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Editorial

Recurrent Respiratory Tract Infection (RRTI) in Children

Children with RRTI pose one of the most difficult diagnostic challenges in pediatrics. Response of clinicians may be anything from reassurance to the diagnosis of a life threatening conditions as they faces a twofold challenge to determine (a) is this child normal [needs only reassurance] or (b) seems to have a serious disease [needs at least minimum number of least invasive tests to reach the diagnosis].

Most children experience one or more respiratory infections, where clinicians to decide when to move from symptomatic therapy to start diagnostic testing; then again when to discontinue the testing and finally to start definitive therapy for the child's underlying conditions. General pointers that suggest early investigation may be expressed by the acronym "SPUR" (Severe, Persistent, Unusual, Recurrent) respiratory infections. Immunodeficiency can present with upper airway problems and factors demanding for further investigations include: eight or more new ear infections in a year; two or more serious (requiring intravenous antibiotics) sinus infections within a year; persistent oral candidiasis; two or more months of continuous oral antibiotics with no effects; need for intravenous antibiotics to clear infection.

Of all upper airway infections, recurrent viral URI is most common. It may occur six to eight times a year. Almost 10 percent of children may have 10 or more colds per year. If the mean duration of an infection is 7 days, then a child may suffer almost 3 months in a year from common cold. Recurrent tonsillitis and pharyngitis, sinusitis and otitis media with effusion all have the footprints in childhood RRTI.

Children with lower airway infections may be divided into three categories: (a) chronic productive cough (b) Recurrent wheeze with LRTI and (c) Recurrent radiological shadowing. Specific conditions that are referred to differential diagnosis in this section are Cystic fibrosis (CF), Primary ciliary dyskinesia (PCD) and Immunodeficiency. Chronic cough for all practicality is taken as a timeframe of more than 4 weeks though British Thoracic Society recommends 8 weeks as the definition of chronic cough. In young children, recurrent wheezing is almost always associated with viral Lower respiratory tract infection (LRTI). Clinicians should try to differentiate prospectively, episodic viral associated wheeze and multi-trigger wheeze. The term radiologic shadowing is used because many of these conditions can cause either consolidation or atelectasis. The patterns are often nonspecific and detailed further investigations usually are indicated. At least two episodes of documented radiological shadowing are required to merit investigation for possible recurrent LRTI. In this context, I would like to mention that "Right middle lobe syndrome" probably is the most common clinically encountered focal consolidation. The definition of right middle lobe syndrome is clinical: atelectasis of right middle lobe or lingula persisting for more than one month or recurring twice or more despite treatment. The families of children who have this syndrome should be advised that they are prone to chronic respiratory infections and asthma and as such should be followed up carefully.

Pointers in the history leading to further investigations include; marked chronic upper airway symptoms like snoring, rhinitis, sinusitis; symptoms from the first day of life; very sudden onset of symptoms; chronic moist cough with sputum production; vomiting and choking symptoms after feeds specially on lying down; any features of systemic immunodeficiency.

Pointers in the physical examination leading to further investigations include; digital clubbing, weight loss, failure to thrive; enlarged tonsils with exudates and adenoids, nasal polyps; severe chest deformity like harrison's sulcus, barrel chest; stridor (monophasic or biphasic); asymmetric wheeze; signs of cardiac or systemic disease.

Overall recurrent respiratory tract infections in children demands maximum attention from a pediatrician and should be managed thoroughly and carefully.

Santanu Bhakta
Editor-in-Chief

Review Article

Neurocysticercosis in Children

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Abstract:

Neurocysticercosis (NCC) is caused by the encysted larvae of the pork tapeworm Taenia solium. It is the most common parasitic infection of human brain and is the leading cause of acquired symptomatic epileptic seizures in the developing countries.

Humans are the definitive host of adult T. solium and are affected by ingesting undercooked pork containing cysticerci. Cysticerci that reach the brain cause NCC.

The clinical manifestations are non-specific even asymptomatic in many cases but presence of seizures, vomiting, headache in the absence of fever should arouse suspicion of NCC in an endemic country like India.

NCC are usually divided into parenchymal which usually presents with seizures and extra parenchymal where signs and symptoms of raised intracranial pressure is common.

The diagnosis is largely based on clinical presentation and neuroimaging CT/MRI. Treatment approaches vary depending on the clinical presentation and type of NCC.

Neurocysticercosis (NCC) is an infection of the brain and its coverings by the larval stage of the tapeworm *Taenia solium*. It is the most common helminthic infestation of the central nervous system (CNS) and a leading cause of acquired epilepsy worldwide[1]. NCC results from ingestion of the eggs of *T. solium*. The oncospheres hatch in the intestine, penetrate the intestinal wall and disseminate to several body tissues, showing strong tropism to the CNS. The clinical manifestations of NCC are non-specific and varied and depend on the number of lesions and the developmental stage of the cysticercus. Seizures are the commonest manifestation, occurring in 50% to 80% of patients[2,3]. NCC has been classified depending on the location of cysts, its clinical presentation, prognosis and cyst viability[4-7].

Life cycle and pathogenesis

T. solium has a complex life cycle, requiring two hosts (Fig. 1). Humans are the definitive host whereas pigs are the intermediate host[8-9]. Humans can also act as intermediate hosts after ingestion of Taenia eggs. The disease manifests itself as two distinct clinical entitites: taeniasis and cysticercosis.

Cycsticerci often live asymptomatically within host tissues for years as they have developed various mechanisms for evading host response[10]. Metacestodes secrete a serine protease inhibitor- taeniaestatin which inhibits complement activation and cytokine production and

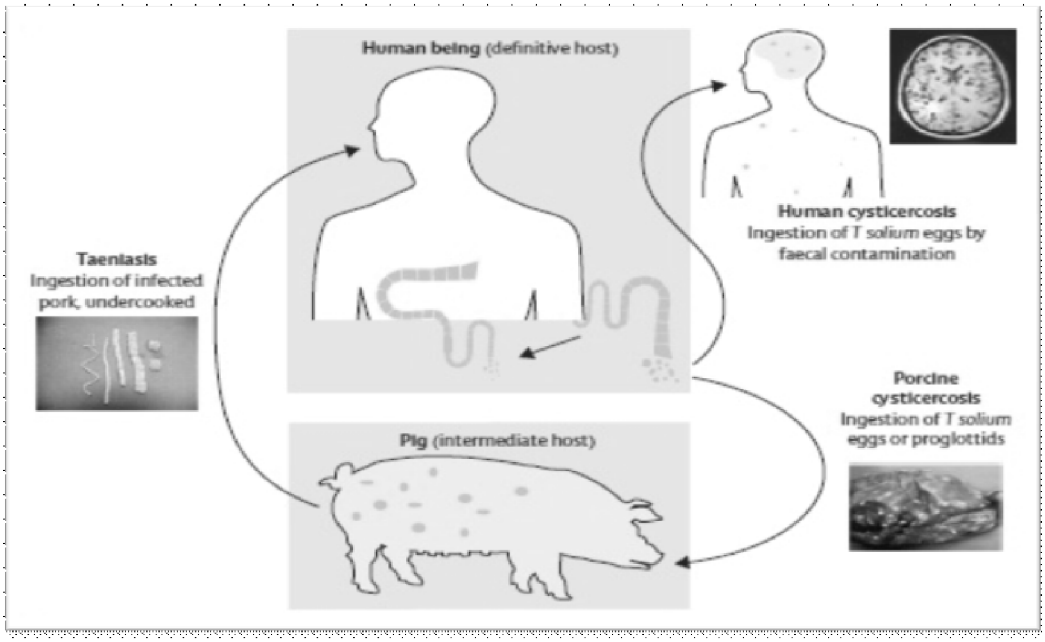

Fig 1: Life Cycle of *Taenia solium* - Reproduced and adapted from Garcia and colleagues: Lancet Neurol 2005.

interferes with leukocyte chemotaxis. Parasite paramyosin also binds to C1q and inhibits the classic pathway of complement activation. The cellular immune response is also suppressed[13].

Clinical manifestations

These are pleomorphic and are determined mainly by the location, number and viability of the cysts as well as by the host response.

Seizure is the commonest symptom, two thirds are partial and one third generalized.

Seizures in NCC –acute symptomatic (70%) or remote symptomatic (30%).

Most children present with single degenerating parenchymal cysts, some with multiple cysts.

Parenchymal NCC – commonest presentation

Most cases of childhood NCC are seen after five years of age although some cases are seen in preschoolers and even in infants. The common clinical manifestations include:

Seizures – Sudden onset of seizures in otherwise healthy children is the commonest presentation. Seizures occur in 70-90% of cases; in a series of 500 children from India, seizures were reported in 94.8% cases[12]. Most children present with partial seizures (84-87%)[12] particularly complex partial seizures; about a quarter have simple partial seizures[15]. Most seizures are of short duration, generally lasting for less than 5 minutes. Status epilepticus has been reported in 1.7% to 32% cases[12]. Seizures are generally single; in a hospital based study, NCC was discovered on imaging in 59.2% of cases with single seizures[13].

Depending on the stage of the disease, the seizures may be considered provoked or unprovoked, and these may co-exist. Although it is presumed that degeneration of the cysts and the associated inflammatory response evoke seizures, this has been questioned in a recent study wherein 29% asymptomatic family members of symptomatic cases were diagnosed to have NCC. A large number of individuals harboring different stages of

cysticerci in their brain were asymptomatic. NCC has been found to be associated with mesial temporal lobe epilepsy due to hippocampal sclerosis -this may provide new insights into epileptogenesis[14].

Extraparenchymal NCC: This is rare in children as compared to adults.

(i) *Intraventricular* – May cause obstruction and hydrocephalous.

(ii) *Subarachnoid* – May show hydrocephalus, arachnoiditis and occasionally infarcts.

(iii) *Spinal* – Radicular pain, paresthesia, sphincteric disturbances and paraplegia.

(iv) *Ophthalmic* – Can be found anywhere in the eyes/ extraocular muscles – pain, diplopia, impaired vision.

Diagnosis of NCC

The diagnosis of NCC is suspected in patients living in endemic areas presenting with the compatible clinical picture and lesions suggestive of NCC on CT scans. Neuroimaging is the mainstay of diagnosis of NCC.

CT scan of the head :

CT scans have a sensitivity and specificity of over 95% in the diagnosis of NCC,[15,16] but it is much lower for diagnosis ventricular or cisternal forms of the disease. CT scans are optimal for detecting calcifications, which occur in about 50% of patients.

MRI of the brain :

Usually done if CT scan brain is inconclusive

MRI is the most accurate technique to assess degree of infection, location and evolutionary stage of the parasite. Superior to CT in detecting intraventricular lesion, subarachnoid lesion, brainstem and posterior fossa lesion, perilesional edema.

Serology :

The parasite load and genotype plays a crucial role in the test outcome and it is important to understand that a positive result supports the diagnosis but a negative result does not rule out NCC.

The Gold standard is Enzyme Linked Immuno Electrotransfer Blot assay (EITB) which uses lentil lectin purified glycoprotein antigens (LLGP) to detect antibodies to *T solium* in serum. The test is highly specific (100%) and nearly 98% sensitive for active lesions, but reduced sensitivity (50-70%) is reported with solitary lesions and calcified NCC. (Lancet Neuro, 2005)

The EITB assay can be performed in both serum and CSF, but sensitivity is higher with serum. Although highly recommended its use is limited because of lack of facility and expertise.

Anticysticercal antibodies in the CSF can be detected by ELISA with a sensitivity of 89% and specificity of 93% and is used when EITB is not available. ELISA is more reliable in CSF than in serum.

Newer investigation like detection of circulating antigen in serum by ELISA with monoclonal antibodies is being carried out with promising results.

Other diagnostic tests :

Nonspecific and does not help in the diagnosis.

(i) Peripheral eosinophilia ~ 10%

(ii) CSF pleocytosis (mononuclear cells <300 cells), ↑ protein, ↓ glucose

(iii) Stool – concurrent intestinal Taeniasis (<10%).

Differential diagnoses

Clinically may mimic encephalitis, stroke, meningitis and most neurological conditions depending on the type of NCC.

On imaging – if scolex is not visible may be difficult to distinguish from tuberculoma, microabscess, toxoplasmosis

Other conditions that can mimic single or multiple ring or nodular enhancing lesions include mycotic granuloma, and primary or metastatic brain tumor.

Neurocysticercosis or Tuberculoma? – Often a clinical dilemma. The distinguishing features are presented in Table 1.

