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ORIGINAL RESEARCH

A study on clinical profile of non traumatic intracranial hemorrhage in children in tertiary care hospital

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ABSTRACT

Introduction: Intracranial bleeding is abnormal accumulation of blood inside the vault of cranium it may occur inside the brain parenchyma as intracerebral bleeding or covering the meningeal space Intra cranial hemorrhage is rare among children but often disabling disease leading to high morbidity and mortality.

Aim: To study the etiology, clinical profile, laboratory and radiological findings of non-traumatic intracranial bleed in children.

Material and methods: Prospective observational study ,The study was conducted in the Department of Paediatrics, Niloufer hospital from November 2019 - November 2021.35 Children were included in this Study. Study was based on the child's clinical presentation, the cause of ICH as well as radiology criteria of hematoma.

Results: The outcome after the intracranial bleeding depends on many different factors such as size and localization of hemorrhage as well as the clinical status at the time of presentation. Intra cranial bleed due to bleeding diathesis has better outcome because of appropriate diagnosis and treatment of the underlying disease, in addition to early surgical intervention when indicated. Study showed a higher frequency of complex chronic illness as risk factor for paediatric ICH. The mortality due to paediatric ICH remains high but risk of death may reflect the underlying risk factors for intracranial hemorrhage and not just the risk from the hemorrhage itself.

Conclusion: All cases of liver failure should be monitored with PT, APTT and INR, timely vitamin K should be given to prevent ICH.

Keywords: Intra cranial haemorrhage, Non-traumatic intracranial bleed, mortality

INTRODUCTION

Intracranial bleeding is abnormal accumulation of blood inside the vault of cranium it may occur inside the brain parenchyma as intracerebral bleeding or covering the meningeal space. It can occur in any form like bleeding inside meninges or related spaces, epidural hematoma, subduralhematoma and bleeding inside ventricles and subarachnoid bleeding.[1]

Intra cranial hemorrhage is rare among children but often disabling disease leading to high morbidity and mortality.The incidence and prevalence of intracranial hemorrhage is notknown. The reported incidence of asymptomatic and symptomaticintracranial hemorrhage varies from study to study probablydue to differences in populations studied and differences inthe sensitivity and timing of diagnostic imaging used.[2]

Focal abnormality of brain functions from ICH are site specific and includemotor deficit, sensory deficit, speech problems, cranial nerves palsies, cerebellar manifestations, visual

abnormalities and pupillary changes. Irritability and fits may occur in about 6–9% of intracerebralhemorrhage. The hemorrhage may expand within minutes or few hours and act as a solid mass, increasing the intracranial pressure.Computed tomography (CT), Magnetic resonanceimaging(MRI), Conventional angiography and Computed tomographyangiography (CTA) or Magnetic resonance angiography(MRA) may be needed to establish the diagnosis of intracranialvascular anomalies.In cases of bleeding in children,the coagulation profile should be checked to exclude coagulationdisorders and DIC that may develop as a result of thromboplastinrelease from the damaged brain tissue.Management of ICH in children depends on the location ofhemorrhage, the volume of the hematoma, the presence ofmass effect, the clinical condition of the patient as well asthe etiological factors involved in the bleeding .[3]

Intra cranial hemorrhage is rare among children but often disabling disease leading to high morbidity and mortality. Thecommon causes of intracranial hemorrhageare vascular malformations, aneurysms, cavernous angiomas. The available literature stresses upon the vascular causes and its clinical profile. The other set of causes are left unexplored. The present study was taken up with the objective of exploring the etiology of nontraumatic intra cranial hemorrhages and its clinical presentation.

MATERIAL AND METHODS

Hospital based prospective observational study was conducted in the Department of Paediatrics, Niloufer hospital, affiliated to Osmania Medical College. It is the largest tertiary care center in the state of Telangana, situated in the heart of Hyderabad from November 2019 - November 2021. 35 Children who were admitted in Intensive Care Unit of Niloufer hospital during the study period.

Children withnontraumatic intra cranial bleed satisfying the inclusion criteria were enrolled into the study and admitted after getting informed consent from the parents/guardians.

Inclusion Criteria: Age group between 1 month – 12 years Children with nontraumatic intracranial bleed

Exclusion criteria: Children / Infants with traumatic intra cranial bleed, Children / Infants with hemorrhagic transformation of venous infarct.

