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How To Avoid Medical Negligence

While there has been an increase in both the number and quantum of claims filed against doctors in recent years, the threat of litigation now hang over the head of the modern doctor... So it is better that we take precautions to prevent the cases of negligence rather than fighting them out in the court of law. The following steps may be helpful in avoiding cases of negligence:

1. Establish good patient-caregiver relationships

Litigations against medical practitioners are rising as the relationship between doctor, patients and Caregiver is deteriorating.

Open and honest communication is vital to fostering positive relationships. It also helps you to know your patients better, and helps their family members gain a good understanding of care and procedures. It all adds up to earning the trust of your patients. And when you have a patient's trust, they are less likely to bring a lawsuit against you if something goes wrong. Open communication is something that ought to be practiced actively.

Maintain a humanistic approach and attitude of care and sympathy when you dealing with a patient. When patients get the feeling that the doctors care genuinely and have nothing but their best interests in mind, they would be more forgiving of inadvertent errors. While a poor outcome to the treatment in itself may not result in a claim of medical negligence. A poor outcome and poor communication is a combination that could land the doctor in trouble. You shouldn't be lethargic about addressing complaints and the expectations that you set for the patient and the family members should be realistic. Maintain a good rapport with the patient, patient family member and professional Colleagues.

2. Don't criticize colleagues

It has been observed that majority of cases are because of the instigation and criticism by some of our own colleagues. We must verify the actual facts and situation in the particular case before making any comment. Avoid adverse criticism of other physician even with casual remark publicly

3. Hospital environment

Technical, scientific advances and corporate hospital culture has resulted in huge expectations in patient's mind. Your hospital staff should be properly trained and adequately experienced. Junior staff and locums should be qualified (vicarious liability) and starting of group practice is a better option in this era.

Keep the hospital instruments and equipments in proper and working condition. Instruments must be properly sterilized. . If proper and adequate facilities are not available, a timely referral to well equipped center is a desirable alternative.

Finances and bills should be properly explained and informed at the time of admission or even before admission. A patient cannot be detained on the grounds of non-payment of hospital charges. Doctors can take advance or fee from the patient before starting the treatment

4. Consult other professionals when in doubt

Pertinent to uncommon ailments and procedures, the probability of things going wrong is higher and thus greater care must be taken to guard against the allegation'. Any expert opinion should be taken if needed and advice shall be recorded in writing

If there are two accepted schools of thought, any particular method may be adopted by the doctor in the patient. This is not negligence. This principle was derived in "Balam's Test".

5. Record keeping

Always ensure adequate record keeping. This provides a practical advantage when evidence is adduced in court – written records are almost always more credible. Proper record must include history, examination, investigation reports and treatment adopted. A well maintained record can be a friend of the doctor in an hour of crisis. Don't try to manipulate the records.

6. Valid consent

The consent is obtained after explanation and reasonable understanding of facts. Consent should be informed and preferably in writing. The consent should preferably be taken in presence of witness (two from patient side and two from hospital side).

Sometimes a child is brought to pediatricians by neighbors (parents of child are immediately not available for consent). In such situations if it is a genuine and real emergency, the child can be managed even without consent. The neighbors consent doesn't have legal validity.

The patient and relatives should always be informed regarding nature of the disease, proper line of treatment. Complications or adverse reactions of drugs and prognosis. While managing a case, give guarded prognosis. You should note down the exact risks and complications of a particular procedure (including refusals). Suppose a patient of hydro-pneumothorax is admitted. The patient is not taken for surgery and dies of respiratory failure. The doctor pleads that surgery was not done as patient or relatives didn't given consent. In this case doctor has to prove that consent was refused (hence it should always be in writing whether the consent is given or refused);

7. Stay updated in standards and training

One possible issue in litigation concerning a negligence claim is whether the doctor has followed current standards of practice or whether s/he treated the patient based

on an outdated standard. Law doesn't expect one to know each and every detailed advance but one must know the things expected of an average prudent man.

8. Insurance

Professional indemnity cover may be helpful whenever there is litigation in the court. It may not be helpful in minimizing the damage to the reputation of the practitioners, but it may help as far as financial liabilities are concerned. The insurance companies may also help by providing services of advocates and legal experts.. It is preferable to know someone in the insurance company so that the dealing and processing of the matter becomes easy. The disadvantage of insurance is that: (i) if the patients or relatives know that the doctor is insured then they may be encouraged to go in for the litigation; and (ii) many times even the insurance companies are willing for the out of the court settlement which is cheaper and of "least resistance" to them rather than fighting out the case.

9. Counter compensation suits

Counter suits have started in western countries and it has been observed that this has resulted in decreased incidences of negligence suits against doctors. Counter suits by doctors against the patients may be helpful in minimizing cases of negligence.

Contributory negligence, known complications, unexpected results, difference of opinion and emergency care are the usual defenses in case of negligence.

Contributory negligence- Sometimes the unexpected results may not be only due to negligence of the doctor but also due to negligence of patients or relatives. This is known as contributory negligence

Example

(1) Failure to follow the instructions given by the treating doctor; the patient was instructed to remain nil orally but the patient was given orally.(2) Investigations advised by the doctor are not done by the patient; (3) Patient fails to take advice of a specialist (for example, in case of acute abdomen or head injury, the Pediatrician has referred to a surgeon but the patient fails to take such a consultation); and (4) patient leaves the hospital against medical advice.(5) patient was instructed to come for regular follow-up but did not comply.

Known complication:

Some drugs or procedures have known complications. For example, anaphylaxis after Penicillin injections is a known complication. A doctor cannot be held responsible if proper sensitivity test was done and all measures for management of anaphylaxis were readily available in the hospital.

Unexpected results:

According to Sir Williams Osler (a USA Physician), medicine is a "science of uncertainty and art of probability". All persons in community do not acquire all diseases. There is always a probability of acquiring a disease. Some acquire the disease while others do not, inspite of being exposed in an equal amount. Every individual has

different body response not only to disease but also to treatment. Hence there is uncertainty in every case. Hence we talk of "most probable diagnosis" and "most probable outcome" of a disease. A doctor can't be held negligent only because there was unexpected outcome

Difference of opinion :

There may be a difference of opinion amongst doctors while treating a case. This is not negligence. This principle was derived in "Balam's Test

Emergency care :

Cardio-respiratory arrest is an emergency situation. Some-times fracture of ribs can occur during cardio-pulmonary resuscitation. A doctor can't be held negligent for causing fracture of rib in such a situation.

> Good, compassionate behavior, proper record maintaining and a valid consent may be of great help whenever there is a case of negligence

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Bedaquiline in Pediatric Drug Resistant Tuberculosis: Is this the Next Big Answer?

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Definitive diagnosis of tuberculosis has always been more challenging when it comes to paediatric population. On top of that crisis, increase in multidrug resistant and extremely drug resistant strains of Mycobacterium tuberculosis bacilli is now an emerging problem globally. In 2016, World Health Organisation (WHO) stated that during 2015-16, nearly half a million people got infected with multidrug-resistant (MDR) tuberculosis (TB) globally[1]. Among these, nearly 32,000 cases were found to have occurred in "less than 15-years-age" population[2]. Extremely drug-resistant (XDR) TB (MDR TB additionally resistant to a fluoroquinolone and a second-line injectable drug) statistics were not very clear in paediatric population due to very limited data. More than 33% MDR TB cases were estimated to be resistant against fluoroquinolone, a second-line injectable drug or both[3].

Worldwide among all the age groups, overall favourable outcomes were observed in 48% patients with MDR-TB and only 22% patients XDR TB in 2011[4]. MDR-TB poses several difficulties for treating paediatricians as they are dealing with infants, children and adolescents. First, treatment options are quite limited. Even if a promising drug comes in use its evaluation in adult patients beforehand takes lot of time. Second, among the disease burdened countries access to appropriate laboratories and effective treatment regimens is still a big problem. Third, drugs used in combination for MDR TB often causes adverse reactions (example: Injectable drug causing deafness in more than 25% children in one cohort)[5].

As the need of newer more effective and less toxic anti TB drugs was felt, researchers started experimenting with several molecules. Among them oxazolidinones, diarylquinolines, nitroimidazopyrans, ethylenediamines and benzothiazinones showed promising results[6]. Bedaquiline or Sirturo (developed by Janssen Therapeutics under the name R207910 or TMC207) is the first novel drug that was approved by US Food and Drug Administration in 2012 and also in Europe in 2014, almost about 40 years after approval of rifampicin[7].

Bedaquiline belongs to group diarylquinolines which is very much close to quinolones but instead of inhibiting DNA gyrase, this novel

group of drugs inhibit mycobacterial adenosine triphosphate (ATP) synthase. They disrupt energy production and intracellular metabolism of both intra and extracellular mycobacterium by interfering with proton transfer chain[8-10]. This action of bedaquiline is more or less specific for mycobacterial ATP synthase activity as human mitochondrial ATP synthase is 20,000 times less sensitive to it[11].

Bedaquiline has activity against both drug sensitive and drug resistant TB bacilli. Minimum inhibitory concentration (MIC) studies showed better potency against drug sensitive strains than isoniazid or rifampicin. It acts in similar fashion against bacilli resistant to first line drugs (all five included) and moxifloxacin[8].

This is an oral drug, has good absorption from the gut, metabolised by hepatic CYP3A4 (also with help of CYP2C8 and CYP2C19), excreted in faeces, is highly protein bound (>99.9%), has a long terminal half-life owing to redistribution from different tissue compartments[12]. It has got no significant drug interactions with isoniazid, pyrazinamide, ethambutol, kanamycin, ofloxacin or cycloserine[13]. Dose adjustment should be considered while co-administering with lopinavir or ritonavir[14].

Bedaquiline is currently in phase III trial[15]. As per WHO interim guidelines, it may be considered when there is difficulty to construct an effective 4-drug regimen using other drugs or in case of fluoroquinolone resistance. But limited data about the drug prompted WHO to declare that it should not be currently used in case of paediatric population[16].

