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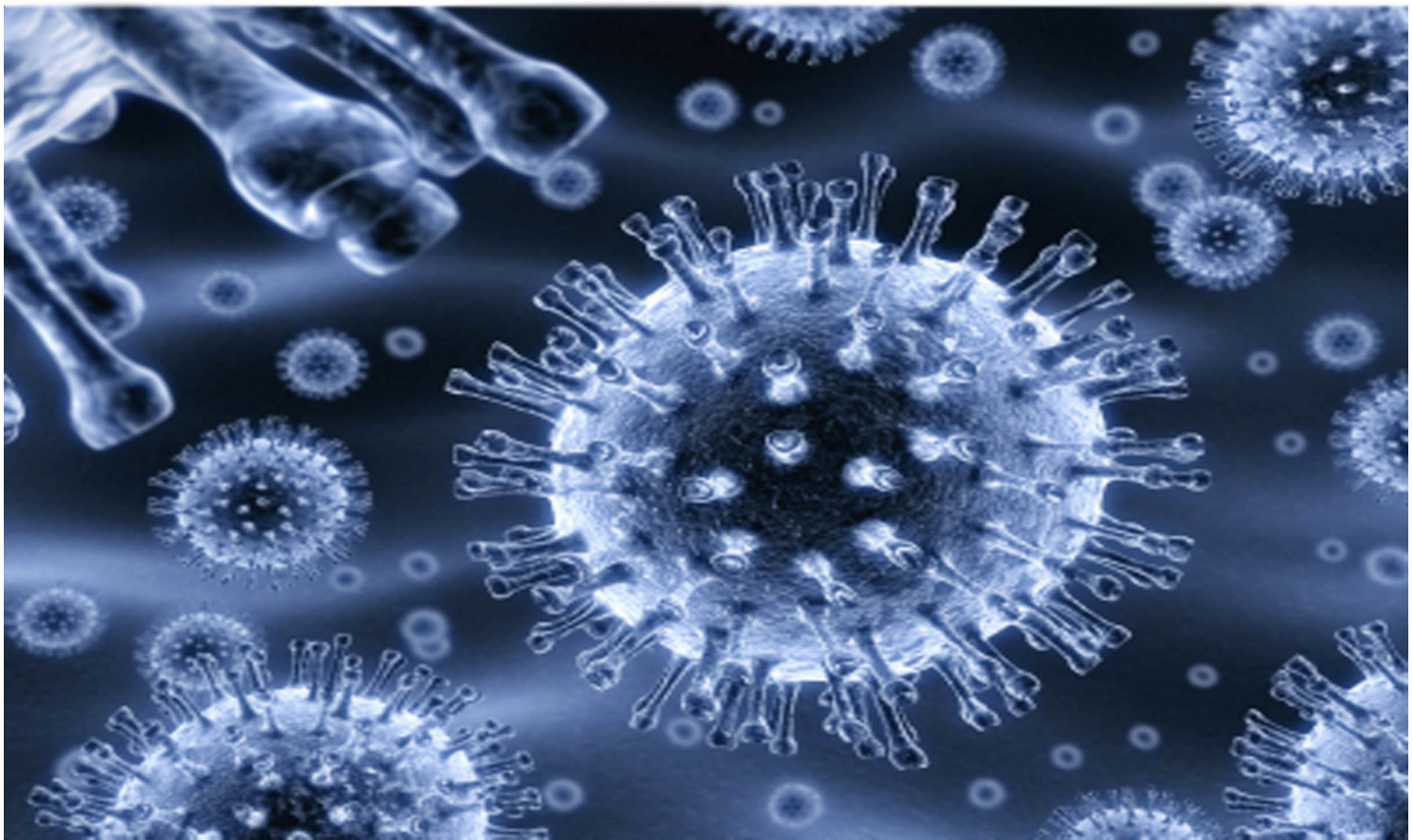
PEDINFECON 2017



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"Modern Medicine does'nt tolerate infectious diseases"

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Civilizations have created different medical systems.

Even though we all are teachers and students of Modern Medicine, most of us have not understood the full picture of Modern Medicine, but only a truncated version. To understand Modern Medicine fully, we must explore its cultural origins.

We have our own Traditional Medicine, a product of Indian culture, with its theories, ideologies and principles. It evolved and blossomed 3000 years ago. The scientific enlightenment of the European culture was only about 300 years ago. As European Medicine was evolving from the 18th century onwards, it was heavily influenced by scientific theories and practices. Therefore it is easy to understand that a pre-science Indian Traditional Medicine and a science-influenced European Medicine have huge methodological differences.

Renaissance, religion-based ideologies, French revolution, industrial revolution, emerging political philosophies and democratization of governments – all contemporaneous phenomena, had tremendous impact on European Medicine and its positioning in society. These details are not taught in medical colleges.

European Medicine was transplanted in India by the British, during colonial rule. No one calls it European any more since it has become universal. Modern Medicine is a better name.

What is common and different, between Traditional and Modern Medicines?

There are a few points of commonality but several points of contrast. In both systems there are theories on the causation of diseases as well as remedial therapeutics. Both address the basic human need to cure individuals when sick. As pre-science system, Traditional Medicine uses herbs, minerals and animal products in its pharmacopeia, while Modern Medicine, as we all know, applies science and statistics to create laboratory-made and evidence-supported pharmacopeia. Both approaches have their successes and failures, but in unequal measure.

It is amazing to realize that surgery for cure of illness was already practiced in Traditional Medicine. Without science and continued experimentation, anesthesia and antiseptics could

not evolve, whereas Modern Medicine developed both and mastered surgical therapies for cure or rehabilitation.

In summary, personal medicine is a common approach of both systems to meet the needs of the individual. That need is to restore health after falling sick. We call it Curative Medicine, Personal Medicine or healthcare. Individual-level preventive interventions are also part of both systems; we call this preventive healthcare. Broadly speaking the similarities are limited to these ideas of healthcare and how the physician treats individual patients.

Many among us may consider that Medicine and healthcare are synonymous; well, they are synonymous in Traditional Medicine, but not in Modern Medicine. Modern Medicine comprises of personal Medicine and Public Health. In medical college we were taught that Public Health and Social Medicine were synonymous at one time.

Thus, the major difference between the two systems is in the way Medicine is applied in societal organization instead of in individual healthcare. Without understanding that difference, we will not understand why we were taught that Modern Medicine was qualified as 'social science' and 'politics' was said to be 'Medicine on a large scale'.

Modern Medicine is much more than therapeutics and surgery for individual healthcare. Its primary goal is to keep people healthy without diseases, particularly diseases against which preventive interventions are available. Modern Medicine excelled in designing and applying preventive interventions at community level, and continues to do so even now. As time progresses, more diseases are converted to the preventable list.

After independence we have been choosy as to what to take in and what to discard from the transplanted Modern Medicine. We chose to take in that part that fits our cultural concepts and beliefs regarding the role of Medicine in society – which is healthcare, using therapeutics and surgery.

What is discordant with our cultural concepts and beliefs about the purpose of Medicine, namely Public Health, which is separate from therapeutics and surgery, was discarded. That discarded part of Modern Medicine is what is intolerant of infectious diseases. After independence, Indian political leadership abolished the Public Health infrastructure established by the British, under the justification that Public Health was being integrated with Curative Medicine.