-Children withnontraumatic intra cranial bleed satisfying theinclusioncriteria were enrolled into the study and admitted after getting informed consent from the parents/guardians were enrolled into the study.

A detailed history for every case was taken from the parent/guardian.Physical examination was conducted and significant findings were noted.The child/infant was sent for the lab investigation to know the etiology.All basic Investigations were done

The data was entered in Microsoft Excel 2010 version. Data was analysed using Microsoft Excel 2010 and Epi Info 7.2.0. Descriptive and inferential statistical analysis were used in the present study. Results on continuous measurements were presented on Mean±SD (Min-Max) and results on categorical measurements were presented in Number (%). Significance was assessed at 5% level of significance. Student t-test is used to compare inter group variation for continuous variables. PearsonsCorrelation Co-efficient was used to assess the relationship between the two variables.

Ethical clearance was obtained from the Institutional Ethical Committee, Osmania Medical College, Hyderabad.

RESULTS

The study was conducted in the Department of Paediatrics, Niloufer hospital, affiliated to Osmania Medical College with an objective of studying the etiology, clinical profile, laboratory and radiological findings of non-traumatic intracranial bleed in children.
The results of the study are as follow:

Table-1:Showing the demographic distribution of study population:

Age group	**Frequency**	**Percent**
Less than 1 year	18	45%
1-5 years	4	10%
6 – 10 years	10	25%
11-12 years	8	20%
Total	40	100.00%
Gender		
Female	16	40%
Male	24	60%
Type of feed		
Exclusively breast fed	32	80%
Formula/Top feeds	8	20%
Type of Extracranial bleeding manifestations		
Ecchymotic patches	5	45.45%
Petechiae	3	27.27%
Joint hematoma	2	18.18%
Bleeding from the mouth	1	9.09%
Total	11	100.00%

Among the study population, majority of cases 45% (n =18)were in age group of less than one year, followed by25%(n=10) 6-10 years,20%(n=8)11-12 years. 10%(n=4) were in the age group of 1-5 years.60%(n=24) were malechildren, 40%(n=16)were female children.80%(n=32) were exclusively breast fed, 20%(n=8) were on top feed.Among the patients who had bleeding manifestations, ecchymotic patch was seen in 45.45%(n=5). 27.27%(n=3) had petechiae, 18.18%(n=2) had joint hematoma, 9.09%(n=1) had bleeding from the mouth.

Table -2: Showing frequency of signs and symptoms in the study population.

Seizures	**Frequency**	**Percent**
Absent	22	55%
Present	18	45%
Bleeding manifestations		
Absent	29	72.5%
Present	11	27.5%
Headache		
Absent	21	52.5%
Present	19	47.5%
Vomitings		
Absent	12	30%

Present	28	70%
Altered Sensorium		
Absent	11	27.5%
Present	29	72.5%
Refusal to feed		
Absent	28	70%
Present	12	30%
Incessant cry		
Absent	30	75%
Present	10	25%
Pupillary size	**Frequency**	**Percent**
Normal	33	82.5%
Anisocoria	**7**	**17.5%**
Pupillary reflexes		
Normal	35	87.5%
Absent (nonreactive pupils)	5	12.5%
Deep Tendon Reflexes		
Normal	32	80%
Increased	6	15%
Decreased	2	5%
Muscle tone		
Normal	32	80%
Increased	6	15%
Decreased	2	5%
Jaundice		
Present	3	7.5
Absent	37	92.5
focal neurological defects		
Absent	31	77.5%
Present	9	22.5%

Among the study population, 45% (n=18) had seizures(focal seizures more than generalized seizures).Extra cranial bleeding manifestations were present in 27.5%(n=11) of the study population.47.5%(n=19) had headache,70%(n=28) had vomiting,72.5%(n=29) presented with altered sensorium, 30%(n=12) presented with refusal to feed and 25%(n=10) had inconsolable cry.

Among the study population, pupils were abnormal(anisocoria) in 17.5%(n=7), Around 82.5%(n=33) had normal pupillarysize.12.5%(n=5) had a nonreactivepupils, 15%(n=6) had increased/exaggerated reflexes. Around 5%(n=2) had sluggish reflexes,deep tendon reflexes were normal in 80%(n=32) and Muscle tone was increased in 15%(n=6) and decreased in 5%(n=2). Muscletone was normal in 80%(n=32), jaundice was present in 7.5%(n=3).Focal neurological defects were found in 22.5%(n=9) of the study population.