Table 1. Clinical features of NCC and Tuberculoma

Clinical Features	NCC	Tuberculoma
Raised ICT	Unlikely	Usually present/midline shift
Progressive focal neuro deficit	Unlikely	present
CSF study	nonspecific	suggestive
Size of lesion	< 2cms	>2cms
Shape	Smooth outer margin	Lobulated andirregular
Location of lesion	Gray white matter junction/ cortex	Base of the brain/posterior fossa

Treatment: The treatment modalities for NCC are showing in table 2.

Principles of therapy

- Symptomatic/Supportive
- Antiepileptic therapy:
  - – Single AED – carbamazepine or phenytoin
  - – Usually 1 yr. seizure free interval (after EEG – Normal, no lesion on neuroimaging)
- Corticosteroids: 1-3 days prior to start of anti-parasitic therapy.
  - – To reduce perilesional edema/? host inflammatory response (oral prednisolone/IV dexamethasone)
- Definitive- medical/surgical treatment for cysts
- Evidence favours antiparasitic drugs hasten resolution of live parenchymal brain cysts
- Both praziquantel and albendazole were found to be effective in destroying viable cysts.
- Albendazole 15mg/kg/day for 4 weeks
- Praziquental 50mg/kg/day for 2 weeks

  – calcified cysts (in the absence of viable lesions) needs -

  – NO anti- parasitic therapy

Cysticidal agents – to kill larvae both praziquantel and albendazole were found to be effective in destroying viable cysts.

- Albendazole vs Praziquental

  Albendazole is less expensive, fewer side effects, better penetration of SA space, do not ? its bioavailability with AED/coticosteroids.

  ICT/ ocular NCC – only steroids, NO cysticidal drugs.

Clinical outcome:

- Single enhancing lesion (NCC) has a good prognosis – by 6 months – 60% lesion disappears.

Table 2 : The treatment modalities for NCC include

Location	Stage	Treatment
1. Parenchymal	Viable or degenerative	Anti-cysticercal therapy and steroids
	Calcified	No anti-cysticercal therapy ± AED
2. Extra parenchymal	Intraventricular	Endoscopic removal
	Subarachnoid	Anti-cysticercal drugs with steroids plus VP shunt if hydrocephalus is present
	Spinal and Ocular	Primary surgical treatment

- Multiple and calcified lesions have frequent seizure recurrences.
- Prognosis is generally poor in cysticercus encephalitis and extra parenchymal NCC, fortunately uncommon in children.

Preventive strategies parasite control and potential elimination:

International Task Force for Disease Eradication – has targeted

T solium for focal elimination and eventual eradication.

- Blocking transmission of tapeworm infections and eggs to humans

Prevention of human exposure

Inspection of pork meat

Proper cooking/freezing – to destroy cysticerci

Prevention of infection in pigs

(i) Restricting exposure to human waste (sewage disposal and open defecation) –

(ii) Mass community antihelminthic therapy to ? prevalence of NCC

Transmission of eggs between humans

(i) Personal hygiene and hand washing

(ii) Proper washing of fruits and vegetables

(iii) Treatment for human tapeworm carriers

(iv) Community education regarding routes of transmission

- Vaccination of pigs with newer TSOL18 and
- Use of Oxfendazole in pigs

Key Points

- Suspect NCC in a child with focal seizure particularly when presenting without fever.
- Commonest cause of acquired focal epilepsy
- Individuals with no history of pork consumption/ vegetarian can also develop NCC.
- Ingestion of infected pork causes adult tapeworm infestation (taeniasis) but not cysticercosis, eggs of *T. solium* cause cysticercosis.
- It is advisable to do a fundoscopy to rule out ocular cysticercosis before staring anti-cysticercal therapy in NCC.

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Review Article

Neonatal Sepsis: A Review

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Sepsis is the most common cause of neonatal mortality. As per National Neonatal Perinatal Database (NNPD) 2002-2003, the incidence of neonatal sepsis in India was 30 per 1000 live birth. Newborn infants are at much higher risk for developing sepsis than children and adults because of their immature immune system—especially premature infants, where 1 out of every 250 will be diagnosed with sepsis. The combination of an immature and slow responding immune system increases the risk of neonatal sepsis. One reason for the increased risk is that antibodies, which help protect mothers from infections, do not cross through the placenta to the fetus until approximately 30 weeks of gestation. The antibodies present at birth take time to reach optimum levels, which also affects the protection provided. The early manifestations of neonatal sepsis are vague and ill-defined. Although blood culture is the gold standard for the diagnosis of sepsis, culture reports would be available only after 48-72 hours. In this era of multidrug resistance, it is mandatory to avoid unnecessary use of antibiotics to treat non-infected infants and at the same time treat the indicated cases to prevent mortality and long-term sequelae.

Introduction

Sepsis is the most common cause of neonatal mortality causing 30-50% of total neonatal deaths each year in developing countries[1-3].Upto 20% neonates develop sepsis and approximately 1% die of sepsis[2]. Neonatal sepsis refers to systemic infection of neonates and comprise of pneumonia, meningitis, arthritis, osteomyelitis, urinary tract infection and septicemia. *Klebsiella pneumoniae* is the most commonly isolated bacteria (32.5%), followed by *Staphylococcus aureus* (13.6%).

Probable (Clinical) Sepsis[3]: Presence of any one of the following criteria along with clinical picture suggestive of septicemia

(a) Predisposing factors: maternal fever or foul smelling amniotic fluid or prolonged rupture of membranes (>24 hrs) or gastric polymorphs (>5 per high power field)

(b) Positive septic screen – Presence of two of the following parameters

1. TLC (< 5000/mm)
2. Band to total polymorphonuclear cells ratio of >0.2
3. Absolute neutrophil count < 1800/cumm
4. C-reactive protein (CRP) >1mg/dl
5. Micro ESR > 10 mm-first hour

(c) Radiological evidence of pneumonia

Culture positive sepsis – Presence of either of the following with clinical septicemia, pneumonia or meningitis: Microbes detected in blood/ CSF/ urine/abscess or pathological evidence of sepsis.

Classification

Neonatal sepsis can be classified into two types: early and late onset

Early onset sepsis (within first 72 hours of life) :

The neonate may be symptomatic in utero (fetal tachycardia, poor beat to beat variability on cardiotocography) in severe cases. It manifests as respiratory distress/ pneumonia. Risk factors is associated with increased risk[4,5].

(a) Low birth weight (<2.5kg)/ preterm baby

(b) Maternal febrile illness within 2 weeks prior to delivery

(c) Foul smelling and/or meconium stained amniotic fluid

(d) Prolonged rupture of membranes (>24 hours)

(e) >3 intrapartum vaginal examinations

(f) Prolonged and difficult delivery with instrumentation

(g) Perinatal asphyxia (Apgar score <4 at 1 minute of age) or difficult resuscitation

Neonates with presence of foul smelling liquor or 3 of above mentioned risk factors should be considered to have early onset sepsis and treated with antibiotics. =2 risk factors should be investigated first[6].

Late onset sepsis (after 72 hours of age) :

Infection may be either nosocomial or community acquired and manifests as septicemia, pneumonia or meningitis[7,8]. Risk factors include:

NICU admission	Poor hygiene
Low birth weight (LBW)	Poor cord care
Prematurity	Bottle feeding
Invasive procedure	Superficial infection (pyoderma, umbilical sepsis)

Clinical manifestations

Altered feeding behaviour is common and early. Others include hypothermia or fever (former is more common in LBW babies), lethargy, poor cry, poor perfusion i.e. prolonged capillary refill time (>2 seconds), hypotonic or absent neonatal reflexes, bradycardia/tachycardia, respiratory distress i.e. apnea or gasping respiration, hypoglycemia/hyperglycemia and metabolic acidosis.

Systemic manifestations include[9]:

Central nervous system – Bulging anterior fontanel, high-pitched cry, excessive irritability, coma, seizures, and neck retraction (clinical suspicion of meningitis)

Cardiac: hypotension and poor perfusion – Gastrointestinal: feed intolerance, vomiting, diarrhea, abdominal distension, paralytic ileus, NEC

Hepatic: hepatomegaly and direct hyperbilirubinemia

Renal: acute renal failure – Hematological: bleeding and petechiae, purpura

Skin: multiple pustules, sclerema, mottling, umbilical erythema/ discharge

Investigations

Blood culture :

This is the gold standard for diagnosis of septicemia. It must be done in all cases prior to the institution of antibiotics. Blood culture should be observed for 72 hours before reporting it sterile. The recent BACTEC culture system detects growth in 12-24 hours at bacterial concentration of 1-2 cfu/ml.

Sepsis screen[10,11] –

Is a panel of tests consisting of:

Component abnormal value –

TLC < 5000/mm^3

ANC < as per Manroe chart for term[12] and Mouzinho's chart for very LBL (VLBW)[13]

Immature/ total neutrophil > 0.2

Micro-ESR > 15mm in 1st hr

CRP > 1mg/dl

All neonates suspected to have sepsis should have a septic screen to corroborate the diagnosis. However, the decision to start antibiotics need not be conditional to the sepsis screen result, if there is a strong clinical suspicion of sepsis.

Sepsis screen is considered positive if two of these are positive. If the screen is negative but clinical suspicion persists, it should be repeated within 12 hours. If the screen is still negative, sepsis can be excluded with reasonable certainty. Presence of two abnormal parameters in screen is associated with sensitivity 93-100%, specificity 83%, positive and negative predictive value of 27% and 100%, respectively in detecting sepsis.

Lumbar puncture (LP) :

The incidence of meningitis in neonatal sepsis is varied (0.3-3%)[3,9]. In early onset sepsis, lumbar puncture is indicated in the presence of a positive blood culture or if the clinical picture is consistent with septicemia. In late onset sepsis, LP should be done in all infants prior to starting antibiotics. LP should not be done in following cases:[14]

(i) Asymptomatic babies being investigated for maternal risk factors

(ii) Premature neonates presenting with respiratory distress syndrome

Urine culture :

Given the low yield of positive urine culture results and costs of processing the specimens, urine culture should not be part of traditional sepsis evaluation in first 72 hours of life[15]. However, neonates at risk for fungal sepsis and very low birth weight infants with poor weight gain should have a urine examination done to exclude urinary tract infection (UTI).

UTI may be diagnosed in the presence of one of the following:

(a) >10 WBC/mm in a 10 mL centrifuged sample

(b) >10 organisms/ mL in urine obtained by catheterization and

(c) any organism in urine obtained by suprapubic aspiration

Radiology :

Chest X-Ray is done in cases of respiratory distress or apnea. Abdominal X-ray should be done for diagnosis of necrotizing enterocolitis.

Newer diagnostic tests for diagnosis of neonatal sepsis :

Isolation of bacteria from blood is a standard and most specific method used to diagnose neonatal sepsis. Positive cultures ranged from 8% to 73%[16]. An additional drawback of culture based diagnosis is the 24–48 hour assay time. Newer diagnostic tests are acute phase reactants, cell surface markers, Granulocyte colony stimulating factor, cytokines, molecular genetics, mol cell proteomics.

Management

Supportive treatment :

(a) Infant nursing in a thermoneutral environment (to avoid hypo/ hyperthermia)

(b) Oxygen saturation to be maintained in the normal range and ventilation initiated

(c) Regular monitoring for hypoglycemia/ hyperglycemia

(d) Colloids and inotropes for maintaining blood pressure and tissue perfusion

(e) Enteral feeds should be avoided till the baby is haemodynamically stable

(f) Packed cells and fresh frozen plasma for managing anemia and bleeding diathesis

Antimicrobial treatment :

The choice of antibiotic depends upon the predominant pathogen and antibiotic sensitivity pattern.

1. Community acquired: ampicillin+gentamicin preferable for septicemia and pneumonia
2. 3rd generation broad spectrum cephalosporin e.g. cefotaxime added for meningitis (due to its reduced toxicity)
3. Ceftriaxone not commonly used: potential of displacement of bilirubin from albumin to cause hypoprothrombinemia and bleeding.
4. Nosocomial infection and resistant strains: cefotaxime in combination with an aminoglycoside preferred for septicemia, pneumonia as well as meningitis.