Bedaquiline has shown to increase hepatic transaminase like many other first line antitubercular drugs. Prolongation of QT interval and need for regular ECG monitoring for corrected QT is another issue to consider. Although some studies have reported increase mortality in bedaquiline group compared to placebo there is considerable doubt whether this increase is directly attributed to bedaquiline use itself[17]. We have to wait for completion of phase III trial and its outcome for this. In low-resource settings, the higher cost of the drug is also a concern for its routine use (24 weeks course costing US$ 28,400 in an adult case)[18].

There are reasons that we are getting hopeful about this drug. Up to 66% of all patients in a large retrospective cohort study have been shown to be benefited from addition of bedaqiline or delamanid to their regimen[19]. The US CDC stated that bedaquiline may be an alternative for children and adolescents when treatment options are limited due to resistance to second line drugs[13]. A recent study which collected data from children and adolescents with advanced resistance to second line drugs showed promising outcome with good compliance. This study concluded that bedaquiline is safe in children>12 years of age with proper monitoring. Although prolongation of QTcF was noted in handful patients with concomitant administration of other cardiotoxic drugs, no patient required bedaquiline cessation[20]. Increasing accessibility to bedaquiline, according to an international group of paediatric TB experts, may reduce the need of second line injectable drugs with resultant irreversible toxicity[21]. So, hopefully after phase III trial results are at hand and also the Janssen group study among<18 years patients publish their results, we may find a valuable drug to fight multidrug-resistant tuberculosis globally.

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A Case of Bronchiolitis Obliterans

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Background: Bronchiolitis obliterans is a disease that results in obstruction of the smaller airways of lungs due to inflammation.

Case characteristics:A 20month old child presented with persistent cough and breathing difficulty and was later diagnosed as Bronchiolitis obliterans.

Message: The case highlights the need for increased awareness for this not so rare disorder.Early detection may help in prevention of irreversible bronchopulmonary damage.

Keywords:Bronchiolitis obliterans,adenovirus,HRCT,Lung biopsy,Bronchiolitis obliterans Syndrome(BOS),Bronchiolitis obliterans organizing pneumonia(BOOP)

"Bronchiolitis obliterans (BO), a chronic obstructive lung disease of the bronchioles and smaller airways, results from an insult to the lower respiratory tract leading to fibrosis of the small airways".Causes include post infections particularly following adenovirus and also post transplantation.In the southern hemisphere and Asian people,post infectious Bronchiolitis obliterans is quiet common.

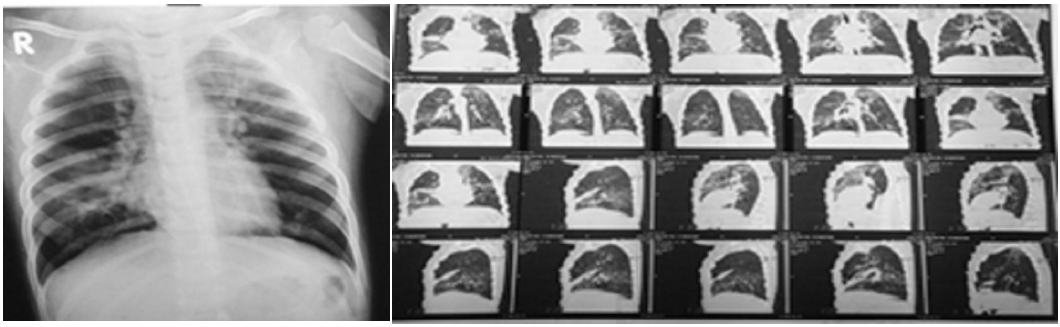
Case report

A 20months old girl born out of non-consanguineous marriage belonging to lower socioeconomic status with father being a smoker,referred to our institute from PHC with the history of cough and cold for 5 days.

She received treatment for LRTI for 10 days and was discharged after recovery with minimal cough.

She was readmitted after a month with similar complaints of persistent cough and difficulty breathing. Physical examination revealed signs of pneumonia with respiratory distress.Chest XRAY showed consolidation over right lower zone and left Upper zone and CT Thorax showed air space opacity at lateral segment of right middle lobe with faint ground glass attenuation of right anterior basal and left upper lobe and lingular segment with lobulated peripheral hypodense area features suggestive of bronchiolitis obliterans. Routine investigations were unremarkable including tuberculin test and CBNAAT for gastric aspirate, which only revealed fungal hyphae.

She was started on steroids and bronchodilators along with antifungal and was discharged on bronchodilator with ICS.



Patient is on regular follow-up and is on ICS and SABA.

Discussion

The clinical differentials in this young girl wereBronchial Asthma/ Multitrigger wheeze,chronic bronchitis or pneumonia

Bronchiolitis obliterans is a relatively rare and severe form of chronic obstructive lung disease due to insult to terminal airways leading to inflammation and scarring.The end result of this disease is necrosis and obliteration of smaller airways.

Causes include post respiratory tract infection,connective tissue disease,toxic fume inhalation,drugs like penicillamine, cocaine, stevenjohnsons syndrome andpost lung transplantation .Very few studies have been done on bronchiolitis obliterans.Vaibhavet alhave shown that there is a possible role of mechanical ventilation in bronchiolitis patients.Colom et al had a finding that adenovirus infection and mechanical ventilation were significant risk factors-34% of patients with post infectious bronchiolitis obliterans required mechanical ventilation with only 3% of controls.Bronchiolitis obliterans syndrome is a clinical entity following lung transplant and is probably the cause for limiting the long term survival of transplantation.

Abnormal repair due to epithelial damage is characteristic of BO.There is concentric narrowing of the distal airways.

"Bronchiolitis obliterans organizing pneumonia(BOOP) is a fibrosing lung disease that includes features of BO with extension of the inflammatory process from distal alveolar ducts into alveoli and proliferation of fibroblasts".

Clinical features include cough, fever, cyanosis, breathlessnessand respiratory distress.Physical examination includes wheezing or crackles.Probably the diagnosis towards bronchiolitis obliterans goes only when there is no response to prolonged oral/ inhaled bronchodilators.

Chest Xray may be relatively normal. Sometimes areas of atelectasis and consolidation are seen.

Ventilation-perfusion scans reveal a typical moth eaten appearance of multiple matched defects.V/Q scan helps only in objective assessment of compromised areas of the lungs.

HRCT is the non invasive diagnostic investigation. It defines the nature,location and distribution of bronchopulmonary lesions. Usually alternate areas of hypo and hyper attenuation in the form of mosaic pattern is seen.Also there will be bronchial wall thickening and air trapping.

Open lung biopsy or transbronchial remains the gold standard for diagnosis although it is not commonly done due to invasiveness and more chances of complications.

Early recognition helps in preventing irreversible damage.

In spite of recent advances,the overall mortality is very high in patients with bronchiolitis obliterans.No definitive treatment exists.Administration of immunomodulators, corticosteroids,fluticasone-azithromycin-montelukast (FAM) combination may be effective.Cidofovir being an antiviral agent is under research which might help in post adenovirus BO.Extra corporeal photopheresis and lung transplantation are also done in severe cases.

The disease has a high mortality rate although the best prognosis is that of post infectious bronchiolitis obliterans.

Further Readings :

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Parenting : Doing Right or Wrong

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Parenting a child rearing is the process of promoting and supporting the physical, emotional, social and intellectual development of child from infancy to adulthood. Parenting refers to the intricacies of raising a child and not exclusively to the biological relationship.

The most common caretaker in parenting is the biological parents of the child in question, although others may be like older sibling, a grandparent, a legal guardian, uncle or other family member or a family friend. Government and society may also have a role in child rearing.

In many cases planned or abandoned children receive parental care from non parent blood relations. Others may be adopted, raised in foster care, or placed in an orphange. Parenting skills vary and parent with good parenting skills may be referred to as 'good parents'.

Parenting styles vary by historical time period, race, ethnicity, social class and other social features.

Additionally, research has supported that parental history both in terms of attachments of varying quality as well as parental psychopathology, particularly in the wake of adverse experience, can strongly influence parental sensitivity and child outcome.

Social class, wealth, culture and income have very strong impact on what methods of child rearing parents use.

Cultural values play a major role in how a parent raises their child. However, parenting is always evoling as times, cultural practices, social norms and tradition change. In psychology, the parental investment theory suggests that basic difference between males and females in parental investment have a great adaptive significance and lead to gender differences in matting propensities and preferences. A family social class plays a large role in the opportunities and resources that will be available to a child. Working class children often grow up at a disadvantage with the schooling, communities and level of Parental attention available compared to middile class or upper class. Also lower working class families do not get the kind of networking that the middle and upper classes do through helpful family members, friends and community individuals or group as well as various professionals or experts.

A parenting style is indicative of the overall emotional climate in the home. There are different type Parenting styles.

(1) Authoritive Parenting

It combines a medium level demands on the child and a medium level responsiveness from the parents. They rely on positive reinforcement and infrequent use of punishment. Parents are more aware of a child feelings and capabilities and supports the development of childs autonomy within resonable limits.

(2) Authoritarian parenting styles

They are very rigid and strict. High demands are placed on the child but there is little responsiveness to them. They have non negotiable sets of rules and expectations are strictly enforced and require rigid obedience. Punishment is often used to promote future obedience.

(3) Permisive parenting:

In these settings of parents, a child's autonomy and freedom is highly valued and tend to rely mostly on reasoning and explanations.

(4) Uninvolved Parenting:

An uninvolved or neglectful parenting style is when parents are often emotionally or physically absent. They have little to no expectation of the child and regularly have no communication, children of uninvolved parents suffer in social competence, academic performance, psychological development and problem behaviour. Skills : Parenting skills are the guiding forces of a "Good parents" to lead a child into healthy adults.

They influence on development, maintenance and cessation of children's negative and positive behaviours. Parenting takes a lots of skill and patience and constant work and growth. The cognitive potential, social skill and behavioral functioning a child acquire during the early years are fundamentally dependent on the quality of their interations with their parents.

Research classifies competence and skills required in parenting as follows :

1. Parent child relationship skills

Quality time spent, positive communications and delighting affection.

2. Encouraging, desirable behaviour

Praise and encouragement, attention, facilitating engaging activities.

3. Teaching skills and behaviour, being a good example, incidental teaching, benovolent communication method of the skill with role playing and other method, communicating logical incentives and consequences.