Microbial causation of diseases

Traditional Medicine teaches that illnesses are mostly of intrinsic origin. The belief was that the person broke some discipline through acts of commission or omission and consequently became sick. Beginning in 19th century, Modern Medicine grew with the concept of microbial causation of diseases. The main purpose of Medicine was human mastery over microbes.

The knowledge that by blocking microbial transmission people can be protected from diseases, was a powerful game-changer. That insight further advanced Public Health that had already been started in many countries. The simple process of sand filtration of drinking water was

enough to see that no one got bacillary dysentery, cholera, typhoid fever, even infectious hepatitis that we call today hepatitis A. Add chlorination and we can have double protection.

Traditional Medicine knew much about these diseases and named them *athisaram, vishoochika, sannipathajwaram* and *manja-kamala*, respectively.

My thesis is that Traditional Medicine had no choice but to tolerate these diseases as the 'given', without understanding their aetiology or risk factors. But Modern Medicine blames the societal organization for causing them by supplying unsafe water. Modern Medicine cannot tolerate them because the societal organization, in other words the State, is at fault. Both politics and Medicine are for citizens' welfare; hence Public Health is an integral part of Governance. Hence infectious diseases are no longer accepted as the 'given', but are the 'intolerable'.

The public does not know that these diseases signal the neglect of Public Health, which is the moral duty of the State. It is also the State's legal duty as the State has agreed that health is a Basic Human Right. Consciously tolerating people's suffering and death due to readily preventable infectious diseases, is unethical.

Public Health and Personal Medicine: 2 sides of the coin that is Modern Medicine.

Beginning in 17th century, Modern Medicine discovered both social determinants of illnesses and social consequences of illnesses. Moreover, many microbial diseases are communicable, passing on from human to human. That insight translated into the essentiality of Public Health as a State instrument for keeping people disease-free and healthy.

Pathogens also came from animals and insects. Thus environmental determinants of diseases were identified and successfully targeted for prevention and control through Public Health – the major tools being sanitation, hygiene and intelligent pest control. Surveillance became part of Public Health so that all communicable diseases could be kept under watch and appropriate action taken to prevent their spread.

In summary, Public Health became an important the arm of the State to mitigate social and environmental determinants of diseases. Public Health is the quintessence of Modern Medicine as far as communicable diseases are concerned. This is why Medicine was perceived as a social science.

The foundation science of Public Health is epidemiology. One of its major functions is to receive reports on every disease case within a list of government-notified infectious diseases. The State notifies which diseases must be reported. Every medical practitioner, irrespective of who employs him/her – State, private sector, or self-employed – has the moral and legal duty to report every case of notified diseases.

Case-based and real-time reporting by every medical practitioner, local level trained staff receiving, collating and analyzing the reports continuously, on a daily basis, to discern patterns, instituting actions to prevent and control diseases, and providing feed-back to the medical professionals on all current disease patters – these are the functions of Public Health and

epidemiology, together called 'Public Health Surveillance'.

Challenged with this definition, we do not have Public Health Surveillance in India. But we do have many modes of information collection – each one may be called some kind of surveillance, but none qualifies as Public Health Surveillance.

Public Health Surveillance was once established in Kerala as 'district level disease surveillance', way back in 1998, but was discontinued in 2003. We teach epidemiology in class rooms and apply it for research studies, but the routine practice of epidemiology through Public Health Surveillance, is missing.

To adopt Modern Medicine in its true nature, and not merely therapeutics and surgeries, we need, collectively, an attitudinal shift from traditional thinking -- political, sociologic, economic and medical.

Public Health does not tolerate preventable diseases. Many infectious diseases are readily preventable. Public Health functions on the principles of social justice and equity, and cannot watch infectious diseases among citizens as mere observers.

Universal Immunisation Programme

We must ask if Universal Immunisation Programme is Public Health. World Health Organization prescribed 6 vaccines and we adopted them without question, since we had lots TB, diphtheria, pertussis, tetanus, polio and measles. Later on WHO recommended the inclusion of hepatitis B vaccine, *Haemophilus influenzae* b vaccine, and India adopted them both. More recently WHO has recommended the inclusion of Inactivated polio vaccine (IPV), Rotavirus vaccine and Pneumococcal conjugate vaccine in UIP and India has complied.

However, UIP has no capacity to conduct Public Health Surveillance. Therefore we do not know the quantitative impact of each of these vaccines, except what have been counted, namely polio and neonatal tetanus. Of course all other diseases have declined; but Public Health demands denominator-based incidence data.

The first step in disease control is Public Health Surveillance. Immunisation is the intervention for disease control. So we have invested inputs in without monitoring the actual degree of disease reduction over time. In 1998 Kerala actually had established Public Health Surveillance for vaccine-preventable diseases, in order to convert the vaccine delivery platform of UIP into a disease control modality. Its purview was enlarged to include all outbreak-prone infectious diseases. Unfortunately it was discontinued, without understanding its real value and importance.

The only case-based surveillance we now have is for acute flaccid paralysis, AFP. It was only because of counting every case of AFP with wild poliovirus infection, that India could be certified as wild polio eliminated. On the other hand we do not know the burdens of any other vaccine-preventable diseases or the exact impact of introducing vaccines, but we immunize almost as a ritual, without being able to calculate the social and economic benefits of UIP.

Other vertical Programmes against TB, malaria, kala azar, filariasis, leprosy are similarly intended

for disease control, but their disease burdens are not monitored for calculating their impact.

AIDS control programme stands out, because it has annual denominator-based infection prevalence monitoring and we therefore know its downward trend. The other 5 vertical programmes were foreign-designed, while AIDS control alone was indigenously designed. The lesson here is that we must autonomously decide what we need and do that, instead of simply following WHO instructions.

Infectious diseases in Kerala

All ubiquitous respiratory infections – viral and bacterial, and enteroviruses except polio, are all here. That is no surprise. We also have measles, mumps, rubella and varicella, all of them vaccine-preventable. Yet, we tolerate them without understanding that Modern Medicine does not tolerate them.

Diseases due to microbes from human or animal faeces contaminating water or food -- cholera, amebic and Shigella dysenteries, typhoid fever, paratyphoid A, hepatitis A, entero-hemorrhagic and entero-toxigenic E coli diseases including haemolytic uremic syndrome, are still with us, I dare say, anachronistically. They had been well controlled in late 19th and early 20th centuries in countries that have Public Health.

Although we know all about prevention of rabies, we have no Public Health infrastructure to control it; instead we tolerate its prevalence and spend a huge amount of funds for post-exposure prophylaxis.

Let me pick out typhoid fever. As an exclusively human pathogen it can be eliminated, using already available tools, in Kerala, if only such a target is proposed and promoted. If you all vote for it, let the Government know, and I promise full support and technical guidance. If we add hepatitis A also to this effort, we can control it, but elimination may not be as easy as for typhoid fever.

Which vector-transmitted disease do we not have? We have malaria, Filariasis, Japanese encephalitis, Kyasanur Forest disease, dengue, chikungunya, West Nile, kala azar, cutaneous Leishmaniasis, scrub typhus, spotted fever.