5. 3rd generation cephalosporins have very good CSF penetration and excellent antimicrobial activity against gram-negative organisms and hence good choice for nosocomial infections and meningitis. However, as per recent reports, 60-70% gram-negative organisms have become resistant to them. Piperacillin-tazobactam or methicillin/vancomycin preferred in resistant cases.
6. Piperacillin-tazobactam with amikacin: pseudomonas sepsis
7. Penicillin resistant staphylococcus aureus: cloxacillin, nafcillin or methicillin. Addition of an aminoglycoside is useful. Methicillin resistant staphylococcus aureus (MRSA): combination of ciprofloxacin or vancomycin with amikacin.
8. Ciprofloxacin has excellent activity against gram-negative organisms but with poor CSF penetration, useful for resistant gram-negative bacteremia after excluding meningitis.
9. Enterococcus: ampicillin and gentamicin (Vancomycin for resistant cases)

Reserve antibiotics (Aztreonam, meropenem): Empirical use of the newer antibiotics should be avoided; they should be reserved for situations where sensitivity of the isolated organism warrants its use. Imipenem is avoided in neonates because of increased incidence of seizures.

Duration of antibiotic therapy:

(a) Clinical sepsis-7-10 days

(b) Culture positive sepsis (not meningitis), UTI - 14 days

(c) Meningitis-2 weeks after sterilization of CSF culture or for a minimum of 2 weeks for gram positive meningitis and 3 weeks for gram negative meningitis, whichever is longer

(d) Bone and joint infection-4-6 weeks

Adjunctive therapy :

Intravenous Immune Globulin (IVIG) – According to Cochrane database systemic review there is insufficient evidence to support its routine use[17]. Immunotherapy used as an adjuvant for the prevention and treatment of neonatal sepsis holds promise[18]. In a recent paper, the authors have reviewed immunotherapies that modulate the immune system of the neonate, including intravenous immunoglobulins and myeloid haematopoietic growth factors[19].

Granulocyte colony stimulating factor (G-CSF)[20-22] – Carr and colleagues reported a randomized trial (PROGRAMS) of GM-CSF for the prevention of sepsis in small for gestational age preterm neonates. This increased the neutrophil count, but had no effect on the primary end point of sepsis free survival to 14 days from trial entry[20]. According to the Cochrane database systemic review there is currently insufficient evidence to support the introduction of either G-CSF or Granulocyte monocyte colony stimulating factor (GM-CSF) into neonatal practice, either as treatment of established systemic infection to reduce resulting mortality, or as prophylaxis to prevent systemic infection in high risk neonates[21].

Exchange transfusion – Has not been extensively studied. It may be used with caution in neutropenia, sclerema, earliest evidence of disseminated intravascular coagulation and metabolic acidosis (pH <7.2)[23].

Pentoxifylline – Pentoxifylline is a methylxanthine that has been postulated to improve outcomes through modulating the activity of the reticuloendothelial system and decreasing the neutrophil activation that contributes to acute tissue injury. Large scale clinical trials have not yet been performed[24].

Conclusion

Clinical diagnosis of neonatal sepsis may not be easy due to the non-specific nature of symptoms and signs. No laboratory test exists with 100% specificity and sensitivity. Blood culture has been the gold standard for confirmation of diagnosis but the test results are available only after 48-72 hours. The neonates with "risk factors" for sepsis are thus treated with broad-spectrum antibiotics and may require prolonged hospitalization. Although many putative markers (acute phase reactants, cytokines) are reported, most are not available to the routine diagnostic laboratory.

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Review Article

Status Epilepticus – A Short Review

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Status epilepticus

(SE) is the most common neurological medical emergency and continues to be associated with significant morbidity and mortality.

In the past few years, the duration of what is accepted as SE has been decreasing progressively and most recently, a length of 5 min has been proposed. Most seizures cease within a minute or two and if the seizure is prolonged beyond a few minutes, it is unlikely to stop by itself. Spontaneous termination becomes less likely in seizures lasting more than 5 minutes, and the longer the seizure continues , the more difficult it is to control the seizure with antiepileptic drugs (AED) and greater the degree of neuronal damage.

It appears that in SE the innate inhibitory mechanisms in the brain that puts a halt to the seizure are no longer effective. Duration of 5 minutes probably is a reasonable cutoff to distinguish isolated seizures from SE. Numerous clinical studies have demonstrated a relation between seizure duration and mortality.

When confronted with a patient with continuous seizures, one cannot wait for 30 minutes or for that matter even for 15 minutes before initiating therapy. Further there is evidence that seizures may become refractory and difficult to control if treatment is delayed.

Approximately 50 million people currently live with epilepsy worldwide. The estimated proportion of the general population with active epilepsy (ie. Continuing seizures or with the need for treatment) at a given time is between 4 and 10 per 1000 people. However, some studies in low and middle income countries suggest that the proportion is much higher between 7 and 14 per people. Globally, an estimated 2.4 million people are diagnosed with epilepsy each year. In high income countries, annual new cases are between 30 and 50 per 100 000 people in the general population. In low and middle income countries , this figure can be up to two times higher.

A primary central nervous system (CNS) disorder or a metabolic abnormality may result in episodes of SE.,some such important conditions are trauma, infections such as meningitis or encephalitis, hypoxia- ischaemic encephalopathy, intra cranial tumours and cerebrovascular diseases. It has been observed that the classical symptoms and signs of acute bacterial meningitis may be absent in SE and a high index of suspicion for infection in the child with SE and fever is paramount. Metabolic abnormalities such as hypoglycemia, hyperglycemia, hyponatremia, hypernatremia, hypomagnesaemia, hypocalcemia and uraemia may precipitate an episode of SE. Systemic disorder like hypertension may occasionally present with SE. Non compliance with antiepileptic medications, abrupt drug withdrawal or even drug overdosage can result in SE. The most important cause of SE varies with the age of the child. Whereas febrile SE is the most common

cause in children less than five years of age, trauma and infections are important in older children. Severe hypoxic encephalopathy and inborn error of metabolism may present with SE in the newborn.

The important risk factors for SE are a history of epilepsy, younger age of patient, genetic predisposition and acquired brain insult. The important precipitating factors includes fever, irregular or overdose of AED medications, sudden discontinuation of AED, sleep deprivation, fatigue, metabolic derangements, concomitant use of other medications, hyperventilation.

The clinical scenario, including the history and physical examination, is the most important factor guiding the specific evaluation that each child will require. The investigations usually considered include blood chemistries, complete blood count, AED level, toxicological studies, lumbar puncture, electroencephalography and neuroimaging like CT scan and MRI. If neuroimaging is considered, it should only be done after the child is stabilized and with no seizure activity. It is done to evaluate the child with SE if there are clinical indications or if the etiology is unknown. The algerithm for management of convulsive SE is presented in table 1.

Table 1 : Proposed Algorithm for Convulsive Status Epilepticus by American Epilepsy Society

From "Treatment of Convulsive Status Epilepticus in Children and Adults," Epilepsy Currents 16.1 - Jan/Feb 2016

Time line	**Interventions for emergency department, in-patient setting or pre hospital setting with trained paramedics**
0-5 Minutes Stabilization Phase	1. Stabilize patient (airway, breathing, circulation, disability - neurologic exam) 2. Time seizure from its onset, monitor vital signs 3. Assess oxygenation, give oxygen via nasal cannula/mask, consider intubation if respiratory assistance needed 4. Initiate ECG monitoring 5. Collect finger stick blood glucose. If glucose < 60 mg/dl then Adults: 100 mg thiamine IV then 50 ml D50W IV Children = 2 years: 2 ml/kg D25W IV Children < 2 years: 4 ml/kg D12.5W IV 6. Attempt IV access and collect electrolytes, hematology, toxicology screen, (if appropriate) anticonvulsant drug levels
	Does Seizure Continue?
5-20 Minutes Initial Therapy Phase	Yes A benzodiazepine is the initial therapy of choice: Choose one of the following 3 equivalent first line options with dosing and frequency: • Intramuscular midazolam (10 mg for > 40 kg, 5 mg for 13-40 kg, single dose, OR • Intravenous lorazepam (0.1 mg/kg/dose, max: 4 mg/dose, may repeat dose Once, OR • Intravenous diazepam (0.15-0.2 mg/kg/dose, max: 10 mg/dose, may repeat dose once, If none of the 3 options above are available, choose one of the following: • Intravenous phenobarbital (15 mg/kg/dose, single dose OR • Rectal diazepam (0.2-0.5 mg/kg, max: 20 mg/dose, single dose, OR • Intranasal midazolam ,buccal midazolam)

	Does Seizure Continue?
20-40 Minutes	Yes
Second Therapy Phase	There is no evidence based preferred second therapy of choice: Choose one of the following second line options and give as a single dose • Intravenous fosphenytoin (20 mg PE/kg, max: 1500 mg PE/dose, single dose,) OR • Intravenous valproic acid (40 mg/kg, max: 3000 mg/dose, single dose,) OR • Intravenous levetiracetam (60 mg/kg, max: 4500 mg/dose, single dose) If none of the options above are available, choose (if not given already) • Intravenous phenobarbital (15 mg/kg, single dose)
20-40 Minutes	Yes
Second Therapy Phase	There is no evidence based preferred second therapy of choice: Choose one of the following second line options and give as a single dose • Intravenous fosphenytoin (20 mg PE/kg, max: 1500 mg PE/dose, single dose,) OR • Intravenous valproic acid (40 mg/kg, max: 3000 mg/dose, single dose,) OR • Intravenous levetiracetam (60 mg/kg, max: 4500 mg/dose, single dose) If none of the options above are available, choose (if not given already) • Intravenous phenobarbital (15 mg/kg, single dose)
	Does Seizure Continue?
40-60 Minutes	Yes
Third Therapy Phase	There is no clear evidence to guide therapy in this phase : Choices include: repeat second line therapy or anesthetic doses of either thiopental, midazolam, pentobarbital, or propofol (all with continuous EEG monitoring)
If seizure stops in between 0 to 60 minutes , then symptomatic medical care	
	This clinical algorithm/guideline is designed to assist clinicians by providing an analytic framework for evaluating and treating patients with status epilepticus.

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Case Report

Leigh Disease:A Case Report

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Abstract:

Leigh syndrome is a devastating neurodegenerative disease, typically manifesting in early childhood.First described by Denis Archibald Leigh in 1951, it has evolved from a postmortem diagnosis, strictly defined by histopathological observations, to a clinical entity with indicative laboratory and radiological findings. Hallmarks of the disease are symmetrical lesions in the basal ganglia or brain stem on MRI, and a clinical course with rapid deterioration of cognitive and motor functions. We describe a child diagnosed as Leigh Disease based on clinical and laboratory parameters who was put on thiamin,CoQ and carnitine supplementation with partial improvement in symptoms,

Keywords: *Juvenile subacute necrotizing encephalomyelopathy;Leigh disease*

Introduction

Neuroregression is a condition where there is gradual loss of already attained developmental milestones. It can be due to white matter disease, grey matter disease (with and without visceromegaly), spinocerebellar causes, basal ganglia lesions, metabolic causes and infections like HIV, rubella etc. So diagnosing a cause of neuroregression usually becomes difficult. After diagnosing the child treating becomes even more difficult as most of the conditions are irreversible and potentially fatal. We hereby report a 2 year 7 month old child presenting with regression of milestones, ultimately diagnosed to be having a rare disease called Leigh disease and who partially responded to supplementation.

Case report

A 2 year 7 month old male baby, product of non-consanguinous marriage presented with complaints of not able to sit for last 1 year, not able to hold neck for last 11 months and not able to speak for last 7 months.He had attained all these milestones at normal expected ages.The parents had noticed the child to repeatedly fall backwards while sitting at around the age of 18 months. Slowly the child developed inability to hold neck followed by inability to walk or crawl or stand followed by inability to speak. By the age of 2 years and 2 months the child had developed abnormal movements and altered sleep cycle. The birth history and family history was insignificant and development was normal till 18 months of age.

On examination the child had a GCS of 13/15, was irritable with a significant finding of vertical nystagmus and chorea. There was no organomegaly.Anthropometry was within normal limits.Eye examination or physical features revealed no other abnormality. Other CNS examination as well as other system examination was unremarkable.

The baseline investigations like complete blood count,

liver function tests, serum creatinine, electrolytes level, blood sugar,lipid profile were within normal limits. CSF analysis was also unremarkable.TORCH, FT4, TSH were within normal limit. Clinically, absence of spasticity and optic atrophy suggested a grey matter disease rather than white matter disease with visceromegaly.