Among the study population, aphasia, blurring of vision, right side weakness were present in 7.5% (n=3)each. 5%(n=2) had left side hemiparesis.Neck rigidity seen in2.5%(n=1).

Table-3: showing the investigations in the study population:

LFT parameter	Frequency	Percent
Total Bilirubin	16	40%

Direct Bilirubin	5	12.5%
Indirect bilirubin	11	27.5%
SGOT/AST	5	12.5%
SGPT/ALT	5	12.5%
Alkaline Phosphatase	10	25%
Total proteins	3	7.5%
Parameter		
Prothrombin time	17	42.5%
Activated Plasma Thrombin time	23	57.5%
INR	23	57.5%
Mode of radiological imaging		
NSG	18	45%
CECT	40	100%
MRI	30	75%
MRA	30	75%
MRV	30	75%

Among the study population, 40% had raised total bilirubin levels. 27.5% had raised indirect bilirubin levels, Alkaline phosphatise was raised in 25%, Direct bilirubin, SGOT, SGPT were raised in 12.5% each Total proteins were altered in 7.5%. Abnormalities in PT were observed among 42.5%, Activated plasma thrombin time and INR in 57.5%.45%(n=18) were subjected to NSG, CECT was done in all the patients100%(n=40). MRIandMRA,MRVwere done in 75%(n=30).

Table-4: showing the frequency of the types of intracranial haemorrhage.

Types of hemorrhage	**Frequency**	**Percent**
ICH(Intracerebralhemorrhage)	22	55%
ICH,SAH(sub arachnoid hemorrhage)	3	7.5%
ICH,IVH(intra ventricular hemorrrhage)	2	5%
IVH(intra ventricular hemorrrhage)	1	2.5%
SAH(sub arachnoid hemorrhage)	1	2.5%
SDH(sub duralhemorrhage)	11	27.5%
location of hemorrhage		
Right fronto-parietal region(SDH)	5	12.5%
Left capsulo-ganglionic region	5	12.5%
Right temporal lobe	4	10%
Left parieto-temporal SDH	3	7.5%
Right frontal lobe	2	5%
Left parieto-occipital lobe	2	5%
Left frontal lobe	2	5%
Left fronto-parietal SAH+ left parietal lobe	2	5%
B/loccipito-parietal region SDH	2	5%
Left temporal lobe	2	5%
Left thalamus	2	5%
4th ventricle	1	2.5%
Left parietal lobe	1	2.5%

Left fronto-parieto temporal SAH	1	2.5%
Right temporal SAH +Right temporal lobe	1	2.5%
B/lfronto-temporo-parietal region	1	2.5%
Right cerebellar	1	2.5%
Left frontal lobe	1	2.5%
Right thalamus, lateral and third ventricle	1	2.5%
Right thalamus	1	2.5%
Right thalamus, lateral ventricles	1	2.5%

Among the study population, ICH was present in 55%(n=22),ICH and SAH were present in 7.5%(n=3). ICH and IVH were present in 5%(n=2). SDH was present in 27.5%(n=11). SAH was present in 2.5%(n=1). IVH was present in 2.5%(n=1)

Table-5: showing the findings in the study population:

Bone marrow Aspiration	**Frequency**	**Percent**
Bone marrow aspiration done	6	15%
Normal	3	7.5%
Lymphoblasts s/o ALL	2	5%
Reduced marrow cellularity	1	2.5%
Investigations		
Factor VIII(<1%)	4	10%
Factor IX(<1%)	2	5%
Gastric aspirate for CBNAAT +ve	1	2.5%
Urine PCR for CMV	1	2.5%
Etiological diagnosis	Frequency	Percent
LHDN	14	35%
Arteriovenous Malformation	5	12.5%
Haemophilia -A	4	10%
ITP	3	7.5%
Haemophilia-B	2	5%
ALL	2	5%
Moyamoya disease	2	5%
Aplastic anaemia	1	2.5%
Liver failure, idiopathic	1	2.5%
Liver failure - Biliary Atresia	1	2.5%
Liver failure CMV hepatitis	1	2.5%
Medulloblastoma	1	2.5%
Idiopathic	1	2.5%
Sickle Cell Anemia	1	2.5%
TBM	1	2.5%