In 2001 a District level Mosquito Monitoring and Management project was approved by the Health Ministry, one programme against all mosquito-borne diseases. Leptospirosis is another disease that is very common but not targeted for control. In 2001Kerala Leptospirosis Elimination Programme was also designed approved by the Health Ministry. In the absence of a Public Health infrastructure neither programme could be implemented. I had tried my best to get the support of medical colleagues, but I did not succeed then. I am trying again now.

In 2011, a Kerala Health Protection Agency was approved by the Government and given 11 crores of Rupees to establish it. It is still not established, because conceptually or culturally, collectively we have not understood Modern Medicine in its totality.

When it came to polio elimination and JE diagnosis, Kerala stood out as a State without any virological expertise. To fill that gap a Virology-cum-infectious diseases institute was established

in 1999, but government support was withdrawn in 2003. Today much of State's needs are fulfilled by a centre in Manipal in the neighboring State of Karnataka. The Rajiv Gandhi Centre for Biotechnology, a Central Government establishment is helping with viral diagnostics in medical colleges, but that is not the ideal solution.

I urge all of you to demand a functional Public Health infrastructure in Kerala so that all preventable infectious diseases could be prevented.

My recommendation for a start is typhoid elimination and hepatitis A control as the first step, followed by our own indigenously designed plans for establishing a Health Protection Agency in Kerala.

I wish you success.

Rational Investigations in Childhood Infections

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It is common to hear of rational antibiotics in this day and age. Antibiotic stewardship and Antimicrobial resistance are at the forefront of academic discussions and concerns. However, it was some years ago that the JSS hospital at Ganyari near Bilaspur in Chattisgarh invited many of us from institutes across India to discuss Rational Investigations. The Jan Swasthya Sahyog (JSS) is a voluntary, non-profit, registered society of health professionals running a low-cost, effective, health program providing both preventive and curative services for the past 15 years to people from the tribal and rural areas of Bilaspur, Chhattisgarh through a community health program and a rural health centre, which includes a hospital. This opened our eyes to the impact in health care diagnostics and the fact that may be India cannot afford this "advancement". Unpublished observations clearly demonstrate that at least 1/3rd of In-Patient costs and even 2/3rd of Out-Patient costs are due to investigations especially in tertiary level hospitals. Costs mount on e one advances to intensive care and as duration of hospital stay increases associated with the many hospital acquired infections that occur.

I'm sure there are many definitions of the term rational, but I simply think that the term indicates, when describing investigations, as an investigation whose result will modify the treatment and management of the patient. If the result has absolutely no impact and changes nothing then it is obviously irrational. The term rational is purely defined as such from the point of view of the patient's wellbeing especially when it is the patient that pays for the investigation. The costs may go beyond financial and commonly include time, pain and discomfort. An example in a child with suspect Dengue would be ordering a NS1 test result that hardly ever changes the management of the febrile child admitted with or without warning signs. However, one may prioritise it if the child is admitted with suspect Severe Dengue in Multiorgan failure simply because a mortality would require documentation for notification. In most cases, the parents of a NS1 positive child run from pillar to post worried out of their wits getting test after test done when Dengue is widespread. It is often that the NS1 test becomes a

social indication to admit due to parental pressures even when the child is well and periodic clinical and basic lab monitoring is a possibility.

Investigations are rapidly becoming the reason why patients visit doctors and doctors certainly encourage this changing scenarios for many reasons. Cut practice from laboratories is most unprofessional and unethical and is one primary reason. However, there are tremendous changes in the quality of medical education that are occurring. The new basic MBBS is now the basic MD where MBBS doctors are no longer confident and competent to practice medicine. One factor in incompetence stems from the fact that clinical reasoning and critical analysis is no longer prime areas of learning and teaching and assessments! Physicians are moving away from the William Osler's art of history taking and meticulous physical examination to reach logical conclusions, a clinical diagnosis or at least narrowed down differentials. Physicians have demonstrate incompetence by ordering investigations from A to Z or should we say from Hb TC DC to MRIs so as to search for some clue originally found in the history and physical examination. The over dependence upon investigations is a crucial consequence of the inadequacies of medical education and stems from clinical incompetence. This trend increases costs of health care and in our country is a crucial factor in the cycle of poverty. We cannot afford this trend.

Investigations were meant for us to narrow down and then confirm a clinical diagnosis we have reasoned out based on history and physical examination. There should be a systematic approach to choose Investigations that need to be prioritised during planning an approach to a patient's problems. Simple first line Investigations lead to second and finally third line Investigations. If the first or second line Investigations chosen carefully based on clinical history and examination, clinches and confirms the diagnosis or adds data that further narrows down the differentials then it probably speeds up the process in most cases and more importantly makes health care affordable.

Some may argue that defensive medicine is the practice of today, but there is no replacement for stepping up not only documentation of rational for decisions (clinical reasoning), but doctor-patient communications explaining to patient's and their families the benefits and consequences of good clinical practice of medicine.

This opportunity will enable us to reflect on clinical reasoning in our own practices and as well as our priorities as academic teachers of pediatrics.

Dengue fever- clinical features and management

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Burden of illness

Re-emergence of Dengue over a few decades as a pandemic-prone infection poses major threat to global health particularly in Southeast Asian, Western Pacific, African and Latin American countries. 4 serotypes of dengue viruses DENV 1, DENV 2, DENV 3 and DENV 4 circulate in India making her a hyperendemic country. India had 1,11,880 cases with 227 deaths and Kerala had 7204 cases with 12 deaths reported in 2016 [1]

The Virus

Infection with one serotype provides lifelong immunity to the same but not to other serotypes. Secondary infection with another serotype or multiple infections with different serotypes may lead to severe dengue. Serotype sequence in secondary infections, viral load and virulence also decides severity. Within each serotype, subtypes or lineages exist. There is no time limit to sensitization after a primary infection.

The Vector

Transmission occurs via mosquitoes mainly by Aedes aegypti and occasionally by Aedes albopictus. The maximum horizontal flight range of these mosquitoes is 400 metres only. Hence, human migration spreads the virus more than mosquitoes per se. Aedes aegypti bites more than one host to complete one blood meal resulting in clustering of cases among family members.

Female mosquitoes have two periods of biting activity; in the morning, several hours after daybreak and in the afternoon, several hours before dark. They generally do not bite at night but may do so in lighted rooms. Preferred indoor resting surfaces are the unsprayable undersides of furniture and hanging objects such as clothes, curtains and walls. Hence, unlike female anopheles mosquitoes, residual spraying cannot be used for vector control here.

After a blood meal, extrinsic incubation period of the virus in the vector is around 8-12 days.

Once the virus reaches the salivary glands of the mosquito, it gets transmitted to humans over the rest of mosquito's lifetime (1- 4 weeks). Genital tract of mosquito is also infected with virus and thus enters the fully developed eggs. About 50-120 eggs are laid singly in small clear water collections at one time. In the eggs, embryonic development is usually completed within 48 hrs. Once embryonic development is complete, eggs may remain in viable dry conditions for around a year and hatch within 24 hrs once they come in contact with water during a rainfall. In optimal conditions, time taken from hatching to emergence of adult can be approximately 10 days.

The Host

Intrinsic incubation period of the virus in host is 5-7 days. Viremia coincides with the febrile phase. Viremiabuilds up high titres two days before the onset of fever and lasts 5–7 days after the onset of fever. It is only during these two periods that vector species gets infected.