4. Managing behaviour

5. Anticipating and planning

6. Self regulation skills

7. Mood and coping skills.

8. Partner support skills

What is good parenting :

Being a good parent means you need to teach your child the moral in what is right and what is wrong. Setting limits and being consistent are the keys to good discipline. Be kind and firm when enforcing these rules. Focus on the reason behind the child's behavior.

Good Parenting Tips :

Good parenting is hard work. A good parent strives to make decision in the interest of the child. A good parent doesnot have to be perfect. No one is perfect, no parent is perfect. No child is perfect either, keeping this in mind is important when we set our expectations. But it doesnot mean that we should not work towards our goal.

10 Tips on improving parenting skills -

1) Modeling

2) Loving

3) Positive parenting

4) Being a safe heaven

5) Communicating and integrating

6) Reflecting

7) Your own well being

8) No spanking

9) Keeping perspective

10) Take a shortcut

What is bad parenting?

It is a series of actions that can seriously harm that childs demeanor and psychology. Bed parenting is not restricted to s single act, it is collection of these acts that are usually what contributes to a harmfull effect on the child.

Signs of Bad Parenting :

There are several actions and incidences that could make you a bad parent. Here are a few examples of bad parenting that you must avoid at all costs

1) Reprimanding the child excessively

2) Discipling the child in front of everyone

3) All advice, no encouragement

4) With holding affection

5) No rules

6) Lack of support

7) Comparing

8) Not proud of his achievements

9) Criticizing tone

10) Not respecting his feelings

11) Being a poor example

12) No choice

13) Too much pampering

14) Overprotective

15) Lack of trust

16) Not giving tour time.

Impact of bad parenting on children :

Bad Parenting can have a lasting adverse impact on your child in terms of behaviour and psychology. Few effects of bad parenting -

1) Antisocial Behaviour

2) Poor resilience

3) Depression

4) Aggression

5) Lack of empathy

6) Difficulty with relationships

Few tips of Positive Parenting :

1) Don't lose your temper or yell at your child. You are only showing the kind of behaviour you want to discourage in your child.

2) Dont tell your child how to do things, Tell her what to do, you will be surprised at how she finds her own ingenious methods of doing them.

3) Dont shield your child from every upsetting situation. It will make him unfit to handle the reality of life.

4) When you discipline your child, make it clear that it is the behaviour that you are condemning, and not him.

5) Never use fear to make your child do something. It could lead to phobias and emotional imbalance later in life. Love, not fear, is the key to a child's healthy development.

6) Respect your child irrespective of his age or size. He too has self esteem, he too has feeling like you.

7) Respect the viewpoint of our child. His saying 'No' to you doesn't mean he is undermining your authority but has a view different from yours.

8) If you want your child to accept his mistakes, first you apologize. Be humble

when you talk to servants of the house if you wish your child to be a good human being. Remember, you are your child's role model.

9) Never talk about your child's teacher with disrespect. The moment the child see his parents make fun of calibre or the authority of his teachers, his listening towards his teachers stops. And so does his learning.

10) It is important to develop in your child the ability to take right decision. But it is important to develop in him the courage to face life when the decision goes wrong.

11) A little patience can make all the difference between wanting to be good parents and actually being one. Whenever you are about to lose patience with your child, just pause and remember your own childhood.

12) A time will come when your child will spend more time with TV, friends, clothes than with you. Accept and respect these changing needs of your child. It certainly doesnot mean that love has vanished.

Happy Parenting

Relax and enjoy it.

MDR TB in Children – An Overview

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Background

Tuberculosis managementover the years has focused more on adults leaving children inappropriately attended as medical practitioners considered children of little epidemiologic significance. This is thought to be an under estimate considering the challenges faced in diagnosing TB in children.

With the emergence of Drug resistant TB, children are victims of contacts and poor case control of adult TB cases. This pool of cases will defeat the ultimate aim of eliminating TB.Although MDRTB is a microbiological diagnosis, children should be treated empirically according to the drug susceptibility result of the likely source case, as often cultures cannot be obtained from the child.

MDRTB treatment in children is guided by the same principles, using the same second-line drugs as in adults, with careful monitoring for adverse effects. Pragmatic and effective infection control measures are essential to limit the spread of MDR-TB.

Key words – MBR TB , Nucleic acid amplification test , Shorter MDR regimen

Introduction

According to WHO- 2015 the estimated number of global TB burden is 10.4 million cases of which it is estimated that over 67 million children are infected with TB and therefore at risk of developing disease in the future – 5 million cases with Isoniazid resistance and 2 million cases with MDR; 100,000 with XDR.MDR-TB in children is mainly the result of transmission of a strain of M. Tuberculosis that is MDR from an adult source case, and therefore often not suspected unless a history of contactwith an adult pulmonary MDR case is known. Every year 25,000 children develop MDR TB and 1200 XDR TB. More than half of the global burden of MDR TB is in three countries - India, China and the Russian federation.

Terminology

Rifampicin-resistant TB (RR-TB)

- Resistant to at least rifampicin
- Need second-line treatment similar to MDR TB patients

Multidrug-resistant TB (MDR-TB)

- Resistant to at least isoniazid and rifampicin

- Need second-line treatment

EXTENSIVELY DRUG-RESISTANT TB (XDR-TB)

- Resistant to any fluoroquinolone and any of the second–line anti-TB injectable agents (i.e. amikacin, kanamycin or capreomycin).

When To Suspect MDR TB

Most important point in suspecting MDR TB is the history of close contact with a drug resistant TB case , infact any child diagnosed as TB should be considered as a potential drug resistance. Treatment failure cases , all retreatment cases , no sputum conversion even after 2 months of ATT , extensive disease at the start of treatment , all HIV patients with TB , extrapulmonary TB not responding to standard ATT regime are all candidates of suspicion.

Factors Responsible For Drug Resistance

- Unreliable treatment regimen by doctors – lesser number of drugs , inadequate dosage /duration
- Addition of a single drug in a failing regimen
- Easy availability of drugs in the private sector
- Poor drug supply
- Poor quality of drugs

How To Approach A Case of MDR TB

A careful history i.ehistory of contact with MDR TB case is critical information.Clinical examination and investigations relevant for suspected PTB or EPTB.It is important to try to get samples for culture and DST (drug sensitivity tests). Respiratory specimens may be obtained by gastric aspirates,induced sputum and/or nasopharyngeal aspirates. Bronchoalveolar lavage offers no advantage over less invasive methods. Older children (>6-8 years) can often expectorate sputum. Moreinvasive methods for obtaining specimens may be justified in children with extrapulmonary TB, for example fine needle aspiration biopsy or formal biopsy from peripheral lymphadenitis,or pus swab if a draining sinus has formed. Other specimens that should be obtained are cerebrospinal fluid in TB meningitis, pleural or pericardial fluid if effusions are present, ascitic fluid, ear swabs in chronic otorrhoea, bone marrow aspiration if disseminated TB is suspected and biopsies/ swabs from other areas such as abscesses or osteoarticular TB.HIV testing is to be considered in all MDR TB cases. Failure to respond to TB treatment should consider HIV-related lung disease that is not TB as well as the possibility of MDR TB.

Culture yieldsare poor with solid media, and culture and DST results could takefrom 6 weeks to 4 months. Automated liquid broth media, such asthe Mycobacterial Growth Indicator Tube (MGIT) 960 system(Becton-Dickinson, Sparks, MD) improved culture yields andreduced time to culture and DST results (10-14 days in specimenswith high organism loads). However, in children with paucibacillaryTB, culture and DST results can still be delayed for 6-8 weeks.These systems are expensive and need well equipped laboratoriesand technical expertise. More rapid culture and DST methods, such as themicroscopic observed drug-susceptibility (MODS) assay, in whichculture and DST is performed at the same time and results areknown within 7-14 days shows benefit, but have not beenimplemented widely.Since many of the genes encoding

resistance have been determined, nucleic acid amplification tests(NAATs) offer great promise for rapid and accurate diagnosis (XPERT MTB/ RIF)

WHO Guidance On The Management of TB In Children

- Do not add a drug to a failing regimen.
- Treat the child according to the drug susceptibility pattern (and using the treatment history) of the source case's M. Tuberculosis strain if an isolate from the child is not available.
- Use at least four drugs certain to be effective.
- Use daily treatment only; directly observed therapy is essential.
- Counsel the child's caregiver atevery visit, to provide support, advice about adverse events and the importance of compliance and completion of treatment.
- Follow-up is essential: clinical, radiological and bacteriological (mycobacterial culture for any child who had bacteriologically confirmed disease at diagnosis).
- With correct dosing, few long-term adverse events are seen with any ofthe more toxic second line drugs in children

Choosing The Mdr-tb Treatment Regimen In Patients With Confirmed Rifampicin-resistant Or MDR-TB

Criteria:Do Any Of The Following Apply ?

- Confirmed resistance or suspected ineffectiveness to a medicine in the shorter MDR TB regimen (except isoniazid resistance)
- Exposure to >1 second-line medicines in the shorter MDR TB regimen for >1 month
- Intolerance to >1 medicines in the shorter MDR TB regimen or risk of toxicity (e.g. drug-drug interactions)
- Pregnancy
- Extrapulmonary disease
- At least one medicine in the shorter MDR TB regimen not available in the programme

If Yes

Indivisualised(conventional) MDR-TB Regimen

Intensive Phase

Duration: Up To 8 Months

Composition: 4 Or More Second-line Drugs

Continuation Phase

Duration: 12 Months Or More

Composition: 3 Or More Second-line Drugs

If No

Shorter MDR TB Regimen

Intensive Phase

Duration: 4-6 Months

Composition: 4 Or More Second-line Drugs

Continuation Phase

Duration: 5 Months

Composition: 3 Or More Second-line Drugs

Shorter MDR Regimen

The shorter MDR-TB treatment regimenswere standardized in content and duration and split into two distinct parts. The first is anintensive phase of four months (extended up to a maximum of six months in case of lack ofsputum smear conversion) and includes the following drugs: gatifloxacin (or moxifloxacin),kanamycin, prothionamide, clofazimine, high-dose isoniazid, pyrazinamide and ethambutol.This was followed by a continuation phase of five months with the following medicinesgatifloxacin (or moxifloxacin), clofazimine, pyrazinamide and ethambutol (prothionamide).