MRI brain of the child revealed bilateral almost symmetrical diffusion restricted altered signal intensity in the basal ganglia, dentate nucleus and thalamus(Fig1).Magnetic resonance spectroscopy revealed a omega shaped reverse lactate peak(Fig 2) suggestive of mitochondrial encephalopathy especially Leighs disease.Serum lactate and pyruvate levels were sent which were raised corroborating the diagnosis(Lactate-3.8 mmol/L,N-1.0-2.4 mmol/L and Pyruvate-1.14mg/dl,N-0.3-0.9 mg/dl). Nerve conduction velocity was normal. Absence of myoclonus and features of peripheral neuropathy ruled out other mitochondrial condition namely MERRF.

The child was treated with thiamine 100 mg/8 hourly, coenzyme Q 10mg/kg/day , L-carnitine 100 mg/kg/day and diet modification with high fat, high protein and restricted carbohydrates.At follow up of 3 months , child's interaction with the parents has increased with improved neck holding.The involuntary movements have decreased.

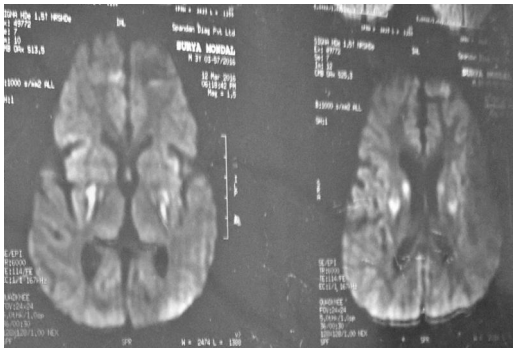

Fig 1. MRI T1 weighted image showing bilateral symmetrical hyperintensities in the basal gaglia and dentate nuceus region.

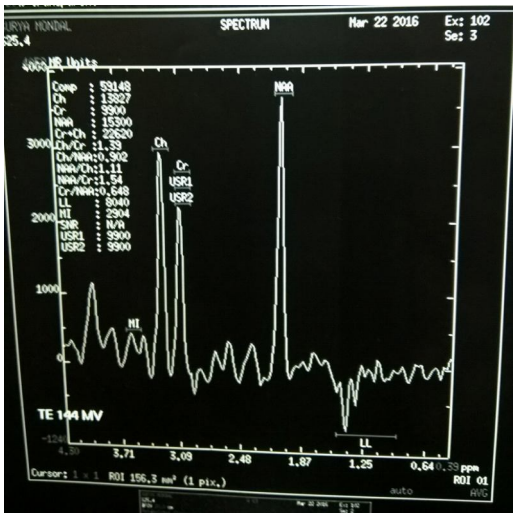

Fig 2. MR spectroscopy showing inverted omega shaped lactate peak in the region of hyperintensity

Discussion

Leigh syndrome is a devastating neurodegenerative disease, typically manifesting early childhood. Since its first description by Denis Archibald Leigh in 1951, it has evolved from a postmortem diagnosis, strictly defined by histopathological observations, to a clinical entity with indicative laboratory and radiological findings. Hallmarks of the disease are symmetrical lesions in the basal ganglia or brain stem on MRI, and a clinical course with rapid deterioration of cognitive and motor functions. Numerous causative mutations in mitochondrial and nuclear genes, encoding components of the oxidative phosphorylation system have been described in the past years[1,2,3]. A genetic diagonosis could not be carried out in the index case due to lack of facilities.

Features of Leigh Disease are delayed milestones, weakness , hypotonia, ataxia, tremor and pyramidal signs and nystagmus.Imaging studies show focal segmental necrosis in thalamus, basal ganglia and tegmental region and peri and paraventricular region.Hypertrophic cardiomyopathy, hepatic failure and renal failure may also be seen[2,3,4,5].

As the index case presented had features of grey matter

degeneration with visceromegaly presenting in early childhood, Rett's syndrome,Menke's kinky hair disease,Neuronal ceroid lipofuscinosis(NCL),Tay Sach disease,MERRF and Leigh disease were considered in the differential diagnosis. Physical characteristics like hair and face as well as sex of the child ruled out Rett's syndrome and Menke's kinky hair disease.Absence of convulsion, retinal pigmentation and optic atrophy ruled out NCL .Absence of convulsion, coarse facies and cherry red spot in eye ruled out Tay Sach disease. Absence of myoclonus and peripheral neuropathy etc ruled out MERRF.The MRI picture, reverse omega shaped lactate peak and elevated lactate and pyruvate levels suggested a diagnosis of Leigh Disease.

Treatment is supplementation with riboflavin, thiamine,carnitine, coenzyme Q, biotin, succinate and idebenone.High fat, high protein and carbohydrate restricted diet is recommended for these children.Different genetic causes and types of Leigh syndrome have different prognoses, though all are poor. The most severe forms of the disease, caused by a full deficiency in one of the affected proteins, cause death at a few years of age[6,7,8] .If the deficiency is not complete, the prognosis is somewhat better and an affected child is expected to survive 6–7 years, and in rare cases, to their teenage years.The index case has been put on carnitine,CoQ,and thiamine supplementation with partial improvement of symptoms.

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An Appeal

Shubho Vijaya and Happy Diwali to you all.

This is fourth time, I am getting the opportunity to communicate with you through this official journal of East Zone Academy of Pediatrics, "Pedi- Info". On the eve of this 24th East Zone PEDICON 2017, I would like to welcome you all in the writing panel of this journal.

In spite of repeated requests, there is a very poor response in contributing the articles for the journals. It will be increasingly difficult for the editors to compile a journal without enough write ups. My request to all of you to please send articles for the regular publication of journal otherwise its future may be in jeopardy.

Case Report

Landau-Kleffner Syndrome : A Case Report.

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Abstract:

A healthy 5 year old boy developed aphasia, attention disorder and hyperkinesia preceded by transient formed visual hallucinations and emotional outburst, immediately after a stressful event of forced separation from his father. EEG showed generalized epileptiform activity. He was diagnosed as Landau-Kleffner syndrome (LKS). CT and MRI of the brain were normal. SPECT showed left mesial temporal hypoperfusion. He improved on antiepileptics and ACTH.

Introduction

Landau-Kleffner syndrome (LKS) is a rare epileptic syndrome characterized by acquired aphasia with epilepsy.

Case report

A 5 year old boy developed episodes of sadness and crying for about one hour, immediately after the departure of his father for higher study. He also had transient formed visual hallucinations for about three to five minutes in the form of 'lion killing his father'. This was followed by inattentiveness, marked receptive and expressive aphasia and hyperkinesia. The child became totally non interactive with his environment within a week's time. He was seen by the author a month later. He could talk well and write simple sentences in his vernacular language before his illness. There was no family history of epilepsy, mental retardation or developmental speech disturbances. On examination, the child was hyperactive but communication ability was interrupted. Cranial nerves were normal and there were no long tract signs.

EEG, done in sleep state, showed generalized epileptiform activity [Fig1]. CT and MRI of the brain were normal. 99m-Technetium brain single photon emission computed tomography (SPECT) study showed left mesial temporal hypoperfusion. He was put on antiepileptic drug (AED) supplemented with a course of injectable adrenocorticotrophic hormone (ACTH). Follow-up

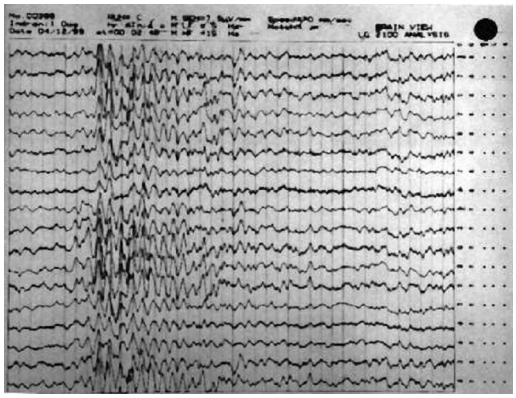

Fig 1. Showing generalized epileptiform activity

revealed significant improvement in ten months, in verbal output, comprehension, attention span and hyperkinesia. Repeat EEG after 8 months showed persistent epileptiform activity.

Discussion

The diagnosis of LKS, in this case, was made on the basis of rapid loss of language in a previously normal child, associated with abnormal EEG compatible with the diagnosis of epilepsy. While this disorder appears to be relatively uncommon, its frequency is questionable due to its unfamiliarity among the professionals and the likelihood of misdiagnosis. It is imperative that communication specialists become alert to the characteristic symptoms of LKS[1,2].

All children diagnosed as LKS have abnormal EEG compatible with the diagnosis of epilepsy, however, only 70% have clinical seizures[2]. The subjective emotional outburst of sadness and crying accompanied by formed visual hallucination in this patient may constitute a seizure. This type of seizure is rare in LKS. The epileptiform activity in LKS is thought to result in a functional ablation of eloquent speech areas. In one study, 13 of the 19 patients had perisylvian magneto-encephalography (MEG) spikes[3].

The cause of the syndrome remains unknown. Relationship between the structural brain lesions and the clinical manifestations of LKS has been tried but not proved[4-6]. Cysticercosis, tumors, arteritis and acute inflammatory conditions have been reported to produce LKS. CT and MRI of the brain did not reveal structural brain lesion in the present case. However, 99m Tc brain SPECT study showed left mesial temporal hypoperfusion. A stressful event, like forced separation from his father, preceding the onset of interruption in communication ability, as observed in this case, has not been reported. LKS might have been provoked by a stressful event, however, this is only conjectural. LKS is difficult to treat. Treatment modalities used include antiepileptic drugs (AED), corticosteroids, intervenous immunoglobulin, ketogenic diet and multiple subpial transections[7]. Accepting the possible role of the epileptic discharge in producing the symptomatology of LKS[8] and the possible autoimmune reations in the etiopathogenesis of LKS[9] the present case was put on AED supplemented with ACTH. He had significant improvement in verbal output, comprehension, attention span and hyperkinesis, in ten months. He had no recurrence of overt seizures, although repeat EEG, eight month after the initiation of the treatment, showed persistent epileptiform activity.

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Case Report

Progeria with Multi-Organ Failure: A Case Report from North-East India

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Abstract:

Progeria is a rare genetic disorder where children are apparently born as normal. They usually develop symptoms at the age of two years reflected by pan los of hair, macrocephaly, cessation of growth and fragile body like elderly. They usually maintain normal mental development but slowly succumb to either kidney failure or cardiovascular complications. Here we report, a male child of four and half years of age who was presented with high grade fever and decreased sensorium. Total baldness was present with absence of eye brows with few amateur eye lashes. There was history of weight loss and stunted growth. X-ray hand with wrist showed presence of 8 carpal bones suggesting bone age of 7 yrs. ECG showed right ventricular hypertrophy with right atrial enlargement. He detoriated and died due to cardio-respiratory failure from multi-organ failure, after one week of hospital stay.

Key words*: Genetic, Multi-organ failure, Progeria*

Progeria is a rare and fatal genetic disorder. Progeria was first described by Jonathan Hutchinson[1] in 1886 and Hastings Gilford[2] in 1897. Hence it is also called as Hutchinson-Gilford Progeria Syndrome (HGPS). The word 'Progeria' comes from the Greek words 'pro' meaning 'before', and 'géras' meaning 'old age'. The disorder has very low incidence and occurs in an estimated 1 per every 4-8 million live births[3]. It is rarely inherited and caused by new mutation in cytoskeleton intermediate filament, Prelamin A gene, LMNA[4].

Case report

A male child born out of a non-consanguineous marriage presented at four and half years of age with high grade fever with cough and cold, and decreased sensorium for one week. The child had vomiting several times. He was referred from Melagarh sub-divisional hospital at 10 am. There was past history of pan loss of body hair and baldness since the age of 18 months, early fall of teeth since 2 years of age. There was history of easy fatiguability since 2 years of age. Perinatal history was uneventful, and the child was delivered by normal vaginal delivery at Melagarh sub-divisional hospital. His mother was 28 years old when he was born. His mother had history of one abortion and one still birth before giving birth to this child. Developmental milestones mainly motor milestones were mildly delayed. Immunisation was complete as per his age. There was history of weight loss and stunted growth. History of recurrent chest infections were given by mother from two and half years of age.