Among the study population, bone marrow aspiration was done in 15%(n=6)cases to rule out hematogical disorders. Among these15%(n=6), 7.5% (n=3)bone marrow was normal,ALL were noticed in 5% (n =2), marrow cellularity was reduced in 2.5%(n =1).
Among the study population,10%(n=4)had low levels offactor VIII(<1%),
5%(n=2) had low levels of factor IX(<1%).
2.5%(n=1) had positive Urine PCR for CMV,

CBNAAT was positive in 2.5%(n=1).
Among the study population, 35%(n=14) had hemorrhagic disease of newborn(late type idiopathic HDN),12.5%(n=5) had arteriovenous malformation.Hemophilia A contributed to 10%(n=4),HemophiliaB contributed to 5%(n=2), ITP contributed to 7.5% (n=3).ALL contributed to 5%(n=2),7.5%(n=3) attributed to liver failure.5%(n=2) had moyamoya disease. Medulloblastoma and TBM , sickle cell anaemia,aplastic anaemia contributed to 2.5%(n=1) each .2.5%(n=1) was idiopathic.

DISCUSSION

In present study 45%(n=18) belonged to age group of less than one year, followed by25%(n=10) 6-10 years ,20%(n=8)11-12 years . 10%(n=4) belonged to the age group of 1-5 years. AlmusawH et al[4] study 62.22% belongs to age group of 1-6 months, followed by <1 month (31.11%), 6-12 months (6.66%). Abbas et al et al[5] study74% belonged to age >1 year, 26% belonged to age <1 year. Liu et al 2015 et al[6]study 12 years was the median age. Khallaf et al et al[7]-6.1 years was the mean age (Range 1-18 years). Zidan et al et al[8]11 years was the mean age (Range 1month -17.5 years). Kumar R et al et al[9]13.8 years was the mean age (Range 2 months-17 years). Meyer-Heim and Boltshauseret al[10]7 years was the mean age (Range 2 months-16.9 years).
In Present study 60% (n=24) were males, 40%(n=16) of females. Almusaw H et al [4]64.44% were males, 35.55% of females. Abbas et al [5] 58% were males, 42% of females. Liu et al[6] 61.44% were males, 38.6% of females. Khallafet al [7]Male to female ratio was 1.4:1. Zidanet al[8] 18/30 were males, 12/30 were females. Kumar R et al 2009[52] et al Male to female ratio was 3:2. Meyer-Heim and Boltshauseret al[10] Male to female ratio was 1.3:1.
In the present study, 72.5%(n=29) presented with altered sensorium.70%(n=28) had vomiting. 47.5%(n=19) had headache ,45%(n=18)of the cases had seizures.30%(n=12) presented with refusal to feed.Extracranial Bleeding manifestations were present in 27.5%(n=11), 25%(n=10) had inconsolable cry.

Table-6:The findings of the present study can be compared with the following studies:

Author	Seizures	Headache	Vomiting	Altered sensorium	Others
Present study	**45%(n=18)**	**47.5%(n=19)**	**70%(n=28)**	**72.5%(n=29)**	**Bleeding manifestations –27.5%(n=11)**
Abbas et al [5]	42%	28%	44%	40%	---
Khallaf et al[7]	17	43%		33%	---
Zidan et al [8]	30%	60%	43%	46%	---
Kumar R et al [9]	28%	70%		50%	---
Meyer-Heim and Boltshauser[10]	26%	61%	45%	42%	----

In the present study, 30(n=12)had boggy fontanelle,17.5%(n=7) had normal fontanelle, 52.5%(n=21) had closed fontanelle. 17.5%(n=7)had anisocoria.Pupillary reflexes were

nonreactivein12.5%(n=5) of the patients. 15%(n=6) had increased/exaggerated deep tendon reflexes. Around 5%(n=2) had sluggish reflexes. Muscletone was increased in 15%(n=6) and decreased in 5%(n=2).