4-6 KM-MFX-PTO-CFZ-Z-HHIGH-DOSE-E / 5-6 MFX-CFZ-Z-E

Longer MDR Regimen

A regimen with at least five effective TB medicines during theintensive phase is recommended,including pyrazinamide and fourcore second-line TB medicines-one

chosen from group A, one from group B, and at least two from group C. If the minimum number of five effective TB medicines cannot be composed as given above, an agentfrom group D2 and other agents fromgroup D3 may be added to bring thetotal to five. The regimen may be further strengthened with high-dose isoniazid and/or ethambutol.

resistant TB or MDR-TB, who have not been previously treated with second line drugs andin whom resistance to fluoroquinolones and secondlineinjectable agents has been excluded or isconsidered highly unlikelya shorter MDR-TB regimen of 9–12 monthsmaybe used instead of a conventional regimen.

GROUP A FLUOROQUINOLONES	LEVOFLOXACIN MOXIFLOXACIN GATIFLOXACIN	
GROUP B SECOND-LINE INJECTABLE AGENTS	AMIKACIN CAPREOMYCIN KANAMYCIN (STREPTOMYCIN)	
GROUP C OTHER CORE SECOND-LINE AGENTS	ETHIONAMIDE / PROTHIONAMIDE CYCLOSERINE / TERIZIDONE LINEZOLID CLOFAZIMINE	
GROUP D ADD-ON AGENTS (NOT CORE MDR-TB REGIMEN COMPONENTS)	D1	PYRAZINAMIDE ETHAMBUTOL HIGH-DOSE ISONIAZID
	D2	BEDAQUILINE DELAMANID
	D3	P-AMINOSALICYLIC ACID IMIPENEM-CILASTATIN MEROPENEM AMOXICILLIN-CLAVULANATE (THIOACETAZONE)

Conclusion

A history of contact with a suspected or proven drug resistant TB case is critical in evaluation and management of child with suspected DR TB or an asymptomatic child contact. Children with suspected DR TB should be referred if possible to specialist for investigation (culture and sensitivity), management (hospitalization for injectables) and monitoring for toxicity to second-line drugs. HIV test is routine in evaluation of suspected DR TB and early ART improves outcome.

In patients (adults and children) with rifampicin-

There is very little evidence and no agreed consensuson the use of or optimal regimen for preventive therapy for asymptomatic contacts of drug resistant TB cases One approach is not to provide any preventive therapy and opt for careful, regular follow-up informing the contact about possible symptoms of TB and that prompt evaluation is needed if symptoms develop An alternative approach, especially for high-risk contacts such as HIV-infected or young children, is to choose a preventive therapy regimen that includes at least two drugs to which the DR TB index case is susceptible or naïve and treat for at least 6 months.

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Congenital Arhynia: A Rare Malformation of Nose

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Abstract:

Congenital arhynia is an extremely rare malformation consisting of absence of external nasal structures and nasal passages. Fewer than 40 cases has been reported. Midface hypoplasia may accompany arhynia, or ear, palatal, ocular or facial abnormalities. It causes severe breathing and feeding difficulties. We are reporting a case of arhynia with bilateral microopthalmia and semilobar holoprosencephaly.

Background

Arhinia is a rare anomaly in which a total absence of the nose and parts of the olfactory system occurs. It is frequently associated with various multiple central nervous system (CNS) and somatic anomalies of different degrees of severity, with high mortality rate. The anomalies that have been found to be associated with arhinia are: lack of olfactory bulbs and nerves, missing paranasal sinuses, high arched or cleft palate, various eye anomalies, low set ears - all in a very high incidence. Various degrees of CNS malformations have been found in part of the cases. Somatic anomalies have been reported in 50% of the cases. In two cases, chromosome 9 anomalies have been reported. A classification is suggested in which arhinia is classified into arhinia (total absence of the nose and rhinencephalon) and partial arhinia (partial absence of the nose), each may or may not be associated with other malformations (facial, CNS and somatic).

Case Report

A full term male baby delivered by normal vaginal route in a peripheral hospital was referred to AIIMS, Patna for abnormal facies and respiratory distress. The baby cried immediately after birth and had a birth weight of 3 kg. Mother was a booked case with 5 antenatal visits to the doctor. There was no history of any maternal illness or drug ingestion except for iron, folic acid and calcium in the pregnancy. All three trimesters were uneventful. Only 2 antenatal USG were available, first of the early pregnancy for confirmation of pregnancy and estimation of expected date of delivery , and the other of last trimester of pregnancy (34 weeks) which suggested mild polyhydroamnios.

On examination, the baby had bilateral micro

opthalmia with inward deviation of upper and lower lid margin and eyeball could not be visualized on external examination, absent nose with a single nasal pit through which he was breathing, cleft palate, tongue tie and large prominent low set ears. Trigonocephaly was present with head circumference of 30 cm. (<3rd centile). Micropenis was present with penile length of 1.5 cm.Baby was active and alert, with normal tone, color and temperature. Capillary refill time was less than 3 seconds, Heart rate 120/min and Respiratory Rate of 34/min. significant xiphoid retraction was present. Examination of cardiovascular, respiratory and abdomen yielded no other significant findings.

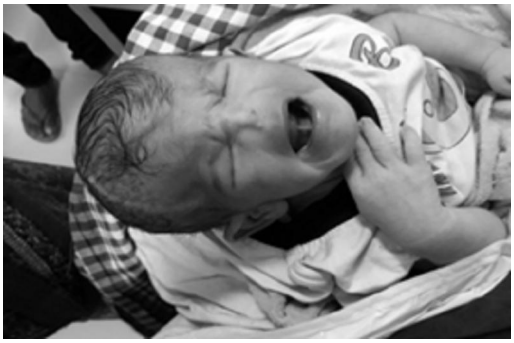

Figure 1. Arhinia

CT scan of head showed absence of septum pellucidum, with rudimentary lateral ventricle horn (monoventricular appearance) , absence of anterior part of corpus callosum, and absence of interhemisheric fissure and falx cerebri except in superior part. Thalami and basal ganglia were separated with fusion of frontal lobe of brain anteroinferiorly. Small cystic lesion of size 6.2 x 5.1 cm was seen in medial canthus of right eye. Eyeballs appeared small with hypoplastic optic nerve. Cribriform plate and nasal bone was not seen. Sphenoid bone appeared hypoplastic. Soft tissue density was seen at the site of nasal cavity suggestive of dysplastic brain tissue. Trigonocephalic skull was seen. Maxillary sinus was not developed. Based on above findings, impression of Semilobar holoprosencephaly was made. Ultrasound abdomen was normal. Karyotyping was 46 (XY). Echocardiography and chest radiography of the neonate was normal.

Discussion

During facial development, cranial neural crest cells migrate from the trigeminal nerve region to the face .Development of the nose and nasal cavities occursbetween the third and tenth weeks of gestation. Nasal Placode appear as local thickening of the surface of the ectoderm and develop from the frontal process advancing laterally between the medial and lateral nasal processes.

The nasal placodes invaginate at the fifth week to form the nasal nuclei. Nostrils develop from the nasal nuclei. The nasal nuclei migrate posteriorly to form nasal cavities. Meanwhile, the oral and nasal cavities are separated bybucconasal membranes that will rupture at the seventh or eighth weeks to form the posterior nares. The nasal septum develops at the ninth week when the palate and inferior septum unite and form the secondary palate. Hard palate development finishes at the eighth or ninth week, and the soft palate finishes at the 11th or 12th weeks[1].

The pathogenesis of arhinia is not clearly understood. The proposed mechanism may be a developmental defect in the medial and lateral nasal processes or overdevelopment and early fusion in the medial nasal processes[2]. Arrest of absorption of the nasal epithelial plates at the 13th through the 15th week may be another possible mechanism. Abnormal migration of neural crest epithelial cells is another possible explanation.

The complete absence of the nose from birth (congenital arhinia) was first described in the French literature in the 1800's. A handful of additional patients with congenital arhinia, some with and some without eye defects, were reported in the early to mid-1900's. Dr. James Bosma, a pediatrician and researcher at the National Institute of Dental Health, however, was the first to observe that these patients frequently had genital and reproductive hormone problems[5]. In his 1981 report, he described two unrelated males (who were first reported by plastic surgeon Dr. George Gifford et al., (1972) with congenital arhinia, eye defects, and genital defects (small penis and undescended testes at birth, with no spontaneous sexual maturation)[3]. Nearly every patient with congenital arhinia has been the first and only one affected in his or her family. However, there have been several reports of multiple patients within the same family, the first by Klaus Ruprecht and Frank Majewski (1978) describing two German sisters with congenital arhinia and eye defects[4]. Several terms have been used in the past for this syndrome to acknowledge the work of Drs. Gifford, Bosma, Ruprecht, and Majewski.

Although researchers have been able to delineate a recognizable syndrome with characteristic or "core" symptoms, much about this disorder is not fully understood. Several factors including the small number of identified cases, the lack of large clinical studies, and other factors have prevented physicians from developing a complete picture of associated symptoms and prognosis. Therefore, it is important to note that affected individuals may not have all of the symptoms discussed below, or may have symptoms that are not discussed. Every case is unique and the disorder can be different in one child when compared to another.

Central nervous system malformations such as absent/hypoplastic corpus callosum, nasalmeningocele/encephalocele, and absent olfactory bulbs and nerves have been reported in association with congenital arhinia. The present patient had holoprosencephaly, which is a complex brain malformation of the developing forebrain resulting from incomplete midline cleavage of the prosencephalon and associated with neurologic impairment and dysmorphism of the brain and face occurring between the 18th and 28th days of gestation. Variations in the severity of craniofacial anomalies may be observed. The most severe facial phenotypes include pronounced microcephaly, cyclopia, synophthalmia, and a proboscis. Less severe facial phenotypes may include microcephaly, hypotelorism, midface hypoplasia with a flat nasal bridge, cleft lip and/or palate, ocular colobomas, and a single maxillary central incisor. It is known that a spectrum of craniofacial anomalies ranging from small and flat nose to arhinia may accompany holoprosencephaly in approximately 80% of the affected individuals. Therefore, radiological evaluation of arhinia should identify anatomic relationships and associated malformations in detail. CT, with preferably three-dimensional(3D) images, contributes valuable visualrepresentations of the bony anomalies, asin this patient. Semilobar holoprosencephaly, presumably a frontal encephalocele, nasal bone hypoplasia and large bony defect of the cribriform plate were demonstrated with a 3D CT in the present patient.