Examination

The child was semi-concious. There was pallor and

moderate dehydration. Total baldness was present with absence of eye brows with few amateur eye lashes. His skin was shiny and scalp veins were visible and also veins of face, dorsum of hands and feet. Relative macrocephaly was seen. Glasglow coma scale (15 point scale) was 5. Anthropometric measurements showed weight 10 kgs, length of 103 cms, head circumference of 48 cms, upper segment 55 cms and lower segment of 48 cms, hence he belongs to underweight category of Protein Energy Malnutrition. His pulse was 90 beats/min, feeble and regular; BP was 80/20 mm of Hg. Oxygen saturation on multi-channel monitor was 95% without oxygen. Cardiovascular system revealed soft systolic murmur. Fundoscopy revealed corneal opacities in both eyes. Oral cavity revealed loose dentures and teeth decay and absent incisors and molars. There was angular stomatitis and oral ulceration. Other systemic findings were within normal limit.

Investigation

Routine hemogram revealed Hemoglobin of 10 gm%, total leucocyte count was 14800/cumm, neutrophils were 76%, lymphocytes were 24%. Total RBC was 2.2 lakhs. MCH, MCV, MCHC were within normal limit. Peripheral smear showed normocytic normochromic RBCs. Blood urea was 50 mg/dl and serum creatinine was 1.9 mg/dl. In liver function tests, alkaline phosphatase was 892 U/l, SGPT, SGOT, serum bilirubin were within normal limits. In electrolyte function tests, serum sodium was 123 mmol/l and serum potassium was 1.62 mmol/l. Serum Cholesterol was 246 mg/dl. In QBC test, malaria parasite was not found. Typhi Dot M test was negative. Chest X-ray was within normal limit. CT-scan brain showed frontal bossing with silver beaten appearance. All cranial sutures were fused. X-ray pelvis was in accordance to his age but there were some osteoporotic changes. X-ray hand with wrist showed presence of 8 carpal bones suggesting bone age of 7 yrs, with some sclerotic changes in the tip of metacarpal bones. ECG showed right ventricular hypertrophy with right atrial enlargement, whereas axis, rate and rhythm were normal.

Management

From the history, examination and investigation, a case of progeria with multi-organ failure was diagnosed. He was managed conservatively by intravenous fluids, injectable potassium. He was given antibiotic and antipyretic for fever. He was given prophylactically aspirin to prevent CVA. Topical antibiotics and vitamins were given for oral ulcer and antibiotic eye drops were given. Unfortunately he detoriated and died due to cardio-respiratory failure from multi-organ failure, after 1 week of hospital stay.

Discussion

Children with progeria are not born with any significant abnormal symptoms. They develop the first symptoms after few months, usually between one and half years to two years of age. Failure to thrive may be the first symptom that may come to notice. Thereafter there is cessation of growth, pan loss of hair, and a special appearance with macrocephaly, retracted jaws, beak shaped nose and prominent eyes. With growing age, the signs and symptoms worsen. Later, the condition causes wrinkled skin, atherosclerosis, kidney failure, loss of eyesight, hair loss, and cardiovascular problems[5]. Many patients have scleroderma like thickened skin conditions. Veins are prominent, especially the scalp veins (made prominent due to baldness and shiny skin). Patients with progeria disorder usually have small, fragile bodies resembling with those of elderly. The face is usually wrinkled, with a larger head in relation to the body. Musculoskeletal degeneration causes loss of body fat and muscle, and stiff joints. Individuals usually retain normal mental development.

In normal conditions, the LMNA gene codes for a structural protein called prelamin A. There is a farnesyl functional group attached to the carboxyl-terminus of Prelamin A. Without its farnesyl group, prelamin A is referred to as lamin A. Lamin A, along with lamin B and lamin C (also called the nuclear v filaments), make up the intermediate filaments of nuclear lamina, which provides structural support to the nucleus[6]. They are involved in the

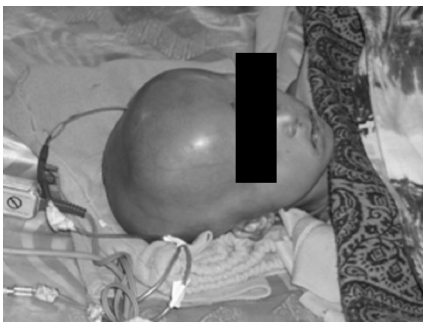
breakdown and reformation of the nuclear envelope during mitosis, as well as the positioning of nuclear pores. A silent point mutation (C1824T) in the lamin A gene, LMNA causes cessation of transcription of the LMNA gene, which creates an abnormal variant of the prelamin A protein whose farnesyl group is strongly adhered, referred to as progerin4, which is permanently affixed to the nuclear rim, and therefore does not become part of the nuclear lamina. Without lamin A, the nuclear lamina is unable to provide the nuclear envelope with adequate structural support, causing problems during mitosis. Progerin may also play a role in normal human aging as per some studies which described it as a biomarker of cellular aging in normal skin[7].

Till date, no treatments have been proven effective for progeria. Most treatment focuses on reducing complications (such as cardiovascular disease) with heart bypass surgery or low-dose aspirin[8]. Growth hormone treatment has been tried[9]. The use of morpholinos has also been attempted in order to reduce progerin production. A type of anticancer drug, the farnesyl-transferase inhibitors (FTIs), has been proposed. A Phase II clinical trial using the FTI lonafarnib began in May 2007 at U.S. National Institutes of Health. In studies on the cells another anti-cancer drug, rapamycin, caused removal of progerin from the nuclear membrane through autophagy[10]. It has been proved that pravastatin[8] and zoledronate[8] are effective drugs when it comes to the blocking of farnesyl group production.

Farnesyl transferase inhibitors (FTIs) are drugs which inhibit the activity of an enzyme needed in order to make a link between progerin proteins and farnesyl groups. This link generates the permanent attachment of the progerin to the nuclear rim. In September 2012 study showed that the cancer drug Lonafarnib (FTI) can be used with definitive result in progeria[12]. It avoids the permanent attachment of the progerin protein to the nuclear rim thus facilitating mitosis.

Fig 1: 1 year age

Fig 2: 18 months with grandparents



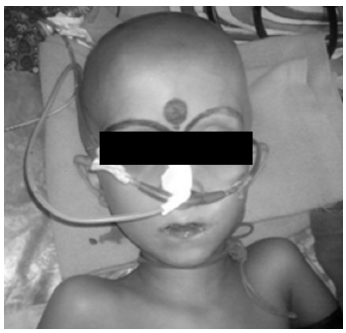


Fig 3,4: Prominent veins on bald scalp and typical appearance of Progeria 4½ years age (1 day before death, 21st Dec 2012)

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Photo Quiz Answer - 1

a) It was hypoplasia of the frontotemporal regions of the cerebral hemispheres, enlarged pretemporal middle cranial fossa subarachnoid spaces, and cyst-like dilatation of the Sylvian fissures giving a classical "**batwing**" appearance.

b) Glutaric Aciduria Type I
High urine 3 OH glutaric acid level confirmed the diagnosis

Case Report

Pediatric Stroke with a Difference

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Abstract:

Although post varicella stroke has been reported since 1900s, most of them have been ischemic stroke. We hereby report a child with post varicella cerebral thrombosis with hemorrhagic stroke with positive evidence of varicella vasculopathy in the background of familial thromophilic state in the form of protein C deficiency with a brief discussion of literature on this association

Key Words : *Pediatric stroke;varicella vasculopathy*

Introduction

The exact etiology and incidence of pediatric stroke varies from country to country. Post varicella stroke have been reported since the 1900s[1]. However, it is post varicella ischemic stroke secondary to varicella vasculopathy which is commoner than hemorrhagic stroke[1]. Herewith we report a boy with post varicella cerebral thrombosis with hemorrhagic stroke in the background of possible familial prothrombotic state with certain unreported associations.

Case report

A 12 year old boy hailing from Nadia was admitted on with complaints of headache for 3 days, weakness of left side of body with deviation of angle of mouth to right for 3 days and one episode of generalised tonic clonic convulsion (GTCS). There was no history of breathlessness, joint pain, bleeding tendencies, or similar episodes in the past. There was a history of fever with vesicular eruptions 12 days ago .The child was a product of non consanguineous marriage and his growth and development was appropriate for age.

On examination the vitals were within normal limit. There was left sided UMN facial palsy with complete left sided hemiplegia, power being 2/5 with brisk deep tendon reflexes. Other systems were within normal limits. The weakness improved over next 72 hours of hospitalisation but there were recurrent episodes of GTCS continued for 3 days post admission ultimate getting controlled by two antiepileptic drugs.

First line investigations for stroke didn't reveal any abnormality which included a normal hemogram and ESR, PT, aPTT and echocardiography. CT Scan of brain [Fig1]revealed a thin subdural haematoma over right high parietal convexity with underlying oedema of the brain tissue. On suspicion of hemorrhagic stroke MRI brain [Fig 2]was done which revealed areas with hypointensity in T1 and hyperintensity in T2 and FLAIR areas involving multiple lobes suggestive of haemorrhagic infarction at (R) fronto-parietal and (L) parieto-occipital lobe. MR angiogram didn't reveal any abnormality but

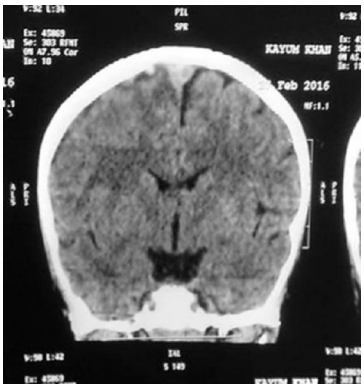

Fig 1: CT scan brain (coronal section) showing thin subdural hematoma over right high parietal convexity with underlying edema of the brain tissue

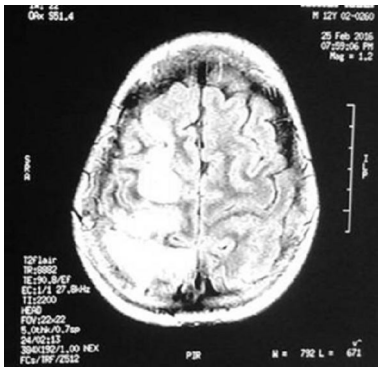

Fig 2: MRI brain hyperintensities in T2 involving multiple lobes suggestive of hemorrhagic infarction at (R) fronto-parietal and (L) parieto-occipital lobe

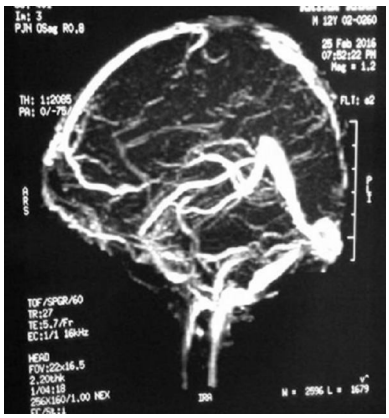

Fig 3: MR venogram showing thrombosis of mid part of superior saggital sinus.

the MR venogram [Fig 3] revealed thrombosis of mid part of superior saggital sinus.

Meanwhile,during the course of admission on day 6 the child developed severe abdominal pain with swelling and pain of bilateral lower limbs, suggestive of deep vein thrombosis (DVT). Although, immobility of the limbs could have caused DVT,we prodded further to enquire if there was any family history of thrombophilia. At this point,it was revealed that his elder brother had DVT after cholecystectomy and presently was on anticoagulants. Doppler study of venous system of lower limbs revealed thrombosis of major veins of legs extending proximally upto left iliac vein. A thrombophilia profile was done in view of such extensive thrombosis which revealed low protein C level that is 11.4% (normal being 70-140%) with normal levels of lupus anticoagulant, antinuclear factor,antithrombin III and protein S level. In view of extensive thrombosis and possibility of thrombotic stroke, low molecular weight heparin (LWMH) was started at a dose of 3mg/kg /day subcutaneous in divided dose. There was a remarkable improvement in terms of improvement in pain abdomen and decline of limb swelling. Slowly therapy was switched to oral warfarin 5mg/day and child was discharged. However, the child came back with reappearance of limb pain and swelling 3 weeks post discharge with PT INR being 1.3 on warfarin. He was treated with LMWH followed by warfarin 10mg/day this time.

On reviewing the history, there was a history of fever with rash, extensive venous thrombosis including thrombotic stroke and family history of thrombosis.Is it a varicella vasculopathy causing the stroke or is it due an underlying familial thrombophilia ,or both factors were contributing to this occurrence of stroke? To explore the role of varicella, CSF study was done which revealed a normal cytology and biochemistry but a high level of varicella zoster IgG antibodies by enzyme immunosorbent assay; CSF/ serum quotient being 1.7 (normal CSFQ ref <1.3).