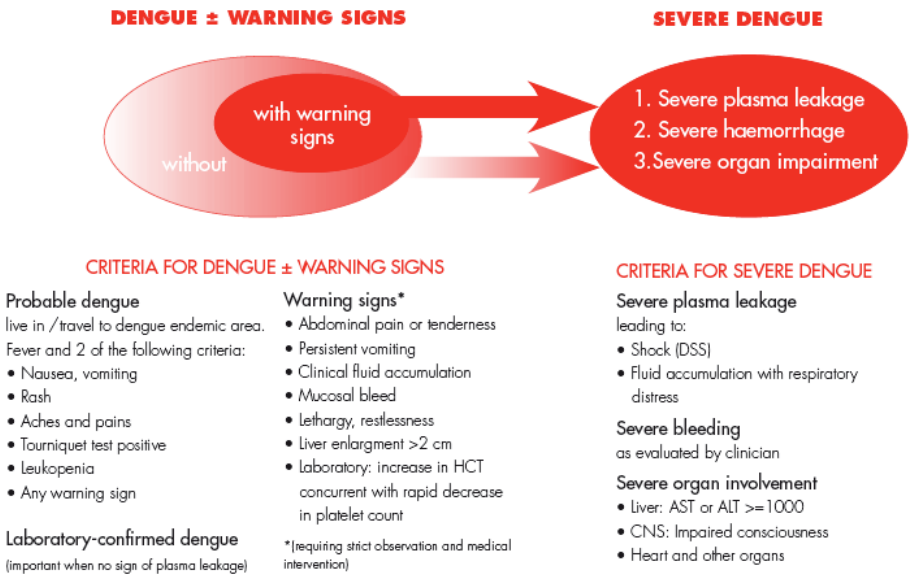
Pathogenesis

Various mechanisms include not only the virus and its antigens but also host genetics, host immune mechanisms, cross reactivity of memory T-cells, enhancing antibodies, complement with its products and various mediators including cytokines and chemokines.

Most favored host mechanisms for progression to severity include role of enhancing antibodies (EA) and memory T-cells in a secondary infection resulting in 'Cytokine Tsunami'[2]. EA bind to the virus and enhance its uptake by monocytes/ macrophages, thus increasing viral burden and disease severity.Immune factors released by EA, infected monocytes & activated cross reactive memory T cells causea transient functional impairment ofthe vascular endothelial glycocalyx. This alters the microvasular permeability leading to plasma leak and shock.

Enhancing antibodies activate complements and release factors that interact with clotting system and platelets causing coagulopathy and thrombocytopenia. Release of heparin sulphate or chondroitin sulphate by the glycocalyx contributes to coagulopathy.Clotting and fibrinolytic systems are activated. Both NS1 and anti-NS1 antibodies have been implicated in pathogenesis of thrombocytopenia and coagulopathy. Thrombocytopenia occurs due to other mechanisms also like auto-antibody mediated destruction, marrow suppression, peripheral sequestration and DIC.

Clinical Features- Revised classification model put forward by WHO (2009) is given below[3].



Febrile phase starts as high grade fever and lasts for 2-7 days. It may be accompanied by aches & pains (arthralgia, myalgia, headache or retro orbital pain), nausea & vomiting and/or rashes. Fever, headache and myalgia develop secondary to host antiviral state in which interferon expression is abundant. Generalized flushing includes face, ears, palms and soles. Flushed areas undergo blanching with digital pressure to produce 'impression sign'. Maculopapular rubelliform exanthems may be seen. Patients may have anorexia, malaise, sore throat, injected pharynx and conjunctival injection. Positive tourniquet test in this phase increases the probability of dengue. Major problem seen in this phase includes dehydration due to fever, vomiting and poor oral fluid intake. Neurological disturbances including febrile seizures can also occur especially in infants.

Defervescence occurs when the temperature drops to 37.5–38^{o}C or less and remains below this level, usually on days 3–7. After defervescence, most of the patients improve without an increase in capillary permeability; they are said to have non-severe dengue.
Around defervescence, capillary permeability increases in some patients. An increase in capillary permeability with rising PCV and appearance of other warning signs mark beginning of critical phase[4]. This phase usually lasts for 24–48 hrs. Severe dengue mostly occurs in critical phase. A low spiking fever may reappear during late critical or recovery phase.

Warning signs

Abdominal pain and tenderness-It is the most common warning sign; it may be due to intestinal wall edema, leaky congestion of abdominal organs, acute hepatitis, acalculous cholecystitis, acute pancreatitis and appendicitis.Tense abdomen due to ascites coupled with liver congestion can also cause abdominal pain; fluid overload should be considered here. 'Pain' is subjective, intensity and site of which is difficult to describe by infants and small children. Abdominal tenderness, a more reliable sign, mostly occurs at right hypochondrium due to hepatitis.

Lethargy/restlessness- Lethargy may be due to fever, dehydration, fasting, vomiting, electrolyte imbalance, hypoglycemia, associated aches and pains, plasma leak with evolving shock or associated hepatitis. During or following a fever, most children appear lethargic. Children prefer to be in bed, sleep most of the time and are uninterested in food or television. Some experience giddiness and fainting episodes if they are made to stand or walk during times of extreme lethargy. A lethargic child or infant feeds and drinks poorly adding to dehydration and metabolic derangement. Restlessness may be the earliest manifestation of hypoxia due to shock. Most children with lethargy have a normal sensorium. Lethargy, a subjective symptom, should not be confused with drowsiness where sensorial alteration occurs. Drowsiness occurs once severe dengue (decompensated shock or CNS organ impairment) sets in.

Persistent vomiting- In Dengue, it occurs due to intestinal wall edema, hepatic involvement or electrolyte imbalance. Coupled with inadequate oral intake, it augments hemoconcentration.A power point module 'Dengue- Clinical Management' prepared by WHO has defined persistent vomiting as 3 or more times per day.

Clinical fluid accumulation- WHO guidelines mention pleural effusion and ascites as features of clinical fluid accumulation. It manifests after reasonable plasma leak or excess intravenous fluid usage. Though radiological evidence precedes clinical signs, latter occurs sufficiently early before onset of severe dengue. Recognition warrants clinical skill**.** Fluid accumulation without respiratory distress is considered a warning sign for severe dengue; once respiratory distress sets in, it marks the onset of severe dengue.

Mucosal bleed- It can occur due to thrombocytopenia, abnormalities of coagulation /fibrinolytic system and possible platelet dysfunction. This includes bleed from GI / GU mucosa, nose or conjunctiva. Most common bleeds occur from the GI mucosa as melena. Minor skin bleeds like petichae and purpurae are not considered as warning signs.

Hepatomegaly>2 cm- Liver involvement is thought to result from hepatocyte apoptosis directly

by the virus, hypoxic damage due to impaired liver perfusion resulting from fluid leakage, oxidative stress or immune mediated injury[5]. Common symptoms that suggest liver involvement are nausea, vomiting, anorexia and abdominal pain. Pathogenesis of hepatomegaly includes hepatitis and vascular leak. In a review by Samanta et al, it is stated that hepatomegaly in dengue is commoner in children (37.5-80.8%) than in adults (4-52%)[6].

Rise in hematocrit concomitant with a rapid fall in platelets to less than 1 lakh/mm^3- Progressive leukopenia (<5000cells/mm^3) in the febrile phase is followed by a rapid decrease in platelet count which in turn precedes plasma leakage. At this point, patients without plasma leak improve without going to critical phase.These lab features may be the earliest warning signs to appear.