In 2017, two independent teams of researchers discovered that the gene that is altered in most patients with Arhinia Microopthalmia syndrome is the SMCHD1 gene[6]. This syndrome is usually caused by a spontaneous (de novo) change in SMCHD1

that occurs in the egg or sperm cell. In such situations, it is not inherited from the parents. Rarely, the abnormal gene can be inherited as an autosomal dominant trait.

Most genetic diseases are determined by the status of the two copies of a gene, one received from the father and one from the mother. Dominant genetic disorders occur when only a single copy of an abnormal gene is necessary to cause a particular disease. The abnormal gene can be inherited from either parent or can be the result of a new mutation (gene change) in the affected individual. The risk of passing the abnormal gene from an affected parent to an offspring is 50% for each pregnancy. The risk is the same for males and females.

Researchers believe that having a change in SMCHD1 is necessary but not sufficient to develop syndrome. This is because there are families, for example, where a child inherited a change in SMCHD1 from his mother who has a very mild form of BAM (for example, no sense of smell but no other defects) or has no medical problems at all. This suggests that the child has a change in a second critical gene, which may have occurred spontaneously or may have been inherited from the other parent, in this example, the father. This type of inheritance, called digenic inheritance, occurs when a change in more than one gene is required to cause disease. Researchers are still trying to identify these other genes.

The SMCHD1 protein is a gene repressor. This means it has the ability to turn other genes off. It is possible that the changes that occur in SMCHD1 in patients with BAM syndrome cause other genes that are important for developing a normal nose and eyes to be turned off at the wrong time. However, more research is needed to understand how changes in SMCHD1 activity cause BAM syndrome.

Several of the same changes in SMCHD1 that cause BAM have also been shown to cause a rare form of muscular dystrophy, called facioscapulohumeral muscular dystrophy type2[7]. FSHD2 patients have not been reported to have any nose, eye, or reproductive problems, and researchers are still trying to understand if some BAM patients will develop signs of FSHD2 as adults, since FSHD2 is an adult-onset condition, with an average age of onset of 26 years.

There are no known environmental exposures during pregnancy that cause BAM. However, studies in animals have suggested that high blood sugar, alcohol, and retinoic acid may cause holoprosencephaly, a severe congenital disorder whose features may overlap with those of BAM (e.g., absent nose, anophthalmia or microphthalmia, cleft lip or cleft palate, hormone problems).

Conclusion

Congenital arhynia is an extremely rare condition. Facial anomalies and other distant concomitant anomalies could be present. These patients experience serious problems with regard to an open airway and feeding. Other associated problems regarding Central Nervous System malformations may be present. Though , the disorder can be detected antenatally by anomaly scan, not every case needs termination. Detailed study is required to characterize the spectrum of disorder and its varied presentation.

Consent

A written detailed informed consent is taken from the parents for publication of this case report and its accompanying images.

Conflict of Interest: None

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Announcement

Laboratory Diagnosis of Malaria

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Malaria diagnosis still remains a challenge is most of the countries. Lack of infrastructure and expertise leads to presumptive diagnosis based exclusively on the clinical symptoms. Various clinical algorithms has both poor specificity and positive predictive value. They invariably lead to over treatment of malaria in endemic areas and missing the diagnosis in low transmission areas. Indiscriminate use of antimalarials leads to increased drug pressure which results in widespread resistance to antimalarials. Hence every effort should be given for a parasitological diagnosis of malaria before commencing treatment. However, in severe life threatening malaria presumptive treatment may be started before confirmation after collecting blood for examination.

Parasitological diagnosis include light microscopy and rapid diagnostic tests (RDTs). As treatment of malaria has become expensive due to use of artemisinin based combination therapy (ACT) parasitological diagnosis beside saving cost has the following advantages :

(i) Improved care owing to certainty of diagnosis

(ii) Search for alternative diagnosis in parasitological negative cases.

(iii) Reducing unnecessary use of antimalarials

(iv) Confirmation of treatment failure

(v) Improved health information

Microscopic diagnosis

Conventional light microscopy by an expert microscopist of a well prepared and stained blood film remains the "gold standard" for detecting and identifying malaria parasite

Collection of blood sample :

Blood should be collected as soon as malaria is suspected irrespective of fever and not necessarily only at the height of fever but definitely before administration of antimalarials which alter the morphology of parasites. Blood should be obtained from finger tip or earlobe as these capillary rich areas contain greater density of developing parasites. Blood obtained by veni-puncture should preferably, collected in EDTA vials and films should be prepared within 2 hours for best result.

Staining :

For quicker diagnosis Fields stain is used whereas longer methods like Giemsa stain

provide best staining for successful indication of species

Examination of blood film :

Both thick and thin films should be prepared. Smears should be prepared soon after blood collection which ensures better adherence of the films to the slide and causes minimal distortion of parasites and red cells. A minimum of 100 fields should be examined before concluding the slide to be negative. Once negative, samples may be examined for at least three consecutive days where clinical suspicion of malaria persists.

Both thin and thick smear should be prepared. Thickness of the thick film should be uniform and correct which may be ascertained by the legibility of printed text seen through the slide. Thick film are nearly 10 times more sensitive for diagnosis of malaria as larger amount of blood are there in a given area as compared to thin film. As in thick film RBCs are lysed which alters the morphology of the parasites making it a good screening test for diagnosis of malaria. They are much better than thin film for detection of low levels of parasitemia and reappearance of circulating parasites during recrudescence or relapse. In thin film as fixed monolayer of RBC are available, morphological identification of the parasite to the species level and stage of parasite can be determined. So, to sum up thick film are used for malaria diagnosis and thin film for species identification. This is important in our country as treatment varies in two different species currently circulating.

Performance characteristic of microscopy:

(i) Skilled microscopist with proper infrastructure can pick up parasites as low as 5-10 parasite/?l of blood. However in actual practice most diagnostic laboratories generally achieve detection when parasite level is 100-500 parasite/ µl of blood.

(ii) Species identification can be done which is vital where treatment differs with different species. Stage of parasite can also be ascertained in the peripheral blood. In general prognosis worsens with predominance of more mature parasite stage. In general if more than 50% of the peripheral blood parasite are at the tiny ring stage (diameter of the nucleus <50% of the diameter of the rim of cytoplasm) the prognosis is relatively good. Presence of pigment containing asexual parasite of P. falciparum indicates bad prognosis if more than 20% of the parasite shows it. It indicates mature trophozoites or schizonts which has been released in the peripheral blood from parasites sequestered in the capillaries of internal organs.

(iii) Determination of the number of circulating parasite (parasite density) is exceedingly important to monitor the severity of malaria, evolution of the disease and assessing therapeutic efficacy. Parasite density can be calculated from both thick and thin film and expressed either as number of parasite present in per micro liter of blood or percentage of parasitised RBC. There in no uniform agreed definition of hyperparasitemia but parasite count of more than 250,000/?l of blood or more than 5% parasitized red blood cells carry poor prognosis. It is important that every positive blood film should have parasite density assessed exactly in the same way on post treatment specimens as on the initial specimen to judge therapeutic efficacy. Counting parasite in a limited area of thick film is acceptable when large number of parasite are encountered

whereas percentage infection of RBC in thin film are method of choice with low parasitemia.

(iv) The presence of malaria pigment in polymorphonuclear leukocyte are diagnostic of malaria. It is particularly useful in anemic children with severe malaria associated with low parasitemia. If more than 5% of polymorphonuclear leukocyte contain visible pigment prognosis worsens

Disadvantage of microscopy

(i) It is time consuming often requiring more than 60 minutes from blood collection to result.

(ii) It is labour intensive, needs significant technical skill and proper infrastructure which are often unavailable at peripheral health centers.

(iii) There is often long delay in providing the results of microscopy leading to treatment without the benefit of the results.

(iv) It cannot detect parasite sequestered deep in the vascular compartment which often requires repeated blood examination.

Rapid Diagnostic Tests (RDTs)

These tests were developed with the hope that it would offer accurate, cheap and rapid results as compared to traditional diagnosis. Tests are sensitive at parasite level of more than 100-500 parasite/?l of blood. However they have shown limitation in sensitivity in low parasite count, ability to differentiate between species and robustness under field condition in the tropics. It was expected they would give information on parasite densities, distinguish between viable parasite from parasite products not associated with viable parasite and prediction of treatment outcome.

They employ monoclonal antibodies targeted against the parasite antigens. The test kit contains specific antibody that is labeled with a visually detectable marker. If the antigen under investigation is present then antigen-antibody complex is formed. The labeled antigen-antibody complex will be immobilized at the pre deposited line of capture antibody and will be visually detectable. Whether the blood contains antigen or not, the control line will become visible as labeled antibody is captured by the pre-deposited line of antibody directed against it. The test time varies from 5 to 15 minutes.

Targeted antigens in currently available RDTs.

(1) Histidine-rich protein II (HRP-II) – This is a water soluble protein produced by asexual stage and young gamatocytes of P falciparum.

(2) Parasite lactate dehydrogenase (pLDH) – pLDH is an enzyme located in the glycolytic pathway of the malaria parasite produced by both sexual and asexual stages of the parasite. It is found in all the four species of malaria namely P falciparum, P vivax, P malarie and P ovale and is known as pan specific.

Distinct isomers of pLDH for each of the four plasmodium species infecting humans exist and they can be detected.

The next antigen is P falciparum specific, pLDH.

Some newer kits target P vivax specific pLDH for detection of vivax malaria.

(3) Certain new antigens like plasmodium aldolase – Plasmodium aldolase is also an enzyme of the glycolytic pathway produced by all four species has been recently developed.

Performance characteristics of RDTs :

It is an important consideration before choosing an RDT which should includes :

The test should be able to distinguish between malaria species at least falciparum and vivax. Falciparum and vivax malaria occurs in nearly equal number as single species infection in our country. Differentiation is essential as treatment of these two malaria differs hence accurate diagnosis is essential.