Discussion

Analysis of literature highlighted the role of varicella in

pediatric stroke. It was found that ischemic stroke in children occurred during recovery from chicken pox, demonstrated with intrathecal production of varicella zoster specific antibody and was attributed to virus reactivation[1]. Vascular stenosis of childhood following varicella infection or post varicella arteriopathy takes a monophasic course, generally with subsequent stenosis regression after arterial ischemic stroke/TIA[2]. Bodensteiner et al[3] showed that primary varicella zoster virus infection with hemiparesis typically occurs approximately 6 weeks after infection. The innervation of the carotid artery and the characteristics of the varicella zoster virus itself may trigger the vasculopathy responsible for this syndrome. In 2009 Ramanan et al[4] had reported a similar incident of post varicella thrombosis with hemorrhagic stroke in a 12 year old boy, where in however, the prothrombotic screen was not done. Post varicella thrombosis has also been associated with factor V leiden mutation as well. Moussalem M et al had concluded that, post varicella thrombosis requires a thrombophilia screening. Varicella vaccination in children with prothrombotic condition is supported[5]. There have been reports of acquired and transient protein S deficiency secondary to presence of anti-protein S in young children at the waning of chickenpox resulting in cerebral thrombosis with spontaneous recovery later.6However, presence of protein C deficiency post varicella as found in our case is yet to be reported.

Ours was probably a case of post varicella cerebral thrombosis with hemorrhagic stroke due to varicella vasculopathy as CSFQ antivaricella antibody was positive in the background of familial thromophilia due to protein C deficiency . The exact contribution of each factor, namely varicella vasculopathy and familial thrombophilic state towards causation of stroke in the index child can only be speculated at this juncture.

Conclusion

To conclude, post varicella stroke in pediatrics is not uncommon. Ischemic stroke secondary to transient varicella vasculopathy is commoner than hemorrhagic stroke following cerebral venous thrombosis. However, diligent search for underlying prothrombotic states in the family is warranted in the later cases.

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Photo Quiz Answer - 2

Drug : Cyclophosphamide

A Surveillance Report

Rotavirus Diarrhea in Under 5 Hospitalised Children with Acute Diarrhea in Meghalaya

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Abstract:

Objective - To estimate the prevalence of rotaviral and non rotaviral diarrhea in children aged 30 days – 60 months hospitalised with acute watery diarrhea in a tertiary care hospital in Shillong, Meghalaya. And to compare the two groups based on their background epidemiological data, clinical features and laboratory parameters.

Setting – 64 bedded pediatrics ward in Nazareth Hospital, Shillong.

Methods – In this analytical cross sectional study carried out over 1 year, all children aged 30 days to 60 months hospitalised with acute watery diarrhea were subjected to Rapid Diagnostic Test for Rotavirus antigen in stool sample and the two groups (rotavirus positive and negative) compared on various background characteristics, clinical features and laboratory parameters using tools for statistical significance.

Results– Out of 170 total cases of acute watery diarrhea aged 30 days to 60 months which were admitted over a period of one year at Nazareth Hospital, Shillong, 100 (58.8 %) were positive for rotavirus. Mean age of presentation was 18.4 months in Rotavirus positive group and 17.7 months in Rotavirus negative group (p = 0.009). Mean duration of fever was 1.2 days in positive group and 1.8 days in negative group (p = 0.041, Z Score 2.04). Mean duration of loose stools 2.02 days (SD 1.34) in positive group and 1.91 days (SD1.71) in negative group (Z score 3.39, p = 0.0006). Duration of hospital stay 2.76 days in positive group and 2.24 days in negative group (p = 0.0004, 95% CI 0.22, 0.81). Mean potassium levels in positive group was 4.38 Mmol/l (SD 0.64) and in negative group 4.81 Mmol/l (SD 0.72) (<0.05).

Conclusion – Study highlights that Rotavirus positive patients were older in age, were admitted in hospital for a longer duration, had shorter duration of fever but longer duration of loose stools and vomiting, were more likely to be from lower economic classes, had less degree of dehydration and lower potassium levels as compared to rotavirus negative patients, this being the first of its kind of study in the state of Meghalaya.

Introduction

Infectious diarrhea is a leading cause of disease and death worldwide. Rotavirus infection is a major cause of acute watery diarrhea in children less than 5 years of age. Rotavirus infection is characterized by diarrhea, fever and vomiting. Stools are watery and rarely contain blood, mucus, or white blood cells. Vomiting often lasts for 2 to 3 days and diarrhea 5-8 days. Vaccination has been shown to be effective against rotavirus infection.

There is very little literature on the prevalence of rotaviral

diarrhea and its associated features, especially in comparison to nonrotaviral diarrhea, in north eastern part of India.

Previous national and international research suggests rotaviral prevalence of 20.2%[1] to 45.6%[2] in children hospitalised with acute watery diarrhea.

The fact that there is wide variation in data about epidemiology of diarrhea caused by rotavirus in different parts of the world, as well as a lack of a comprehensive study to document the incidence and associated features of rotavirus diarrhea in this part of the country, form the basis of the need to conduct this study.

Methods

The study was carried out in Nazareth hospital, Shillong which has a 64 bedded ward for children between 0-18 years. Study was carried out from 1st January, 2015 to 31st December, 2015. A detailed clinical history, general and systemic examination and various relevant laboratory investigations were done.

All hospitalised children between 30 days to 60 months of age with acute onset of watery diarrhea after obtaining informed consent from the father, mother or guardian were included in the study. Exclusion criteria comprised of presence of blood in stools of the patient and duration of diarrhea more than 14 days.

All data was recorded according to a standard questionnaire. Patients were divided in 2 groups – 'Rotavirus Positive' and 'Rotavirus Negative' based on presence or absence of rotavirus antigen in their stool samples, which was determined with Rapid Diagnostic Test, a rapid immunochromatographic assay.

Statistical analysis was done based on chi-square test and Z-Test, as applicable. The associations and correlations were considered statistically significant if the p value was = 0.05. All data were analysed and processed on SPSS Version 22.0 on a Windows 10 operating system.

Ethical approval for the study was obtained from the Institutional Ethical Committee of Nazareth Hospital, Shillong.

Results

The study flow diagram is given in figure 1.

Out of 170 cases, 100 (58.8 %) patients were positive for rotavirus and 70 were negative for rotavirus. No association between sex and rotavirus status was found in the present study. On an average, rotavirus positive group had lesser number of episodes of loose stools per day as compared to rotavirus negative group, but the duration of complain of loose stools was more in rotavirus positive group. Additionally, lesser proportion of patients in rotavirus positive group had dehydration as compared to rotavirus negative group. Three (3) patients of acute watery diarrhea were already vaccinated against rotavirus. All the three patients were negative for rotavirus antigen.

Discussion

In the present study, the prevalence of rotavirus positive patients among those admitted with acute watery diarrhea was found to be 58.8%. This was higher than values obtained in studies carried out by N Teotia et al[3](2014), P Saravanan et al[4](2004), Bahl et al[5](2005), V Vashishtha et al[2],(2016), Kang at al[6], Shu-Yan Yang et al[1] (2014), John et al[7] and Lintao-Sai et al[8](2013) which found prevalence of 25%, 22.6%, 23%, 45.6%, 39%, 20.2%, 24% and 41% respectively.

In the present study, most cases in rotavirus positive group were in age group of 2 – 3 years (48%), and most cases in rotavirus negative group were in age group of 6 – 12 months (44.2%). In comparison, study carried out by Bahl et al[5] found that incidence of rotavirus diarrhea was low during the first 3 months of life, peaked at age 9–11 months, and decreased sharply after age 18 months. While P Saravanan et al[4] found that major proportion of the rotavirus positive cases fell in the age group of 7-18 months (62.5%).

In the present study, it was concluded that rotavirus status of a patient with acute watery diarrhea does not have any sex predilection (p = 0.240). Similar findings were found in studies carried out by Teotia et al[3], Saravanan et al[4], Shu-Yan Yang et al[1].

According to the present study, the mean number of days of admission of a patient in rotavirus positive group was longer (2.76) than the one in rotavirus negative group (2.24) ($p<0.05$). The results obtained were different from studies done by Teotia et al[3] (3.17 vs. 3.18 days) and Shu-Yan Tang et al[1] (5.2 days vs. 5.1 days).

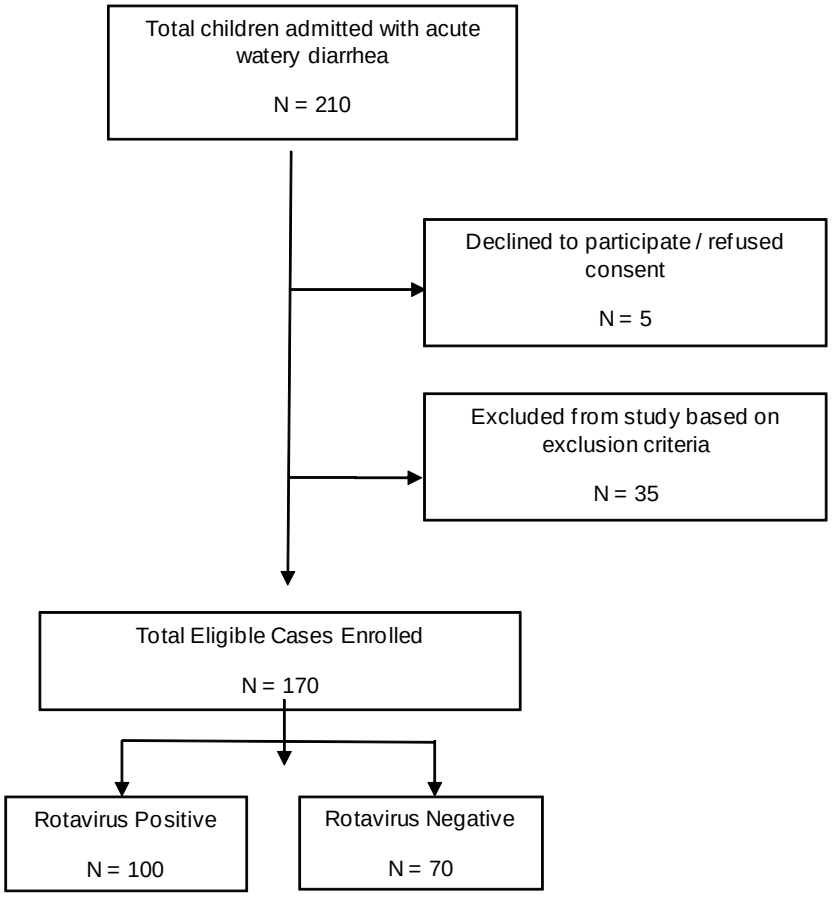

Fig.1: Flow Chart of the cases of present study. Exclusion criteria were presence of blood in stools and duration of diarrhea more than 14 days.

In the present study, 54% of rotavirus positive patients complained of fever as an associated symptom. 70% patients of rotavirus negative group complained of fever. Patients in rotavirus positive group had shorter duration of fever (at the time of admission), as compared to rotavirus negative group (1.2 days vs. 1.8 days) (p = 0.041). Similarly, Lintao-Sai et al[8], Teotia et al[3] and Shu-Yan Yang et al[1] found that fever was found in 85.5%, 72.5% and 94.7% of rotavirus positive patients, respectively.

According to the present study, the average duration of diarrhea (at the time of admission) was 2.02 days for rotavirus positive group and 1.91 days for negative group. Vashishtha et al[2] also found that average duration of diarrhea was more in positive group (6.1 days vs. 4.5 days).

According to the present study, of the rotavirus positive group, 53% patients were dehydrated at admission (3% were suffering from severe dehydration, 50% from some dehydration) and 47% had no dehydration. According to study by Bahl et al[5], none of the hospitalized children with rotavirus diarrhea had disease of mild severity, 73.7% had moderately severe disease, and 26.3% had severe disease.

In the present study, most of the patients in the study group were from Socioeconomic Class 4 (Lower Middle Class), according to Modified Kuppuswamy Scale, 2015,

Table-1: Summary of findings of rotavirus positive cases (N=100) and rotavirus negative cases (N=70). Significant difference was noted with respect to age, duration of loose stools and hospital stay.