Low platelet counts have a poor correlation with hemorrhage but correlates more closely with the severity of plasma leakage.Clubbing them as one warning sign by WHO has definite scientific backup; the word 'rapid' needs clarification. WHO 1997 classification has defined 'rising hematocrit' as 20% above baseline of that person. 'Rising hematocrit' has not been defined in the WHO 2009 classification probably due to the fact that baseline hematocrits and age appropriate region specific reference hematocrits are difficult to obtain. The degree of increase above the baseline reflects severity of plasma leak. Rise of hematocrit may not be detectable in infants because normal value in infants is usually low and may be even lower in iron deficiency anemia.

In initial stages of **compensated shock**, subnormal body temperature, tachycardia, low volume pulse, quiet tachypnea,cold clammy extremities, oliguria and delayed capillary filling occur with a normal blood pressure. Diastolic BP rises towards the systolic pressure and pulse pressure narrows to ≤20mm Hg as the peripheral vascular resistance increases. Consciousness will be preserved in compensated shock due to dengue; both the caretaker and the treating doctor may fail to identify gravity of the situation unless they are highly vigilant. If not intervened in time, **decompensated shock** ensues with hypotension. An irritable combative patient indicates severe shock with declining cortical perfusion. Confusion, seizures and coma will set in very quickly. Other causes of change in mentation are fulminant hepatic failure, hypoglycemia, electrolyte abnormalities and rarely, intracranial bleed and encephalitis. With prolonged shock, organ hypoperfusion results in progressive multiple organ dysfunction, metabolic acidosis and DIC.

Patients at risk of **severe bleeding** are those who have profound/ prolonged/refractory shock and those who have hypotensive shock and multi-organ failure or severe and persistent metabolic acidosis. Coagulation abnormalities per se are insufficient to result in severe bleeds in dengue. Further decrease in hematocrit and increase in total count occur in patients with severe bleed. **Severe organ impairment** such as severe hepatitis, encephalitis or myocarditis may develop rarely without plasma leak or shock.

If the patient survives 24–48 hrs of critical phase, a gradual reabsorption of extravascular fluid back into circulation takes place in the next 48–72 hrs; this phase is the recovery or convalescent phase. General well-being improves, appetite returns, GI symptoms abate, haemodynamics stabilize and diuresis ensues. Rashes described as 'isles of white in a sea of red' may be seen especially in extremities. A band of intense erythema may be seen along the margins of soles called Surat sign. Itching may be associated with rashes. Bradycardia and ECG changes are common. PCV stabilizes or may be lower due to dilutional effect of reabsorbed fluid. Total count usually rises soon after defervescence; this is followed by recovery of platelet count.

One criteria for severe dengue is severe plasma leakage leading to fluid accumulation with respiratory distress. Hypervolaemia and pulmonary edema (respiratory distress, wheeze, rhonchi and fine crepitations) occurs only if IVF has been excessive and/or has extended into recovery phase.Fine crepitations may not be audible if breathing effort is poor. For pleural effusion to cause respiratory distress, it should be substantial and easily detectable. Diaphragm may be splinted by tense ascites. This scenario usually occurs after unrestrained aggressive IVF, not before. Patients with respiratory distress from pulmonary causes like pleural effusion and pulmonary edema will mostly sit up for optimal chest wall mechanics. In metabolic acidosis, breathing become deeper and faster and patient prefers to lie down due to hypovolemia.

Dengue fever in infants and children-Children are at higher risk for morbidity and mortality due to capillary leak, shock, severe bleed and organ failure. Children are more likely to develop plasma leakage and shock; adults more likely to develop organ impairment and severe bleed[7].Children may be more susceptible to shock-related GI bleed than adults because of differences in integrity of vascular endothelial barriers. Severe dengue can occur during primary dengue infections too, most frequently in infants whose mothers are immune to dengue. Burden of severe dengue lies mainly in infants 4–9 mo of age. Upper respiratory tract symptoms (cough, nasal block, rhinitis, dyspnoea), GI symptoms (vomiting, diarrhoea) and febrile convulsions are more common in infancy. Febrile convulsions may be confused with CNS manifestations of severe dengue. Hepatic involvement is more pronounced in infants. Splenomegaly is seen in almost 10% infants. It is often not possible to differentiate between dengue and other infections in infants such as pneumonia, measles,meningitis and rotaviral diarrhea in the febrile phase. Septic shock should always be kept in mind in infants who present with fever and shock.

Investigations-

NS1Ag (ELISA) done on days 1-5 is a marker of acute dengue infection with high sensitivity and specificity. Virus genome detection with RT-PCR done on day 1-5 confirms acute infection.

Development of IgM antibody coincides with disappearance of fever and viremia and is the best marker of recent infection. Primary infections are characterized by high levels of IgM and low

levels of IgG, while low levels of IgM with high levels of IgG characterize secondary infections. IgM antibodies can be detected in primary infections in 50% of cases by days 3–5,80% by day 5 and 95–98% by days 6−10. In primary infections,IgG appear by day 7,but persists for years. In dengue endemic countries, acute clinical cases with a positive IgM are classified as probable dengue cases. Low levels of detectable IgM response or even absence of it in some secondary infections reduces the diagnostic accuracy of IgM ELISA tests. A positive IgG may indicate past infection and is not diagnostic. Though not practically possible, study of paired sera (acute and convalescent samples with second onecollected 15–21 days after the first)allows for serological confirmation by demonstration of either IgM or IgG seroconversion or 4 fold rise in IgGtitres.

During a secondary infection, a rapid and higher increase of IgG with slower and lower increase of IgM occurs.High IgG levels remain for 30–40 days. Classification into primary or secondary infection can also be determined using the IgM/IgG optical density ratio. Ratios greater than 1.2 (using the patient's sera at 1/100 serum dilution) or 1.4 (using serum dilution of 1/20) suggest primary infection. IgGtitres higher than 1/1280 by HIA or ELISA suggests secondary infection.Values below this are generally observed in convalescent sera from patients with primary responses.RDT kits for IgM and IgG antibodies and NS1Ag are available which give results within 15 to 25 minutes. But due to poor accuracy, they are not recommended.

Treatment[4] -

Patients without warning signs (Group A) may be sent home if they can tolerate oral fluids. They should undergo daily clinical review(look for appearance of warning signs and features of shock) and lab monitoring (TC, PCV, platelet count). Paracetamol may be given for fever. Avoid aspirin or NSAIDS. Danger signs should be explained.

Patients with any warning sign or coexisting condition (infancy, obesity, diabetes mellitus, chronic renal/liver/heart disease, chronic hemolysis, socially deprived) (Group B) should be admitted for close monitoring (pulse rate, pulse volume, capillary filling time, color and temperature of extremities, respiratory rate, BP, pulse pressure, urine output, sensorium).Cases of dengue with warning signs will usually recover with early intravenous rehydration[3,4]. After obtaining a blood sample for PCV, start an isotonic IVF (0.9% NS) 5–7 ml/kg/hr for 1–2 hrs, then 3–5 ml/kg/hr for 2–4 hrs, and then 2–3 ml/kg/hr. Reassess clinically and monitor PCV 4-6 hrly. Give minimum IVF to maintain good perfusion and a urine output of 0.5ml/kg/hr. Baseline LFT and RFT should be done. If clinical condition worsens and PCV rises rapidly, give 10ml/kg/hr for 1-2 hrs and reassess clinical status and PCV.