In our country where falciparum and vivax malaria parasite cocirculate, typically occurring as a single species infection an RDT which can detect both falciparum and vivax malaria and distinguish between them is warranted (13).There are some commercially available kits which detects falciparum specific LDH and panspecific LDH.So they can distinguish between falciparum from non falciparum malaria . Problem with these kits are two fold firstly they can not distinguish falciparum malaria from mixed infection ,secondly as vivax malaria is almost the only non falciparum malaria in our country so often they equate non falciparum malaria with vivax malaria

1. The sensitivity and specificity for detection of each of the species should be noted. World Health Organisation has recommended those are RDTs in accordance to certain criteria listed below :

(i) Panel detection score against P falciparum should be at least 75% had 200 parasite/?l.

(ii) Panel detection score against P vivax should be at least 75% had 200 parasite/ µl.

(iii) Falls positive rate should be less than 10%.

(iv) The invalid rate should be less than 5%.

2. RDTs using HRPII are generally more sensitive than RDTs detecting P falciparum specific PLDH. P vivax specific monoclonal antibodies have undergone limited evaluation. Unfortunately independent peer reviewed evaluation for most commercially available RDTs are not available. In general with high parasite density these tests are fairly sensitive but with low parasite load sensitivity decreases often yielding false negative results. False positive result may also develop when gametocytes are present but asexual stage parasites are eradicated by therapy.

One of the US FDA approved RTD was extensively investigated for its performance in tropical country. The trial showed for detection of any plasmodium species overall sensitivity of the test was 82%. The overall sensitivity for detection of P. falciparum was 95%, with a sensitivity of 99% for parasitemia in excess of 1000 parasite/?l, dropping to 89% for parasitemia of 100 to 500 parasite/?l of blood. The overall specificity of P falciparum was 94%.

There are some reports of occasional failure of RDTs to detect high parasite densities. Reports of failure to defect both P falciparum and P vivax has been demonstrated even when parasite densities exceeded 5000/?l of blood.

3.HRPII antigen persist at detectable levels for more than 28 days even after successful therapy.

Aldolase and PLDH rapidly fall to undetectable levels after initiation of effective therapy but these antigens are expressed in gametocytes which may appear after clinical infection is cleared. So none of the RDTs are useful for monitoring the response to treatment for which microcopy is the investigation of choice.

4. Test are usually simple without much training requirement, easy to interpret, does

not need electricity and results are available rapidly. The stability of the kit in high environmental temperature and humidity of tropics should be taken into account.

Disadvantages of RDTs

1. RDTs are not quantitative but qualitative gives a yes or no answer. Hence they are not suitable for prognostication and cannot be used for assessment of therapeutic efficacy of antimalarial drugs.
2. Persistance of antigenemia after parasite clearance preludes using the test to monitor response to therapy.
3. RDTs have decreased sensitivity at lower levels of parasitemia yielding false negative results in non immune patients with low levels of parasitemia.
4. False positive results when gametocytes are present but asexual stage parasite are eradicated by therapy may lead to unnecessary treatment.
5 RDTs cannot determine the stage of parasite ie, early ring form or late schizonts and thus does not help is prognostication.

Advantages of RDTs

1. Relatively easy with minimal training and results are available quickly.
2. They are able to detect falciparum infection even when the parasite are sequestered in the deep vascular compartment and thus undetectable by microscopic examination of a peripheral blood smear.
3. HRP II can be used in patients who have received incomplete treatment for malaria in whom microscopy can be negative.

Role of RDTs in the diagnosis of malaria in our country

In comparison to high transmission areas, malaria in our country occurs less frequently, in all age groups and almost always symptomatic. Drug resistance including multi drug resistance has started developing in our country so laboratory confirmation of malaria is an essential component of disease management. Expert microscopic diagnosis is available in central levels of health care system like metro cities but it is often unreliable or unavailable in areas with poor health facilities. So RDTs will be useful in following situations in our country :

(i) In far away communities with poor health care facilities where microscopic diagnosis is not available. Also in areas where laboratory service is inadequate, of an unacceptable standard or not available at odd hours.

(ii) In places where quality microscopy is available, RDTs and microscopy can run in parallel. RDTs will provide rapid or screening diagnosis whereas microscopy reserved for resolution of confusing cases, confirmation of negative result in RDTs with high clinical suspicion of malaria.

(iii) US FDA has approved RDT with a note that negative results by the RDT be confirmed by microscopy.

(iv) In some cases of severe and complicated malaria peripheral parasitemia may be negative due to sequestration but RDTs are expected to provide evidence of antigenemia.

(v) According to the new National drug policy of malaria (2008) that an fever cases clinically suspected of malaria should preferably be investigated for confirmation of malaria by microscopy or RDT so as to ensure full therapeutic dose with appropriate drug to all confirmed cases.

So in conclusion RDTSs permit on the spot

confirmation of malaria even at the peripheral health care system , by unskilled health worker with minimal training . Rational use of RDTs as a complement to microscopy might offer following benefits :

(i) Early treatment will reduce mortality and morbidity

(ii) In multi drug resistance areas expensive drugs and drug combination will be given to only to those who needs them

(iii) Avoidance of unnecessary treatment will reduce drug pressure and delay progress of drug resistance

Other methods of malaria diagnosis

Immunodiagnosis :

Detection of antibodies against parasite is not recommended is patients suspected to have malaria. They may be useful in epidemiological studies and has no place in routine diagnostic evaluation.

Fluorescent microscopy :

The test is based on the ability of fluorescent dyes to detect RNA and DNA of the parasite. As mature red blood cells have no nucleus anything which binds the dye is presumed to be the parasite. They are of no use in routine practice.

Quantitative buffy coat assay :

A modification of fluorescent microscopy in which blood is first centrifused to separate the parasite below the grannulocyte layer. Subsequently the parasites are detected by fluorescent microscopy and are suitable for screening a large number of samples. Again this method is not useful for routine practice.

Polymerase chain reaction (PCR) :

They are able to identify genetic material of the parasite. The test is highly sensitive which enables to detect even negligible amount of parasite DNA. It can also identify different species and mutations co-relating to resistance. However their use is limited to only research laboratories.

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Hypocalcemia in Neonates

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Introduction

Abnormalities of calcium and magnesium metabolismare not infrequent occurrences among infants admitted for neonatal care. Unfortunately this entity is seldom considered during management in peripheral health centres.

Definition

Neonatal hypocalcemia is defined as a total serum calcium concentration of <7 mg/dl or an ionized calcium concentration of <4 mg/dl. In VLBW infants, ionized calcium value of 0.8 to 1 mmol/L are common and the babies are usually asymptomatic. The ionized calcium value for term infants over the first 72 hours of life are 1.22-1.24.

Incidence

It affects both preterm and term infants. It occurs in upto 30% of infants with birth weight < 1500 gm. Late onset hypocalcemia is more common in developing countries where cow's milk or formulas with high phosphate concentration are used.

Pathophysiology

Ionized calcium is the biologically important form of calcium. The total calcium levels have repeatedly been shown not to be predictive of ionized calcium levels. Regulation of serum and extracellular fluid ionized calcium concentration within a narrow range is critical for blood coagulation, neuromuscular excitability, cell membrane integrity and function, cellular enzymatic and secretory activity. The principal calcitropic hormones are parathyroid hormone and 1,25[OH]2D.

Vitamin D is synthesized from pro vitamin D in the skin after exposure to sunlight. Vitamin is transported to liver, where it is converted to 25[OH]D (the major storage form of the hormone). 25[OH]D is transported to kidney, where it is converted to the biologically active hormone 1,25[OH]2D also known as calcitriol. Calcitriol increases intestinal calcium and phosphate absorption and mobilize calcium and phosphate from bone.

Etiology: During the third trimester of pregnancy, the human fetus receives at least 140mg/kg/day of elemental calcium via umbilical cord. Most of this calcium is readily incorporated into the newly forming bones. After delivery , this massive supply of calcium is suddenly stopped and therefore, calcium must be supplemented enterally.

(a) Prematurity: Preterm infants are capable of mounting PTH response to hypocalcemia, but target organ responsiveness to PTH may be diminished. Calcium levels (both total and ionized) usually return to normal within 48-72 hours regardless of whether supplemental calcium is given.

(b) Infants of diabetic mother have a 25-50% incidence of hypocalcemia if maternal control is poor.

(c) Severe neonatal birth asphyxia is frequently associated with hypocalcemia and hyperphosphatemia. Use of alkali (sodium bicarbonate) during resuscitation result in hypocalcemia and relative hyperphosphatemia secondary to increased circulating endogenous phosphorus following post asphyxia renal impairment. The combination of bicarbonate infusions and hypocarbia secondary to hyperventilation is associated with profound hypocalcemia.

(d) IUGR: Sporadic hypocalcemia occurs.

(e) Nutritional deprivation: Infants unable to take enteral feeds by 3 days of age need calcium supplementation. Because hypocalcemia is associated with hypomagnesemia, both elements require supplementation to prevent secondary suppression of parathormone recurrence of hypocalcemia.

(f) Congenital abnormalities: Di George sequence with absence of parathyroid glands often present with hypocalcemia.

(g) Drugs: Frusemide, citrated blood transfusions, inadequate prenatal vitamin D supplementation of mother or the infant during the first 6 months of life.

Clinical presentation:

(a) Early onset hypocalcemia (First week of life): Apnea, stridor, irritability, jitteriness, tremor, hyperreflexia, clonus, tetany or seizures and arrhythmia secondary to prolonged Q-T interval.

(b) Late onset hypocalcemia (After first week of life): Lethargy, Apnea, feeding intolerance, abdominal distension, bone demineralization, increased alkaline phosphatase and skeletal fractures.

(c) Paradoxically, neonatal hypocalcemia may be asymptomatic and only a high index of suspicion on the basis of risk factors will lead to a correct diagnosis.

Diagnosis:

(a) Laboratory studies:

(i) Total and ionized calcium levels – Serum total calcium of <1.75 mmol/L is usually diagnostic and further confirmed by ionized calcium levels of <1 mmol/L.