In Present study 17.5%(n=7) had normal fontanelle, 30% had boggy fontanelle.Meyer-Heim and Boltshauseret al[10] 5.8% had boggy fontanelle.In Present study Focal neurological defects were found in 22.5%(n=9) of the study population. Aphasia, blurring of vision, right side hemiparesis were present in 7.5%(n=3) each.5% (n=2) had left side hemiparesis. Abbaset al[5] 8% had hemiparesis, T Khallaf et al[7]20% had hemiparesis, Zidanet al[8] 36% had limb weakness, Kumar R et al[9] 36% had limb weakness, Meyer-Heim and Boltshauseret al[10] 13% ad focal neurological deficit.

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Table-7: The findings can be compared with the following studies:

Author	CT/CECT	MRI	MRA	MRV	Others
Present study	100%	75%	75%	75%	45% - NSG
Abbas et al[5]	98%	34%	10%	---	14%- CTA
Khallaf et al[7]	100%	---	42.02%	----	40.57% - CTA
Zidan et al [8]	100%	40%	30%	----	10% - CTA
Meyer-Heim and Boltshauser[10]	94.11%	23.5%	8.82	---	---

In Present study ICH was present in 55%(n=22), ICH and IVH were present in 5%(n=2). ICH and SAH were present in 7.5%(n=3). SDH was present in 27.5%(n=11). SAH was present in 2.5%(n=1). IVH was present in 2.5%(n=1).Almusaw H et al[4] SDH – 31.11%, SAH, IVH – 8.88%, ICH – 6.66%, EDH – 2.22%, SDH+SAH:6.66%, SAH+IVH:4.44%, SDH+IVH:6.66%, SDH+ICH:4.44%, ICH+IVH:11.11%.

In the present study, bone marrow aspiration was done in 15%(n=6).Among the 15%(n=6) BMA done,7.5%(n=3) bone marrow was normal, ALL with lymphoblasts was noticed in 5%(n=2). Cellularity was reduced in another 2.5%(n=1).

In the present study, 10% (n=4)had low levels for factor VIII (<1%), Factor IX was reduced in 5%(n=2).Urine PCR for CMV, gastric aspirate for CBNAAT was positive in 2.5% each.

The incidence of intracranial hemorrhage resulting from idiopathic vitamin K deficiency has decreased greatly since the introduction of vitamin K2 prophylaxis.In the present study, 35%(n=14)infants had hemorrhagic disease of newborn and it was the most common etiology of nontraumatic intra cranial hemorrhage identified. These 14 patients were diagnosed aslate type of HDN.All these14 cases were breastfed infants, delivered in hospital.

Out of these 14 cases ,5 cases hadhistory of receiving vitamin K at birth,In the remaining 9 cases,the evidence regarding administration of vitamin K was lacking. In few places vit K was not available when physically verified during the COVID Times. All corrective measures were taken in these hospitals and vit K provided on regular basis and ensured that every newborn received Vit K at birth.

In Present study 35%(n=14) had late type of vitamin k deficiency bleeding as the mostcommon etiology of nontraumatic intra cranial hemorrhage identified.Suzuki K et al [11] reported a case of a healthy 3-month-old male infant who had been born at full term to a gravida 2 para 2 mother presented with gradually decreased activity and acute onset of generalized convulsions without traumatic episodes. The infant received 3 doses of vitamin K. Three days before the onset of bleeding he had developed a fever of about 38.6°C and was diagnosed as having an upper respiratory infection which was not responding to the treatment. Later CT scan was done which revealed acute subdural hematoma and intracerebralhemorrhage. Sutor et al[12] the incidence decreased from 5.13 per 100,000 to a tenth of that. Matsuzakaet al[13] the incidence decreased from 34.3 per 100,000 to 10.1per 100,000. Nishioet al[14] review of the findings of CT in ICHs in 84 cases of Idiopathic Vitamin K Deficiency in Infants reported that subdural hematomas were present in41 cases (48.8%), subarachnoid hemorrhages in 72 cases(85.7%), intracerebralhemorrhages in 36 cases (42.9%),and intraventricularhemorrhages in 9 cases (10.7%). In addition, multiple hemorrhages occurred in 69% of the cases.