WHO guidelines recommend IVF if any warning sign is present[3].If close observation is possible, decisions regarding IVF administration may be individualized. A clinically stable child taking plenty of oral fluids may not be given IVF simply because his PCV is high. A study was conducted in our institution to develop a prognostic prediction model to determine severe dengue using 7 WHO warning signs[8]. Among the 7 warning signs, clinical fluid accumulation, PCV≥40 with

platelet count<1 lakh/mm^3, persistent vomiting and mucosal bleed were found to be significant predictors.

Patients with severe dengue (severe plasma leak leading to shock and/ or fluid accumulation with respiratory distress, severe bleed or severe organ failure)(Group C)-
In compensated shock, start with **0.9% NS 10-20ml/kg/hr for 1 hr.**
If improving, gradually titrate IV crystalloid as 10-7-5-3 over 6-8 hrs. Stop IVF after 24-48 hrs.
If not improving & PCV rising, second bolus of 0.9% NS or colloid 10-20ml/kg/hr for 1 hr and titrate as above if better. Repeated boluses of 0.9%NS may cause hyperchloremic acidosis. So it is better to switch to another isotonic fluid after 2 boluses of 0.9%NS. Gel based colloids are better than starch based colloids as the latter may sometimes cause bleed and renal failure
If the patient is not improving and PCV is falling, suspect severe overt bleed. Fresh whole blood (10-20ml/kg) or fresh packed cells (5-10ml/kg) should be given if there is severe bleed. If no evidence of bleed is obtained, colloid 10-20ml/kg/hr for 1 hr is given and titrated as above.

In decompensated shock due to dengue, initiate **crystalloid or colloid at 20 ml/kg as a bolus given over 15–30 minutes** (colloid preferred in those with pulse pressure less than 10 mmHg).
If the patent improves, give one more colloid infusion 10 ml/kg/hr for 1 hr, then crystalloid 7.5 ml/kg/hr for 2 hrs, 5 ml/kg/hr for 4 hrs and 3 ml/kg/hr, which can be maintained for up to 24–48 hrs. The total duration of IVF should not exceed 48hrs.
If vitals are unstable and PCV is falling after the first bolus, look for severe overt bleed. Fresh whole blood (10-20ml/kg) or fresh packed cells (5-10ml/kg) should be given if there is severe bleed. If no evidence of bleed is obtained, colloid 10-20ml/kg/hr for 30min-1 hr is given and titrated depending on the clinical condition and PCV. If vitals are still unstable and PCV is falling, this indicates bleeding;transfuse blood as soon as possible. Concealed bleeding may take several hours to become apparent.
If vitals are unstable and PCV rising after first bolus, give colloid 10−20 ml/kgover 30min-1 hr. After the second bolus, reassess the patient. If the condition improves, reduce the colloid rate to 7−10 ml/kg/hr for 1−2 hrs,then change back to crystalloid and reduce the rate of infusion as mentioned above. If vitals are still unstable and PCV rising, continue colloid 10−20 ml/kg over 1 hr. After this dose, reduce the colloid rate to 7−10 ml/kg/hr for 1−2 hrs,then change back to crystalloid. Reduce the rate of infusion as mentioned above when patient's condition improves.
Higher the fluid infusion rate, the more frequently the patient should be monitored to avoid fluid overload.There is no evidence that supports transfusion of platelet concentrates and/or fresh-frozen plasma for severe bleeding. This may cause fluid overload as well. Role of inotropes and vasopressors are not primary; fluid management is the crucial element in dengue shock management. Clear evidence has not been obtained with steroids as well.

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Infections in Children – The Inside Story

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The classical teaching over the years has been that infections in children are largely determined by modifiable risk factors like their environment and child rearing practices. Thus mothers took the brunt of the blame- either the house and environs were too dirty or overcrowded, or the parents needed advice on how to look after their children in the proper way. Often, their educational background was called into question or they were seen as failing in their responsibility. The socioeconomic history was often seen as damning.
We have known all along that the first years at school bring with them coughs and colds and boils and weepy eyes, and that children with a family history of atopy sneeze and wheeze like nobody's business.

While these risk factors are not to be trifled with, the recognition of the primary immune deficiency disorders is leading to a paradigm shift in our thinking. These are inherited disorders of various components of the immune system that may raise alarm bells in childhood or much later in life. Thus a heightened susceptibility to infections runs in the genes. With high rates of consanguinity in our population, these disorders are being increasingly recognized.
Severe, persistent, unusual, recurrent or recalcitrant infections should make us sit up and think.
Phenotypic features often hold the key to the diagnosis.
Thus the distinctive facies of Di George syndrome and the old man facies of the hyper IgE syndrome or the eczema, draining ears and bleeds of Wiskott Aldrich syndrome or the silvery hair and blonde appearance of Chediak Higashi syndrome are dead give aways.
Sometimes, the organism causing the infection leads us in the right direction.
The severe EBV infections in X linked lymphoproliferative syndrome, Neisserial meningitis and sepsis in terminal complement deficiencies and environmental mycobacteria in Mendelian Susceptibility to Mycobacterial disease are cases in point.

There is a genuine need for pediatricians to recognize these disorders that are not very common, but which if diagnosed and treated early can make a world of difference. Early diagnosiscan tilt the balance between life and death or between a normal life or one with

severe disability.

Investigations for these disorders are however, just becoming available in referral centres in our region and often need expertise in interpretation apart from being expensive. Hence, it is important to have a high index of suspicion. There are warning signs put forward by the European Society for primary immune deficiency that are applicable to our situation as well.
More than four new ear infections in a year, more than two episodes of pneumonia in a year, recurrent deep seated abscesses, two episodes of meningitis or septicemia, two months or more on antibiotics with poor response, a positive family history and persistent oral thrush are included.

There is a genuine need to learn more about these disorders, most of which are single gene defects, so that young lives can be saved. Moreover, a genetic diagnosis is becoming a reality with access to next generation sequencing and therapeutic options like stem cell transplants are within our reach. Genetic counseling assumes significance with antenatal diagnosis becoming available.
The time is ripe to improve our understanding about these disorders so that our patients and their families can reap the benefits. A knock on the door could mean a friendly neighbor, the milkman, the postman….. But it could also be a deadly criminal with the intent to kill or maim.

RNTCP – What is new ?

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India contributes one fourth of total world TB cases. Forty percentage of Indian population is infected with TB. Pediatric cases represent 6-10% of TB burden of India. The risk of developing active disease following primary infection is 43% below 1yr, 24% below 5 yrs and least in 5–10 year age group. Pulmonary parenchymal disease and intrathoracic adenopathy account for 60%–80% of pediatric TB. Extra pulmonary disease more commonly occur in children which represent 25 - 30 % of childhood TB. Adolescents develop an adult-like disease and could be infectious also.

Lack of typical symptoms and signs, unusual sites of disease, absence of a gold standard for diagnosis, increased prevalence of extra-pulmonary disease and a lower public health priority are the challenges facing TB care in children. In 2016 RNTCP has revised the management guidelines of TB which will be introduced in a phased manner from march 2017 onwards. Important changes will be discussed here.