(ii) Serum magnesium levels of <1.5 mg/dl is indicative of hypocalcemia because they often follow one another.

(iii) Elevated alkaline phosphatase levels can be seen in chronic hypocalcemia.

(iv) Urinary calcium – to - creatinine ratio (spot urine specimen) >0.21-0.25 and 24 hour urinary calcium level >4 mg/kg/day are indicative of hypercalcemia.

(b) Radiologic studies done for bone demineralization, metaphysical lucencies, rib and long bone fractures may be helpful for the diagnosis of late onset hypocalcemia. Absence of thymic shadow on X Ray chest suggest Di George sequence.

(c) ECG studies: Identify arrhythmia due to QT-interval changes.

Treatment:

Therapy with calcium is usually adequate for most cases. In some cases concurrent therapy with magnesium is indicated. Rapid IV infusion of calcium should be reserved for treatment of hypocalcemic crisis because it can lead to bradycardia or other dysrhythmias (If heart rate drop by >20 beats/minute, the infusion should be stopped for some time). Infusion by means of umbilical vein may result in hepatic necrosis if the catheter is lodged in hepatic vein, rapid infusion by means of umbilical artery can cause arterial spasmswhich may lead to intestinal necrosis.

IV calcium solutions are incompatible with sodium bicarbonate since calcium carbonate will precipitate.

Calcium preparations: Calcium gluconate 10% solution is preferred for intravenous use.

i. If ionized calcium level drops to 1 mmol/ L or less (>1500 gm) or 0.8 mmol/L or less(<1500 gm), a continuous IV calcium infusion may be commenced.

ii. To prevent the onset of hypocalcemia in newborns with cardiovascular compromise due to severe RDS, perinatal asphyxia, septic shock and PPHN, a continuous calcium infusion is preferred to maintain an ionized calcium of 1-1.4 mmol/ L (<1500 gm) or 1.2-1.5 mmol/L (>1500 gm).

iii. Emergency calcium therapy (for active seizures and profound cardiac failure associated with severe hypocalcemia) consists of 100-200 mg/kg of 10% calcium gluconate by IV infusion over 10-15 minutes. Monitor heart rate nad rhythm and the infusion site throughout infusion.

iv. Maintenance treatment : Infants with limited enteral intake need early and continuous treatment with an IV dose of 45mg/kg/day of elemental calcium with a calcium to phosphate ratio ranging from 1.3:1-2:1.

Human Papilloma Virus (HPV) Vaccine and Cancer Prevention

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Introduction

It is now a well established fact that 100% of cervical cancers are caused by the Human Papilloma Virus (HPV). There is growing evidence of HPV being a relevant factor in other anogenital cancers (anus, vulva, vagina and penis) and head and neck cancers. HPV is also responsible for other diseases such as recurrent juvenile respiratory papillomatosis and genital warts.

Some hard hitting statistics

HPV is now consider to be the most common sexually transmitted disease and about 80% of sexually active people will become infected with this virus sometime in their lives. HPV 16, 18, 9, 11, 31, 33, 35, 45, 52 & 58 are known to be oncogenic. Data from 2012, shows that worldwide about 5,27,624 new cases of cervical cancer occur annually and it is the second most cancer among female from 15-45 years of age. Globally about 728 women die every day amounting to 2 deaths per hour of cervical cancer. In India too, the situation is grim with 1,22,844 new cases occurring every year and again being the second most common cancer in women of 15-45 years of age. In India about 185 women die daily amounting to 1 death every 8 minutes of cervical cancer. Another interesting data from India shows that cervical cancer occurs equally in the urban and rural women. So there is a huge burden of cervical cancer in India. Coupled with this, is the many fold rise of genital warts in India over the last decade. The incidence of genital warts was strongly associated with the incidence of anal, vulvar, vaginal, cervical and head & neck cancers, with HPV being the common link in this spectrum of diseases.

HPV 16 has been most commonly detected from oropharyngeal cancer lesions. Recent data suggests that more oropharyngeal cancers are attributed to HPV 16 than cervical cancers. The National Cancer Institute (NCI) Surveillance Epidemiology & End Results (SEER) cancer registry shows that the incidence of HPV positive oropharyngeal cancers are more common in men than women and will keep on rising to a large number till 2030.

Cancer prevention:

There are currently two ways one can prevent cancer:

Screening :

Though screening programs are available for

cancer prevention esp. for cervical cancer, competing health care priorities, insufficient resources, weak health systems and a huge population especially in developing countries like India has resulted in a not so effective screening program. The coverage of cervical cancer screening is as low as 2.6% in India. No such screening exists for head and neck & other genital cancers.

Vaccination :

Vaccination on the other hand has great potential for an effective cancer prevention program.In India it has been seen from various studies that 50% of the women harbour HPV 16 & 18 and 83% of the cervical cancers are caused by HPV 16 & 18. Similarly,> 90% of the general warts are caused by HPV 6 & 11. Where screening can prevent up to 29% of cervical cancers, a combination of vaccination and screening increases the prevention rates to > 61%. The NCI has projected that if the HPV vaccine is given to nearly all children in the USA, it would reduce the incidence as follows:

(a) Oral cancer in both sexes by 70%
(b) Anal cancer in both the sexes by 95%
(c) Cervical cancer by 70%
(d) Vaginal cancer by 65%
(e) Vulvar cancer by 50%
(f) Penile cancer by 35%

Therefore, HPV vaccines offer a more effective way in cancer prevention than only screening methods.

Vaccines available

There are two types of HPV vaccines currently available in India - quadrivalent HPV (qHPV) containing serotypes 6, 11, 16 & 18 (Gardasil by Merck) and the bivalent HPV vaccine (bHPV) containing only serotype 16 & 18 (Cervarix by GSK). The qHPV protects against cervical, oropharyngeal as well as vulvar, vaginal and genital cancers as the serotype 6 & 11 would also protect against the development of genital warts. The bHPV protects only against cervical and oropharyngeal cancers. There is also a 9 valent HPV vaccine covering serotypes 6,11,16,18,31,33,45,52 & 58 which has replaced the previous vaccines in the USA. It is expected that this vaccine should be available in India by 2019 after field studies of its efficacy in India are over.

HPV Vaccine recommendations in India

Currently the Indian Academy of Pediatrics (IAP) and the Federation of Obstetric & Gynacelogical Society of India (FOGSI) recommends the HPV vaccines to be administered in girls as early as possible and preferably before the first sexual exposure. Both the bodies advocate for early HPV vaccination in adolescents.

Current recommended vaccine schedule

For girls 9-14 years, 2 doses of either the qHPV/bHPV vaccine 6 months apart

For girls 15 years and above or those who are immunocompromised, 3 doses are administered at 0, 2 and 6 months.

The Centre for Disease Control (CDC), USA recommends the vaccine in both adolescent boys and girls. IAP says that one can use in boys too but currently the vaccine is not licensed in India for boys.

Catch up vaccination: Any women between 13 - 45 years of age can take the vaccine if not taken earlier.

What about those who have taken a single dose and missed the subsequent doses?

For this group one can administer the remaining dose/doses within a maximum of 15 months.

What's the real world efficacy of the vaccine?

The prevalence of HPV related disease has drastically reduced in countries who have taken up universal HPV vaccination in adolescents and young women. Studies from USA show a 64% reduction of HPV infection in females 14-19 years and 34% reduction in women 20-24 years. In Australia, the incidence of genital warts and precancerous lesions has decreased by 72% in women >21-30 years and 92% in women < 21 years. Similarly studies in New Zealand has shown a reduction of genital warts in both males and females <20 years by 63%. In Sweden it was demonstrated that the vaccine effectiveness was > 93% in preventing genital warts if used < 14 years of age. There was significant reduction in the incidence of cervical dysplasia in women who had received the vaccination in USA and Canada. In Australia, the overall prevalence of qHPV serotypes in women reduced significantly in the population in the post-vaccination period irrespective of their vaccine status indicating a herd protectiveness of the qHPV vaccine.

Current vaccination programs in the world:

Many countries in the world like USA, Australia, New Zealand, Sweden and other European countries have now adopted universal vaccination of adolescents with the qHPV vaccine. In the USA, the 9-valent HPV vaccine has replaced the qHPV and bHPV vaccine. In India too some of the states have adopted the HPV vaccine for adolescent girls. The government of Sikkim has recently introduced universal HPV vaccination in the adolescent girls of the state.

What's the W.H.O. position on HPV vaccines?

The W.H.O. position paper on HPV vaccines states that all the three currently available HPV vaccines (bHPV, qHPV & 9 valent vaccines) offer good comparative immunogenicity, efficacy and effectiveness for prevention of cervical cancer with excellent safety profiles. Globally by 31 Mar 2017, seventy one countries have introduced HPV vaccines in their national program for girls, and in 11 countries for boys also. It recommends that HPV vaccine must be introduced in all national immunisation programs. It is to be given at 9-14 years of age before the child becomes sexually active in a two dose schedule in a six month interval between doses. A maximum interval of 12-15 months is allowed for completion of the second dose.

The choice of the HPV vaccine (bHPV or qHPV) is based on assessment of locally prevalent data, prevalence of HPV associated health problems like carcinoma cervix, anogenital cancers and warts. The W.H.O. therefore advocates that HPV vaccines should be introduced as a part of a coordinated and comprehensive strategy to prevent cervical cancer and other diseases caused by HPV.

What Radiation and Medical Oncologists and Oncosurgeons can do?

There is a great role that can be played by Oncologists and Oncosurgeons in the prevention of HPV infection. Children and spouses of survivors of HPV related cancers (cervical, anogenital and head & neck cancers) can be advised to be vaccinated against HPV. In some cancer centres in Canada, the HPV vaccine is routinely given to the spouses of the patients with cervical

and anogenital cancers to clear the HPV infection in these populations. For other non HPV related cancers, it presents a unique opportunity for intervention by recommending to the patient as well as the relatives to take the HPV vaccine and prevent another dreadful cancer.

How I do it?

Along with routine vaccination for children and adolescents, the immunization facilities managed by the Department of Paediatric Disciplines at my workplace routinely offers vaccination to adults against a host of diseases including HPV.