CHARACTERISTICS	ROTAVIRUS POSITIVE N = 100 (Mean)	ROTAVIRUS NEGATIVE N = 70 (Mean)	Total Cases N = 170 (Mean)	p-Value
Age (in months)	18.43(SD 8.74)	17.7 (SD 12)	18.6(SD 12)	0.009
Sex	MALES – 52 FEMALES - 48	MALES - 32 FEMALES - 38	MALES – 84 FEMALES - 86	0.240
Duration of Fever(days)	1.2 (SD 1.5)	1.8 (SD 2.1)	1.43	0.053
Duration of Loose Stools(days)	2.02 (SD 1.34)	1.91 (SD 1.7)	1.98	0.00069
Frequency of Loose Stools (episodes/day)	4.48 (SD 2.3)	4.53 (SD 2.75)	4.5	0.9
Duration of Vomiting(days)	1.4 (SD 1.39)	1.36 (SD 1.18)	1.4	0.053
Frequency of Vomiting (episodes/day)	2.75 (SD 2.2)	3.14 (SD 1.7)	2.9	0.07
Hospital Stay (days)	2.76 (SD 1.2)	2.24 (SD 0.77)	2.56	0.0004

Table-2: The present study found that more rotavirus negative patients had some and severe dehydration compared to rotavirus positive group (p = 0.083).

Degree of dehydration	Rotavirus Positive (N = 100)	Rotavirus Negative (N = 70)
No dehydration	47 (47%)	26 (37%)
Some dehydration	51 (51%)	40 (57%)
Severe dehydration	2 (2%)	4 (6%)

Table-3: Rotavirus positive group had higher proportion (73% vs. 72%) of patients from Classes IV, V of Modified Kuppuswamy Socio Economic Scale, 2015 compared to rotavirus negative group(p = 0.001).

Socioeconomic class	Class I	Class II	Class III	Class IV	Class V
Rotavirus positive	0	1 (1%)	26 (26%)	64 (64%)	9 (9%)
Rotavirus negative	0	0	20 (28%)	48 (69%)	2 (3%)
Total cases	0	1	46	112	11

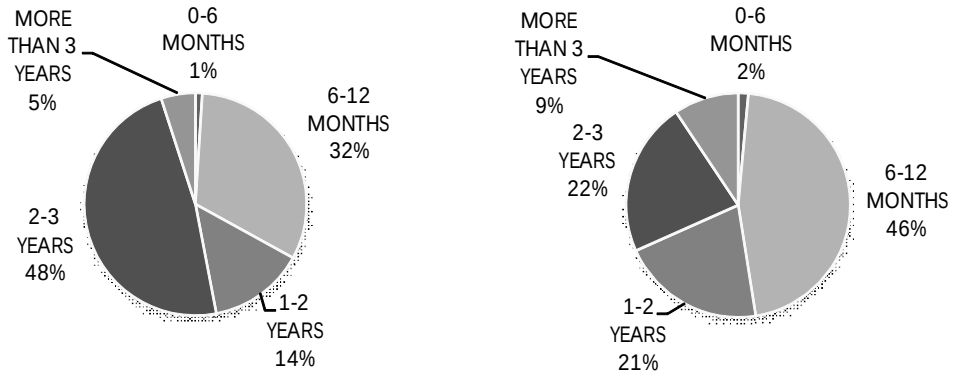

Fig 2. Shows distribution according to age of patients in rotavirus positive and negative group in the present study.

in both Rotavirus positive and negative group. 3% patients of rotavirus positive group and 9% patients of rotavirus negative group were from socioeconomic class V. In the study carried out at Bijnor by Vashishtha et al[2], of all the Rotavirus positive patients, 23% from lower SES and 30% of all rotavirus negative patients were from lower SES

Conclusion

This study shows that prevalence of rotavirus infection in hospitalised patients of acute watery diarrhea, aged 1 month to 60 months, was much higher than values obtained in previous studies. Rotavirus positive patients were older in age, were admitted in hospital for a longer duration, had shorter duration of fever but longer duration of loose stools and vomiting, had less degree of dehydration and were more likely to be from lower socio-economic class as compared to the rotavirus negative patients.

There was no difference between the two groups in terms of sex, frequency of loose stools or vomiting per day or the serum electrolyte profile.

It is desirable to create awareness amongst pediatricians regarding difference in signs and symptoms of Rotaviral diarrhea in this part of the country. Considering the high prevalence of Rotaviral diarrhea, it is recommended that all patients of acute watery diarrhea be tested for rotavirus antigen in stool. Such high prevalence of disease also warrants encouraging vaccination against rotavirus in the community. But this being a hospital based study, more studies are needed at the community level to establish the true association between rotavirus infection and its associated features in the general population.

What this study adds –

1. *Prevalence of rotavirus diarrhea amongst hospitalised under five children with acute diarrhea has been estimated for the first time in a study from the north east*
2. *Prevalence is much higher than expected. There are several differences in clinical features from past published literature from other parts of India and the world.*

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Conflict of Interest– Nil

Reference

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Announcement

Fact Sheet

Safety Issues in Pediatric Care – A Shared Responsibility for all the Current Status in the North East and the Road Map Ahead

Rashna Dass Hazarika
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Introduction

As we stand at the threshold of welcoming the year 2018, the total population of our vast country stands at a staggering 1.35 billion. What is interesting is that of this 1.35 billion mass of humanity, 41% falls in the age group below 20 years, 50% between 21 to 59 years of age and a mere 9% above 60 years of age[1]. What is also mind blowing is that about 51 babies are born every minute in this country. Seventy percent of this population still remains concentrated in the rural areas and 30% live in the urban regions. So even as you finish reading the first few lines of this article, hundreds of babies have already been born and huge numbers of children are added to this humungous population on a daily basis raising concerns about their health, safety and shared responsibility in comparison to adults. Is the government or the private players doing enough for the children of this country? The entire population of 1.35 billion is thus composed of humans at different stages of life and the pediatric age group forms an important part of this human mass. So before we discuss about safety or health issues in paediatrics, one has to be very clear of which are the individuals which fall in to this category.

Pediatric population : definition

Even today a lot of confusion exists as to which age group would define the pediatric population and this confusion exists even in the medical community. The first attempt to define the pediatric age group was tried in 1938 by the Council of Health in the USA[2]. In 1938, the American Academy of Pediatrics took formal action and defined the age limits of paediatric practice as follows: "The practice of pediatrics begins at birth and extends well into adolescence and in most cases will terminate between the sixteenth and eighteenth year of life". By 1972, it was further recognized that this period may begin with conception and pregnancy and extend well in to the 21st year of life[3]. The World Health Organization (WHO) further states that an adult is one who is older than 19 years of age unless the national law of the country defines a person being an adult at an earlier age, a little ambiguous to say the least. In India we still do not have a uniform law defining a child as different laws define children at different age ranges. However the clearest definition comes from the United Nations Convention on the Rights of the Child where the first article clearly states that all under the age of 18 years has all the rights in the convention[4]. So for all practical purposes, pediatric age group is now considered to be from 0-18 years of age.

Safety issues in pediatrics

When one starts to discuss about safety issues in pediatric care, one has to first understand that care of the ill child is a continuum of care involving prevention, transport, emergency care, critical care services and rehabilitation (Fig 1) and we need to discuss each of these components one by one.

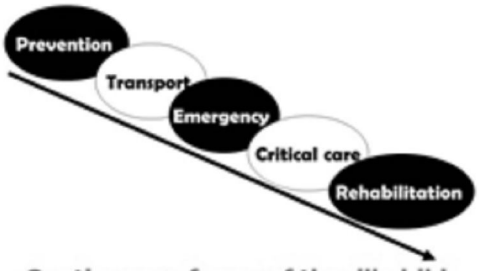

Fig 1: Continuum of care of the ill child

Pre-hospital transport:

Let me begin with by looking at the state of pediatric transport in this part of the country. Pre-hospital transport is critical to patient care and still remains dismal in spite of introduction of 108 ambulance services in this part of the country for a number of years now. Most of these ambulances are manned by paramedics unfortunately not trained to resuscitate children or newborns, and the equipment in these ambulances are also questionable as far as their availability and functionality for this age group is concerned. Even today most of these ambulances only serve to function as a government provided for free transport vehicle. As far as neonatal transport is concerned, it remains grossly neglected and under developed and there is no pre-hospital stabilization, no continued care during transport, absence of trained paramedics and people continue to use their own vehicles in more than 50% of the times as per the Government's own survey[5]. My own online survey among 54 colleagues, working in both the government and private sector, in north east (NE) India in September-November of 2015 reveal that there are no skilled personnel who accompany these newborns during transport, most are transported without proper thermal control, 85% of the babies received were both hypothermic and hypoglycaemic at arrival, only 52% had intravenous lines in situ, transport distance ranged from 50 to more than 200 kms, 81% had no referral slips and transport ventilators are totally non-existent[6]. The end result is a high pre-hospital mortality.

Pediatric emergency care services:

As of today, only two medical college hospitals in the entire NE region (Assam Medical College and Jorhat Medical College) have designated area to receive exclusive pediatric patients. There are only 2 exclusive private pediatric hospitals in the entire region (one in Imphal and one in Shillong) with 24 x 7 pediatric services. The rest of the hospitals, be it government or private, have a common emergency shared with other departments. In such shared emergency services, firstly the sick child may or may not receive adequate and timely response and secondly the ill child may or may not be seen by a paediatrician. So the end result is a lack of focussed care, loss of the most important golden hour of response time and action in the emergency care and finally an increased mortality and morbidity.

Pediatric critical care services:

This is generally a neglected branch of pediatrics not only in NE India but also in the entire country. The first exclusive Pediatric Intensive Care Unit (PICU) in the government sector was started by me at the North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences (NEIGRIHMS), Shillong in March 2007. This is a 13 bedded unit with all tertiary care facilities such as invasive procedures, mechanical ventilation, dialysis etc. Subsequently PICUs were established in 2009 at the Guwahati Medical College, 2010 at the Jorhat Medical College, and then at medical colleges at Silchar, Agartala and Imphal which are functional. Facilities have also been set up at the Fakiruddin Ali Ahmed Medical College at Barpeta and at the Assam Medical College but as of writing this article these units are yet to become functional. Many district hospitals in Assam have also been provided with designated PICU areas but are yet to be functional. In the private sector, PICU facilities were started in a few hospitals in Guwahati in the early 2000's due to individual efforts of a few pediatricians but most of them were operating in adult ICUs and with limited resources. It is important to note that PICUs require dedicated staff for handling of these critically sick children.

After hours laboratory facilities for the critically sick child:

Even today 24 x 7 x 365 laboratory facilities are not available at many hospitals and even if available in a few hospitals, they are restricted to a few tests, and the cost and reliability becomes an issue as most of these would be reported by technicians and not confirmed by consultants. Therefore the end result is a delay in decision making, sub-optimal care and exposing the patients to additional risks.

Hence overall all these children who require critical care in PICUs are actually subjected to sub-optimal care due to a lack of PICUs and after-hours laboratory facilities. The main reasons for this are because children have different range of diseases compared to adults with different pathology, the care is more family centric rather than patient centric where empathy for parents becomes a major issue and a more humanized approach is required. The skills for nursing care therefore need to be more refined; assessment scales, fluid and drug management protocols are totally different from adults as they are not whole numbers but are dynamic and dependent on body weight measurements.

Rehabilitation

In contrast to adults, it is important to remember that 90% of PICU or NICU graduates can lead normal lives. Therefore nursing care during hospital stay is to be done carefully to prevent injury or deformity. Rehabilitation therefore forms an important component of pediatric care but it is often forgotten or not advised correctly.

Prevention and safety issues

Preventive care is the most important cog in the wheel of the continuum of the care of the sick child. It actually starts in utero and is better appreciated now as we have come to understand that most diseases in adults have origins in fetal or intrauterine life, and maternal age and health, toxin exposure and maternal nutrition hold the key to prevention of many adult diseases. In addition to this, neonatal, infant and child nutrition and immunization are other key preventive strategies. So have we really succeeded in these preventive strategies? Probably yes to some extent as we have been able to eliminate polio and neonatal and maternal tetanus. But overall the immunization coverage still remains grossly inadequate and below expected targets and diseases like measles still kills about 92,000 children every year in India[8]. Intrauterine growth retarded babies and premature births are on the rise due to multiple factors and are an important cause of future metabolic diseases in adults and a strain on the resources of the country. Health education still remains sub-optimal and we have new dangers such as effects of radiofrequency waves from mobile phones and television on pregnancy and language development in children looming in the horizon[7].

So therefore when we look at each aspect of the continuum of the care of the sick child from prevention to rehabilitation, safety concerns abound in all areas.