Symptom characterization for case finding:

Very important in the evaluation to avoid over diagnosis and under diagnosis.

Fever: Should be persistent (>2weeks), unexplained, >38ºC, reported by guardian or recorded at least once.

Cough: should be **persistent** & **unremitting**. Cough and fever which is recurrent and, associated with cold is not a TB suspect.

Definitive weight loss or FTT: No weight gain in 3months or unexplained weight loss(more than 5% of the highest weight recorded in the past three months).

Lymph nodes: Maximum prevalence between 5 and 9 year and neck is the most common site (jugular, posterior triangular, supraclavicular). Nodes enlarge over weeks or months. Systemic symptoms are common. Lungs are usually the primary focus.

History of contact: In a symptomatic child, contact with a person with any form of active TB within *last 2 yrs* and in an asymptomatic child, exposure to a smear positive TB patient are considered as a positive contact history.

Diagnosis of Tuberculosis

Cartridge Based Nucleic Acid Amplification Tests(CBNAAT): Theseare fully automated nucleic acid amplification that integrate sample preparation with real-time PCR amplification. Line Probe Assay and Xpert MTB/RIF are the two CBNAATs endorsed by the WHO. GeneXpert is the available Xpert MTB/RIF test under RNTCP. It detects tubercle bacilli and RIF resistance from sputum and also specimen from extra pulmonary sites. It is an automated cartridge based closed system. Once loaded with specimen, DNA of bacilli is amplified and detected on real time. Results are displayed on the system in printable form in 2 hours. Minimum Bacilli needed for detection is 131/ml. It is 37% more sensitive on sputum and 44% more sensitive on gastric aspirate compared to direct microscopy. In extra pulmonary samples, yield is good for CSF, lymph node specimen etc, but poor for pleural fluid. Currently Xpert MTB/RIF is the first line bacteriological test recommended by RNTCP in suspected DR TB, presumptive TB in children, extra pulmonary TB and (PLWHIV).

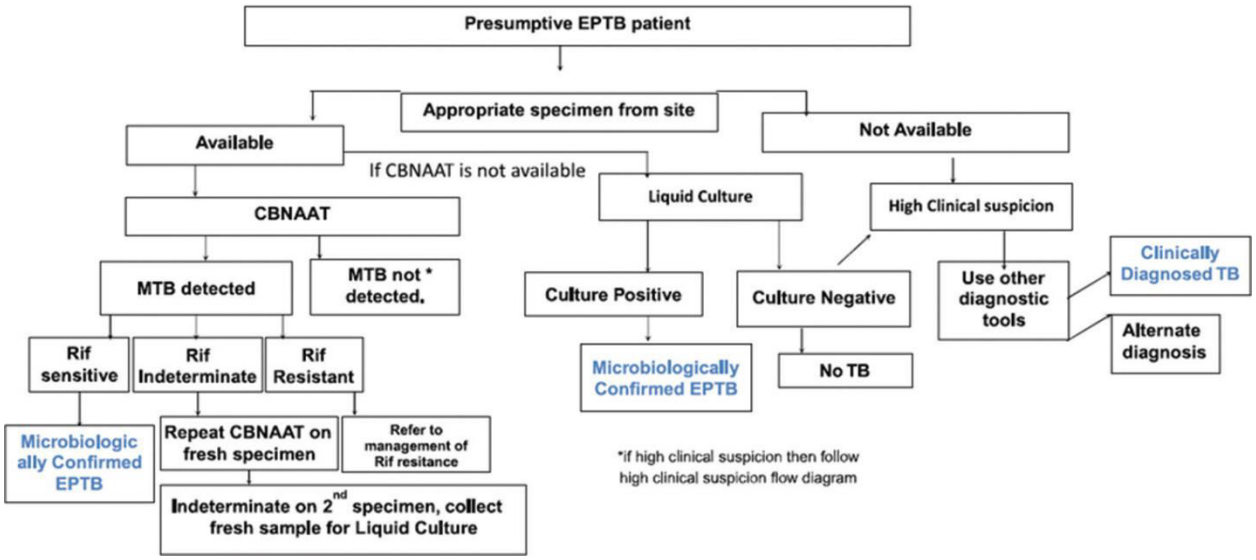
Direct microscopy

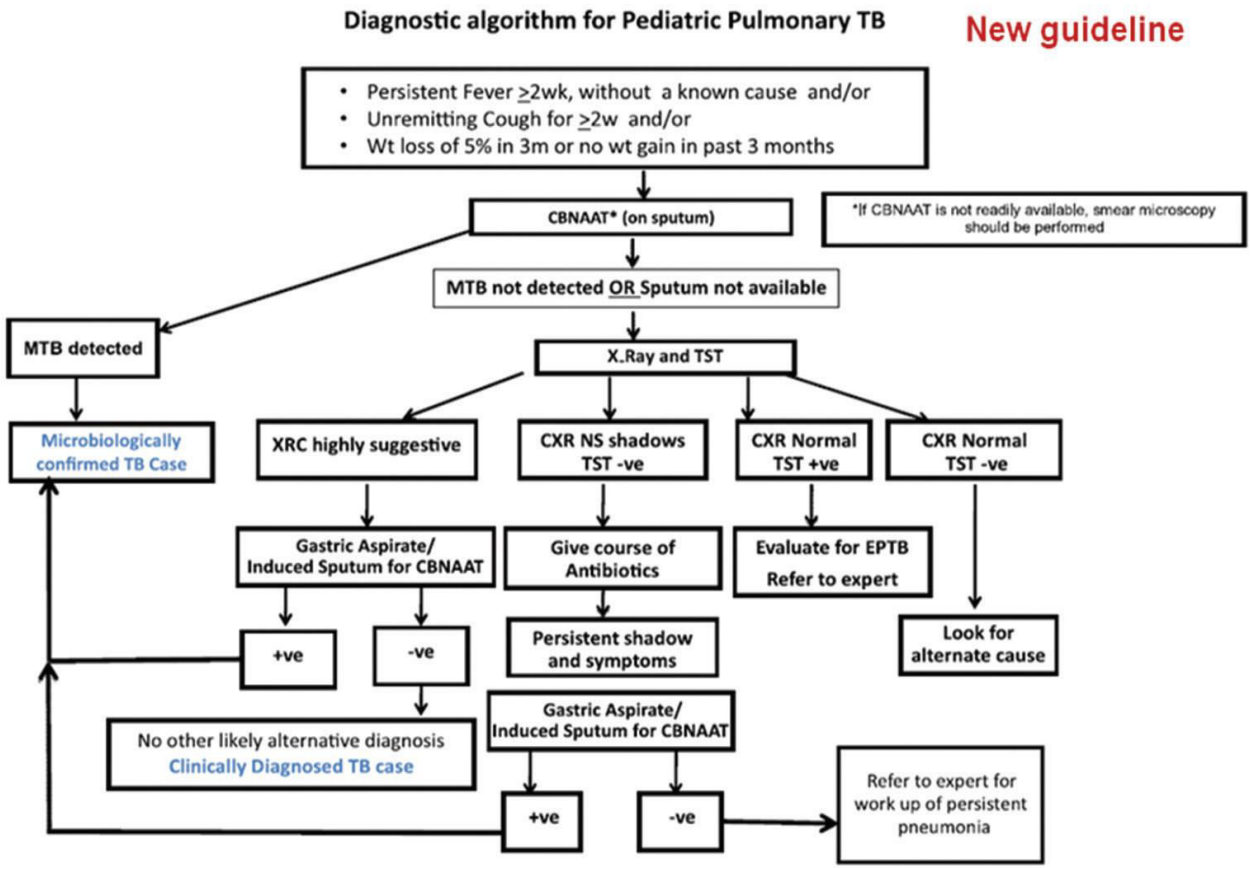
In children and Patients Living with HIV (PLWHIV), smear microscopy is now advised only if NAAT is not readily available

Sample collection: Gastr ic aspirate (GA),Induced sputum(IS) and Bronchial alveolar lavage (BAL) are the various modes. Can be performed in ambulatory setting also.