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Situs Inversus in A Child With Urinary Tract Infection

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Background: Situs inversus an incidental finding in a case of urinary tract Infection.

Case characteristics : A 10 year old presented with fever associated with chills and rigors and pain abdomen for 2 days. E.Colipositive UTI. Intervention: Clinically improved with antibiotics.

Message: Situs inversus is not a predisposing factor for urinary tract infection and is an incidental finding usually.

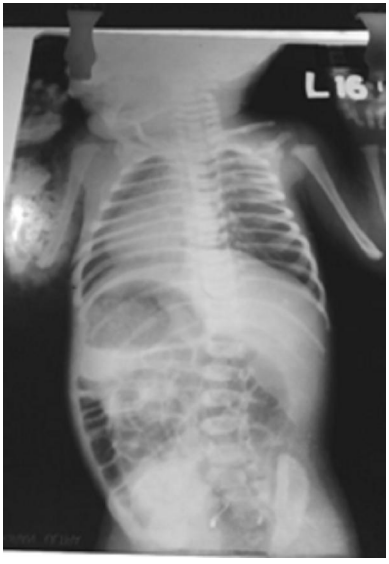
Keywords: Situs inversus , incidental finding , urinary tract infection.

"situsinversusviscerum"is a latin word and its short form is "Situs Inversus" which is describedas the inverted position of chest and abdominal organs.Situs inversus are of two types– Situs inversustotalis (Situs inversus with dextrocardia) or Situs inversusincompletus (Situs inversus with levocardia).

Case Report

The patient a 10year old female who was hospitalised in Pediatrics Department with complaints of fever associated with chills and rigors and abdominal pain of 2 days duration.

On examination, child was toxic and febrile. Per abdomen examination was normal.CVS:Heart sounds were heard bilaterally but more on the right side. On investigating, CBC revealed Neutrophilic leucocytosis and CRP was strongly positive. Urine routine and microscopy revealed leucocyte esterase+,WBC:1-2cells/hpf.Urine culture showed E Coli >105.Inj Ciprofloxacin



and Inj Gentamycin was started based on sensitivity report.USG Abdomen showed Situs Inversus as Liver,Gall bladder,IVC were on the left side and Spleen and aorta on the right side.Chest Xray was done which showed apex of heart and fundal shadow on the right side.The child recovered with the treatment and was diagnosed as having Urinary Tract Infection with Situs InversusTotalis.

Discussion

Situs inversus is a congenital condition, mostly autosomal recessive although can be X linked also[1]. 1 per 10000 individuals is the incidence[2]. Medical symptoms or complications are not seen in most of the people;only 5-10% have congenital heart disease out of which most common is Transposition of great vessels.Primary ciliary dyskinesia - an underlying condition in case of situs inversus is seen in 25% of the individuals[3].

Situs inversus is associated with a triad known as Kartagener syndrome which includes situs inversus,chronic sinusitis and bronchiectasis.

Radiological investigations including Chest Xray,ultrasonography,CT and MRIare usually used to diagnose Situs inversus[4]. Chest xray shows cardiac apex pointing towards right,aortic arch on right side and stomach bubble located on right side[1]. In asymptomatic individuals usually no treatment is required.

The conditions associated with situs inversusdecides the prognosis;usually asymptomatic with isolated situs inversus.

Conclusion

Situs inversus is an incidental finding in most cases and is usually asymptomatic.

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Zika Virus (ZIKV) Infection- An Emerging Threat To Fetal Life

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Introduction

Zika virus (ZIKV) is an emerging pathogen belonging to Flavivirus family. This viral infection is transmitted in non human primates and humans by Aedes mosquito, which also transmits dengue, yellow fever, chikungunya viruses. It was first documented in Uganda in 1947 in monkeys. But first human infection was identified in 1952, again in Uganda and United Republic of Tanzania. Till 2007 sporadic infections were reported from Africa and Asia, when the first documented outbreak was reported in Yap state, Federated States of Micronesia and subsequently in South- East Asia and Pacific islands. In 2015, Brazil experienced the largest outbreak, with an estimated 440, 000 to 1, 300,000 persons being infected with Zika disease and 1000 babies born with serious birth defects. Thereafter, evidence of association between Zika infection and increased risk of neonatal malformations and neurological disorders propelled WHO to declare the ZIKA outbreak as a Public Health Emergency of International Concern in February, 2016.

At present, more than 80 countries including India are harboring ZIKV. In India, for the first time three confirmed cases were reported in May 2017 in Ahmedabad, Gujarat. Since then a handful of cases were reported from Gujarat and Tamilnadu. But recently, more than 135 people have been found to be affected, including 40 pregnant women in Jaipur, Rajasthan which is a popular tourist destination, thus threatening unpredictable spread to any place at any time.

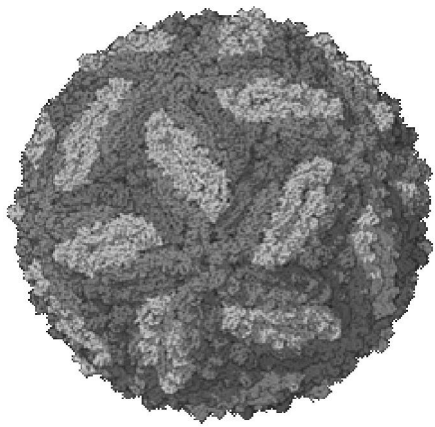

Fig 1: Capsid model of Zika virus

Transmission

The primary mode of transmission of ZIKV is by day- time bite of Aedes mosquito. Other modes are sexual transmission, intra- uterine, intra- partum and through blood transfusion.

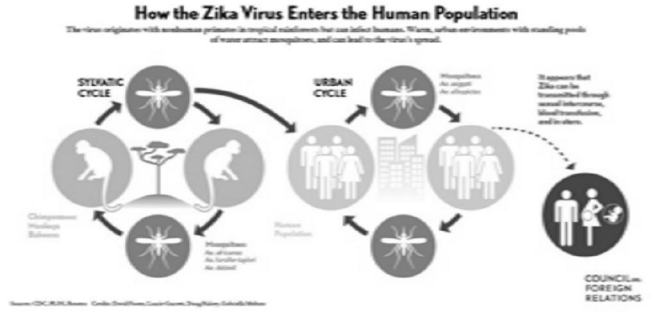

Fig 2: ZIKV transmission cycle

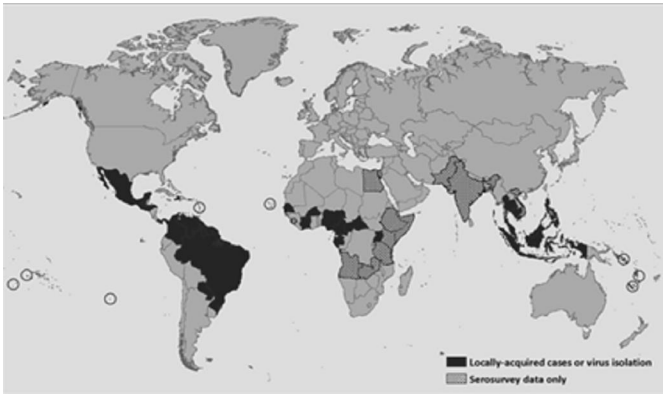

Fig 3: Countries with present/ past zika virus transmission (till January 2016)

Clinical manifestations

ZIKV infection mostly remains asymptomatic. Characteristic clinical features are acute onset of fever with maculo- papular rash, arthralgia or arthritis, conjunctivitis. Other symptoms include myalgia, headache, retro- orbital pain, edema and vomiting. Fortunately, the case fatality is low. More severe presentations include thrombocytopenia, GBS, meningo-encephalitis and acute myelitis. Congential ZIKV infection is of special concern in child health. Infection in pregnancy leads to birth defects, especially microcephaly. Other brain and ocular abnormalities have also been reported.

Classical features of congenital Zika virus infection

Zika virus is a neurotropic virus. It particularly targets neural progenitor cells. Infection during pregnancy leads to placental infection and injury, followed by transmission of the virus to the fetal brain. Infection of brain kills neuronal progenitor cells and disrupts neuronal proliferation, migration, and differentiation, which slows brain growth and reduces viability of neural cells. The spectrum of neuropathological features includes ventriculomegaly, lissencephaly, and cerebellar hypoplasia.

Zika virus is also associated with a higher rate

of fetal loss throughout pregnancy, including stillbirths, particularly if the infection occurs during the first trimester. Placental insufficiency is the proposed mechanism behind adverse fetal outcomes.

Five classical features have been identified in fetuses infected with ZIKV in- utero:

1. Severe microcephaly with partially collapsed skull.
2. Thin cerebral cortices with sub-cortical calcifications.
3. Macular scarring and focal pigmentary retinal mottling.
4. Congential contractures.
5. Marked early hypertonia with extra-pyramidal involvement.

But full spectrum of fetal and infant outcomes as a result of symptomatic and asymptomatic infections in pregnant women is yet to be determined.

Diagnosis

WHO Case definitions of Zika virus infection:

Suspected case – A person presenting with rash and/ or fever and at least one of the following signs: Arthralgia, arthritis or conjunctivitis.

Probable case – A suspected case with presence of IgM antibody against Zika virus and an epidemiological link.

Confirmed case – Presence of Zika virus RNA or antigen in serum or other (samples- saliva, tissue etc) or IgM antibody against Zika virus and PRNT90 for Zika virus with titers >20 and Zika PRNT90 titers ratio > 4 compared to other flavivirus are considered as confirmed case.

Management

No specific treatment against ZIKV is available at present. Supportive therapy is the mainstay of management.

Prevention

1. Personal protective strategies: Wearing repellants, staying in screened or air conditioned buildings and avoiding outdoor activities in times during which probability of bites by Aedes mosquito is highest (day time).
2. Measures to decrease the abundance of vector mosquitoes.

Vaccine

The goal of ZIKV vaccine is to elicit protective antibodies against the virus to prevent infection and severe disease, particularly congenital defects in the newborn. Though a number of vaccines like DNA vaccine, mRNA vaccine, Purified inactivated vaccine (ZPIV), Live attenuated vaccine and viral vector based vaccines are currently under clinical trial, none of them have been approved for clinical use as of July 2018.

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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.