Motohara et al[15] was done a screening for late neonatal vitamin k deficiency by acarboxyprothrombin(PIVK-II) in dried blood spots in infant with one month of age.Acarboxyprothrombin (protein induced by vitamin K absence or antagonist-II (PIVKA-II)) concentrations in dried blood spots were determined in 19 029 infants at about 1month of age as an indicator of vitamin K deficiency. They observed 51 cases with raised bloodconcentrations of PIVKA-II (>4 AU/ml), nine of whom showed very high concentrations (>20AU/ml).For infants who did not receive vitamin K prophylaxis at birth, the incidence of the PIVKA-IItest yielding positive results was significantly higher in those solely breast fed (0.51%) comparedwith those fed formula milk (0.18%). Among solely breastfed infants, the incidence of a veryhigh result of the PIVKA-II test was 0-14% in those who had not received vitamin K prophylaxisat birth, 0.04% in those who received 2 mg orally, and 0.03% in those who received 2 mg orallyplus a further dose of 2-4 mg orally at 7 days.

Thus vitamin K prophylaxis at birth did not completely prevent vitamin K deficiency at 1month. They administered vitamin K therapeutically to all infants whose PIVKA-II test yielded apositive result at 1 month. To prevent the disease the optimal dose of vitamink needs to be determined.[12]

In the present study, 12.5%(n=5) had arteriovenous malformation, Among blood clotting disorders, haemophilia A contributed to 10%(n=4), haemophilia B contributed to 5%(n=2). ITP was contributed to 7.5%(n=3) and ALL contributed to 5% (n=2), 7.5%(n=3) attributed to liver failure.5%(n=2) had moyamoya disease. Medulloblastoma and TBM, Sickle cell anaemia contributed to 2.5%(n=1) each.2.5%(n=1) was idiopathic

Table-8: The findings of the present study can be compared with the following studies

Author	Vascular	Haematology	Tumours	Idiopathic	Misc.
Present study	12.5% - AVM	10% - Haem.A 5% - Haem.B ITP -7.5%, ALL- 5.% SCA – 2.5%	2.5% - Medulloblastoma 2.5% - TBM	2.5% %	5% had moyamoya disease
Abbas et al [5]	12%-AVM 2%-Aneurysm	8% - Acute Leukemia 12% - Aplastic anemia 6% - ITP	---	---	Meningitis – 2% Acute Liver Failure – 2%

		14% - Vit K 2% - DIC 6% - Fact VIII 4% - Fact XIII 2%- Thalesemia			Viral hepatitis -2%
Liu et al [6]	62.9% - AVM, Angiocavernoma – 5.7% Aneurysm -2.9%	Hemophilia-1.4%	2.9%	20%	Moyamoya-2.9%
Khallaf et al [7]	26% - AVM, 7%-Cavernoma, 3% - Aneurysm	ITP (12 cases), Hemophilia (11 cases), DIC (5 cases), Vit k Prophylaxis(4 cases), TTP (3 cases), I case of Chronic hemolyticanemia	6%	6%	3% Meningitis
Zidan et al [8]	17% - AVM 1 patient - Aneurysm	30%	2 patients	---	---
Kumar R et al[9]	44% - AVM Aneurysm – 34%	4%	4%	4%	Moyamoya – 6%
Meyer-Heim and Boltshauser[10]	47% - AVM Aneurysm-15% Complex vascular malformation – 6% Cavernoma – 6%	12% 2 casesof thrombocytopenia, 1 Fanconianemia	3%	4%	1 case of liver failure 9% hypertension .

CONCLUSION

Ministry of Health and Family Welfarerecommends that all newborns weighing more than 1000 gm should be given 1 mg of Vitamin K intramuscularly after birth . For babies weighing less than 1000 gm, a dose of 0.5 mg is recommended.This should be strictly followed in all the govt., private and setting where deliveries are being conducted. Any deviation can lead to neonatal and infant mortality.Regular follow up and timely intervention for AVM cases wouldprevent ICH.Blood clotting disorders likely severe haemophilia-A and B must beregularly monitored and prophylactic factor VIII and IX should be given to prevent ICH.

All diseases associated with thrombocytopenia, platelet count must be Monitored regularly and treated appropriately to prevent ICH. All cases of liver failure should be monitored with PT, APTT and INR,timely vitaminK should be given to prevent ICH. It is possible that risk factor profile of this study is unique to the institution and simply reflects the referral bias.

LIMITATIONS:

The study was a prospective observational study, in a high volume center. All the cases that were admitted were either referred from other centers or directly admitted in critical stages. Hence the complications or abnormalities observed were high. The results of the study cannot be generalised to general population and other hospital settings.

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