage in newborn infants [19] and iscaused by transfer of maternal antibodies raised against alloantigen (most commonlyHPA-1a and HPA-5b) carried on fetal platelets.[20]. Infants with NAIT can present with severe bleedingmanifestations in the hours to days following birth and can have severely low platelet counts(< 10k/uL). The treatment for affectedneonate with bleeding or severe thrombocytopenia (<30,000/uL) is typically transfusion ofABO compatible random donorplatelets in addition to IVIG.[21].

In this study mostcommon site of bleeding was GIT especially among neonates with DIC due to septicaemia and birth asphyxia. In this study percentage ofGIT bleeding is more high as some studies showed upper GI bleeding occurs in 10 to 40%of neonates, especially among those suffering from infections, preterm birth,thrombocytopenia and birth asphyxia [22,23]. Second most common site of bleeding was intracranial haemorrhage and mainly present in birth asphyxia. This is explained by some studythat birth asphyxia causesneonatal thrombocytopenia, which is associated with an increased risk of pulmonary, gastrointestinal, and intraventricular haemorrhage (IVH). [24].In our study neonatal mortality wasseen in 31.81% of cases and59.25% neonate were died due to septicaemia and 27.77% were that of complication of birth asphyxia. This finding is supported by some other studies [15,25,26]. Follow up done at 1 month,3month and 6 month. At six month 40% babies were achieved normal milestones and 6.16% were showed developmental delay.Only in 3 (2.72%) neonate coagulation defect was present,two had family history of haemophilia and one had factor xiii deficiency.

V. Conclusion

In our study common causes of neonatal bleeding were VKDB, septicaemia, birth asphyxia and commonly present in low birth weight and IUGR babies.PIH was most common antenatal factor associated with neonatal bleeding. Regular ANC should be done to detect obstetric complications like PIH and any congenital coagulation defect in utero. Proper diet rich in vitamin k, iron and calcium should be supplemented during pregnancy. Full aseptic and antiseptic precaution should be taken during birth of baby. Labour should be monitored by cardiac tocography (CTG) to avoid birth asphyxia and vitamin K prophylaxisis mandatory at birth. Any high risk pregnancy managed by senior obstetrician. This study was done on small population which is the limitation of our study.

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