Pediatric care - A shared responsibility for all

Next in line for discussion is whether pediatric care is a shared responsibility for all? When we look at the current status of health care delivery, it is interesting to note that most of the resources in most hospitals, be it public or private, are focussed on the 59% of the population above 20 years of age (i.e the adult and geriatric population). Most upcoming hospitals are concentrating on providing speciality and super specialty tertiary care services for patients with cardiac, kidney or metabolic diseases (diabetes mellitus and its complications) and cancer. Most of these centres are again urban concentrated. In doing so, an important issue that has been forgotten is that the country's majority population and the young vibrant future population are the 41% below the age of 20 years i.e. the child and the adolescent. If we look at healthcare distribution in the entire NE states of India both in the government and private sector (Fig 2 and 3), we have a total of approximately 5 crores population with 31208 and 9976 hospital beds in the government and private sectors respectively. This amounts to 1 bed for every 1200 persons.

If we look at the status of pediatric beds, we find that in the whole of the NE region, there are approximately 1261 designated pediatric beds (mostly in medical colleges),

Healthcare distribution-Govt...

State	Population (census 2011)	Districts	Hospital beds
Assam	31,169,272	27	14408
Tripura	3,671,032	8	3395
Meghalaya	2,964,007	11	3320
Manipur	2,721,756	9	2839
Nagaland	1,980,602	11	2168
Arunachal Pr	1,382,611	16 (20)	2180
Mizoram	1,091,014	9	1311
Sikkim	607,688	4	1587
Total	**~ 5 crores**		**31208**

Fig 2: Healthcare distribution NE states – Government

Healthcare distribution -Pvt...

State	Population (census 2011)	Districts	Hospital beds
Assam	31,169,272	27	6000
Tripura	3,671,032	8	1000
Meghalaya	2,964,007	11	600
Manipur	2,721,756	9	776
Nagaland	1,980,602	11	300
Arunachal Pr	1,382,611	16 (20)	300
Mizoram	1,091,014	9	500
Sikkim	607,688	4	500
Total	**~ 5 crores**		**9976**

Fig 3: Healthcare distribution NE states - Private

neonatal intensive care/ sick newborn care unit (NICU/ SNCU) beds about 1319 (in medical colleges and district facilities) and PICU beds about 146. In the private sector, majority of the hospitals have no designated separate pediatric beds except for the 2 pediatric hospitals in Shillong and Imphal and one or two charitable hospitals in Assam and Mizoram, most would have 4-10 bedded NICUs depending on their obstetric load, PICU in a handful of hospitals totalling about 400 pediatric beds, 665 NICU/SNCU beds and 60 PICU beds. Thus for 41% of the 5 crore population in NE India i.e. for approximately 2 crore population of patients below 20 years of age, we have 1 bed per 15,860 persons compared to 1/1200 overall population and 2/1000 as recommended by WHO. So as far as shared responsibility of pediatric care is concerned, we can see very clearly that there is a gross inequality of healthcare distribution. Pediatric care started off as an off shoot of adult medicine and continued to remain so for many decades and even today it is neglected in the planning stage of all major healthcare facilities both in the government and private sectors. The skewed development of the SNCU/NICU services in both the government and private sectors is not because of special love of the newborns but because is in reality driven by 2 main reasons – the first and foremost is a poor infant mortality rate which has forced the government facilities to try and improve the survival of the newborn baby, and secondly in the private sector because of pressure of obstetric cases. In spite of all efforts, about 6,60,000 newborns continue to die every year in India[8].

The roadmap ahead!!

To tackle the problems still persisting with pediatric care in north east India and probably the country, there are few issues which need to be taken care of at all levels of the continuum of care of the ill child. Let us start with preventive care – here it is important to understand that only a health pregnancy can lead to the delivery of a healthy child and therefore services for the mother and the child has to be provided under one roof. For this one needs to develop the concept of standalone maternal and child health (MCH) centres. The first and only government sector MCH centre developed as early as 1935 is the Ganesh Das Hospital in Shillong but even after 82 years of its existence, this hospital still does not have a separate pediatric emergency or PICU services and only a recently developed SNCU unit. The MCH building at the Jorhat Medical College is a step in the right direction. In the private sector few maternity hospitals are present but the pediatric component can be developed better. There is therefore a great need to develop standalone tertiary level MCH centres both in the government and private health sector with a greater focus on developing specialty and subspecialty pediatric services both at the rural and urban regions.

As far as pediatric and neonatal transport is concerned,

there is a huge lacunae as this is still in a very primitive stage of development. A lot can be done as far as pre-hospital, inter-hospital and intra-hospital transport is concerned along with skilled manpower development.

As far as emergency and pediatric critical care is concerned, health planners need to understand that children need a different approach and handling to make these services exclusive for children, separate from adult ICUs and in such a way that parents develop both trust and comfort to walk in to these centres at any time of the day or night.

As far as rehabilitation is concerned, it needs more focus as 90% of the children can lead normal lives after surviving a critical illness. The government has already come up with the concept of the comprehensive rehabilitation centres known as the Rashtriya Bal Swasthya Karyakram (RBSK) but the private sector is lacking in this and needs to do a lot in this regard.

Is it possible to provide exclusive high quality pediatric health services in India?

Yes, there are ample examples in India, both in the government and private sectors of exclusive pediatric centres which have come up and carrying out extremely good work as far as providing tertiary specialty and sub-specialty care in various branches of paediatrics. Some outstanding examples are the Advanced Pediatric Centre at PGIMER, Chandigarh, the Kanchi Kamakoti CHILDS Trust hospital, Chennai and nearer home the Institute of Child Health, Kolkata and the Dr B C Roy Postgraduate Institute, Kolkata. However most of these centres are urban centric as of now.

Conclusion

As far as safety and shared responsibility in pediatric care is concerned, a lot is lacking that can be corrected. Opportunities galore and one only needs to grab them and take action to improve the care and safety of this neglected but important population group of the country. Investment in the health of this 2 crore population in north east India is actually smart business sense especially for the private players and its high time the hospital planners start incorporating this in the design and services of any new hospital setup or while thinking of up gradation of old hospitals rather than concentrating only on provision of curative services for non-communicable diseases to the small 9% of the geriatric population. In fact early intervention can prevent morbid adult non communicable diseases and thus save on long term health expenditure on chronic diseases.

I end with these beautiful but relevant lines from Gabriel Mistral (1948) which sums up our responsibility towards each and every child from 0-18 years of age: ***We are guilty of many errors and many faults, but our worse crime is abandoning the children, neglecting the foundation of life. Many things that we need can wait. The child cannot. Right now is the time his bones are being formed, his blood is being made and his senses are being developed. To him we cannot answer "Tomorrow". His name is "Today"!!!***

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1995-96	Dr LSN prasad	Dr Dilip Mukherjee	Dr Tapan kr Ghosh
1997	Dr A C Barua	Dr R K Satapathy	Dr A Swain
1998	Dr Dilip Mukherjee	Dr Tapan kr Ghosh	Dr Ritabrata Kundu
1999	Dr R P Sinha	Dr Tapan Kr Ghosh	Dr Ritabrata Kundu
2000	Dr S K Behra	Dr N P narain	Dr Neelam Verma
2001	Dr Bikas Roy	Dr N P narain	Dr Neelam Verma
2002	Dr Ksh Chourjit Singh	Dr Shabina Ahmed	Dr Sanjeev Chakraborty
2003	Dr S P Srivastava	Dr Shabina Ahmed	Dr Sanjeev Chakraborty
2004	Dr Gokul Borah	Dr Krishna Kumar	Dr O P Mahansaria
2005	Dr Tapan Kr Ghosh	Dr Krishna Kumar	Dr O P Mahansaria
2006	Dr B R Master	Dr Th Nabachandra	Dr S K Laishram
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2017	Dr P S Chakraborty	Dr N L Bhaumik	Dr Nihar Ranjan Das

Fact Sheet

How Television Affects Your Child

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Television has become a part and parcel of our life. It has a great influence on children from an early age and it will affect childrens' cognitive and social development.

Usually children do not become a constant viewer until they are 2-3 years of age. They do not watch the TV program constantly because their span of attention is very short and hence they get easily distracted.

Now a days the working parents are so busy that they don't have enough time to spend with their kids. The mother has to do cooking and look after the children simultaneously. There may not be servant also. So she switches on the TV and puts the child in front of it. Not only that some of the mothers feed their children showing the TV images. Slowly it becomes a habit. After some time the kid becomes addicted to television . And finally the mother blames the child for watching TV.

The first 2 years of life are considered a critical time for brain development. TV and other electronic media can get in the way of exploring , playing and interacting with parents.

American Academy Of Pediatric (AAP) recommended that kids under 2 years old should not watch any TV and those older than 2 years watch not more than 1-2 hours a day of quality programme.

Researches have confirmed that children watch TV about 2 hours a day. The time spent in front of TV significantly increasing through childhood and it peaks at about 4 hours per day during middle school.

In the beginning children only glance at TV content, later on they start understanding it, their attention grows.

TV watching is not interactive and hence many a times speech is delayed.

A child of 4-5 years of age may not be able to fully understand a programme but he does form some impression of what he has viewed. He obviously can't differentiate between fantasy and reality. To him everything on TV is true to the life . A violent scene could be as terrifying to him as violence in real life.

By 8-12 years of age, the child's understanding of TV improves considerably. Now he is able to draw conclusion from certain programmes. There is considerable improvement in the memory of programme content.

Children who constantly spend more than 4 hours per day watching TV are more likely to be overweight. Eating snacks while watching TV and reduction in activity results in obesity.

Kids who view violent acts are more likely to show aggressive behavior but also fears that the world is scary and something bad will happen to them.

Elderly people , specially who are retired and over 65 years of age, watch TV even more than the children. They simply spend all their time inside the homes and their only means of entertainment is TV watching. Often

there is a clash between children and the grand parents regarding the channel they prefer . The elderly would prefer the serials, while children want their favourite cartoons. In apartments where there is limited space, TV viewing by the grandparents disturbs the study time as the children get distracted easily.

There is a positive aspect to this situation as well. The presence of older people will prevent the children from watching the horror and adult movies.

Educational TV programmes can enhance the cognitive development, specially the preschoolers, in reading, readiness and acquisition of vocabulary.

For older children TV is an excellent source of current events, science, history, politics and entertainment.

Recommendations For TV Viewing

TV should not be kept in childrens' room.

Television should not be viewed for more than 1-2 hours a day.

Parents should also watch the shows their children are watching.

Television should not be put on during meal time.

Parents should limit their own television viewing.

School should play a positive role in utilizing creative and beneficial aspects of TV.

East Zone Mid Term CME 8,9 July 2017, Bhubaneswar, Odisha

Criteria of Award

1. Purbanchal Shishu Visheshagna Award
ONE : If membership strength is up to 500
TWO : If membership strength crosses 500

2.IAP Purbanchal Pioneer Award
ONE : If membership strength is up to 500
TWO : If membership strength crosses 500

CRITERIA for Selection

Purbanchal Shishu Vishesagna Shiromoni Award

1.Age above 58 yrs- 3 yrs gap from Pioneer award
2. Central IAP Membership for at least 15 yrs
3.Must be member of IAP State branch 15yrs.
4. Attended 1 EZ conference out side own state.
5.Any of following criteria is Mandatory –
   a). Teaching experience of 20 yrs.
   b) Out standing contribution in IAP, State or IAP, East Zone namely, ornamented the Post of President IAP East Zone/ his or her State, active involment in IAP activities for 15 yrs.
   c) Published 20 papers I Medical Journals of National /International or State.
   d) Did out standing research activities

Purbanchal IAP Pioneer Award

1. Age not exceeding 55 yrs except Org Secretary of EZ Pedicon who is automatic choice
2. Central IAP Member
3. Must be the member of State of East Zone – at least 10 yrs.
4. Must attended 3 East Zone conference excluding own state.
5. Application must forwarded by State branch of IAP.
6) Any one of following is mandatory-
   a) IAP activities of State- President , Vice President, Secretary, Treasurer, Jt Secretary, Org. Secretary of State Pedicon, Editor at least for one term or EB Member at least for 2 yrs.
   b) have involvement in IAP East Zone activities office bearer for atleast one term & /or Executive member or Scientific Committee member (any one) of IAP CC at least 2 terms) & /or Central Executive for 2 terms
   c) 10 published papers in medical journals.