Tuberculin skin test: Can be used along with radiology. Tuberculosis cannot be diagnosed based on TST alone. Radiology has an important role in the diagnosis. X ray lateral view of chest, CT thorax, etc, can be used in selected cases.

Diagnostic Algorithm for Extra Pulmonary TB





Changes in the drug regimen in the new guidelines

1. Principle of TB treatment is being shifted from intermittent to daily regimen
2. First-line Anti TB drugs will be available in the form of **daily fixed dose combinations (FDC)** of as per appropriate
3. Drugs available in different weight bands.
4. Drugs in FDC will meet the revised drug dosage of WHO.
5. Ethambutol included in IP and CP.

Treatment for *new TB cases*

Intensive phase: 8 weeks of INH,Rifampicin, Pyrazinamide and Ethambutol in daily dosages as per four weight bands.No extension of IP (previousely in poor or no response at 8 weeks of IP, 4 wks extension was done). Only Pyrazinamide will be stopped in CP while other three drugs will be continued for another 16 weeks as daily dosages.

Previously treated:. IP will be of 12 weeks, where Streptomycin stopped after 8 weeks and the remaining four drugs in daily dosages for another 4 weeks. No need of extension of IP.

Extra-pulmonary TB: CP in both new and previously treated cases extended 3–6 months in CNS, skeletal, disseminated TB. Extension beyond 3 months is based on recommendation by experts in this this field.

Treatment regimen for drug sensitive TB

Type of Patient	Intensive Phase	Continuation Phase
New	2HRZE	4HRE
Previously Treated	2HRZES + 1HRZE	5HRE

Prefixed number denotes months

Fixed Drug Combinations (FDC):

Currently available drug combination and some commercially available FDC are having an INH/RIF ratio of 1:1. The ratio in newly recommended FDC is 2:3. It provides INH 10 mg/kg, RIF 15 mg/kg and PZA 30 mg/kg

Weight category	Number of tablets (dispersible FDCs)		
	Intensive phase		Continuation phase
	HRZ	E	HRE
	50/75/150	100	50/75/100
4-7 kg	1	1	1
8-11 kg	2	2	2
12-15 kg	3	3	3
16-24 kg	4	4	4
25-29 kg	3 + 1A*	3	3 + 1A*
30-39 kg	2 + 2A*	2	2 + 2A*

A = Adult FDC for IP - HRZE=75/150/400/275 ; A*for CP - HRE= 75/150/275

Government of India has taken a decision to introduce daily regimen for treatment of drug-sensitive TB patients under Revised National TB Control Programme in a phased manner. Initially, it is going to be introduced for all TB patients in five states, that is, Bihar, Maharashtra, Sikkim, Himachal Pradesh and Kerala. ***In addition, all HIV-infected TB patients and paediatric TB cases in entire country will be provided daily regimen under the programme***

PREVENTION OF NOSOCOMIAL INFECTION: VAP & CLABSI

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Nosocomial infections are a threat to patient safety.They are the most frequent adverse events in health-care delivery worldwide. Hundreds of millions of patients are affected by health care-associated infections worldwide each year, leading to significant mortality and financial losses for health systems. Of every 100 hospitalized patients at any given time, 7 in developed and 10 in developing countries will acquire at least one health care-associated infection.

Ventilator associated pneumonia

Ventialtor associated pneumonia is a serious complication of ventilation with high mortality. Possible VAP is defined as Gram stain evidence of purulent pulmonary secretions or a pathogenic pulmonary culture in a patient on ventilator. Probable VAP is defined as Gram stain evidence of purulence plus quantitative or semiquantitative growth of a pathogenic organism.

Basic practices (intervention lowers VAP rates and minimal risks of harm; potential benefits likely outweigh potential risks).

- Use noninvasive positive pressure ventilation for selected populations when feasible
- Assess readiness to extubate daily using spontaneous breathing trials in patients without contraindications
- Avoid unplanned extubations.
- Provide regular oral care (ie, toothbrushing or gauze if no teeth)
- Elevate the head of the bed to 30°–45°
- Change ventilator circuits only if visibly soiled or malfunctioning
- Use cuffed endotracheal tubes
- Prevent condensate from reaching the patient.

Special approaches (Unknown impact on VAP rates, but risk of harm likely minimal; reasonable to consider implementing if rates remain elevated despite basic practices)

- Interrupt sedation daily.

- Prophylactic probiotics
- Utilize endotracheal tubes with subglottic secretion drainage ports for older pediatric patients expected to require greater than 48 or 72 hours of mechanical ventilation.

Generally not recommended (Unknown impact on VAP rates and inadequate data on risks **)**

- Systemic antimicrobial therapy for ventilator-associated tracheobronchitis
- Selective oropharyngeal or digestive decontamination

No impact on VAP rates

- Oral care with antiseptics, such as chlorhexidine (in adults found to be useful but no impact in children)
- Stress ulcer prophylaxis
- Early tracheotomy
- Thromboembolism prophylaxis
- Silver-coated endotracheal tubes (lower rates in adults).

Central line associated blood stream infection (CLABSI) prevention.

CLABSI is an important cause of hospital acquired blood stream infection. The following strategies may be undertaken to decrease BSI in hospitalised patients.

Factors associated with increased risk include

1.Prolonged hospitalization before catheterization
2.Prolonged duration of catheterization
3.Heavy microbial colonization at the insertion site
4.Heavy microbial colonization of the catheter hub
5.Internal jugular catheterization
6.Femoral catheterization in adults
7.Neutropenia
8.Prematurity (ie, early gestational age)
9.Reduced nurse-to-patient ratio in the ICU[26,27]
10.Total parenteral nutrition
11.Substandard catheter care (eg, excessive manipulation of the catheter)
12.Transfusion of blood products (in children)

Factors associated with reduced risk.
1.Female sex
2.Antibiotic administration
3.Minocycline-rifampin-impregnated catheters

Basic practices recommended for preventing and monitoring CLABSI for all acute care hospitals include

- Units should have a guideline for indications for CVC use to minimize unnecessary CVC placement .
- Educate healthcare personnel involved in insertion, care, and maintenance of CVCs about CLABSI prevention. Include the indications for catheter use, appropriate insertion and maintenance, the risk of CLABSI, and general infection prevention strategies.
- Bathe ICU patients over 2 months of age with a chlorhexidine preparation on a daily basis The role of chlorhexidine bathing in non-ICU patients remains to be determined.The optimal choice of antiseptic agents is unresolved for children under 2 months of age. However, chlorhexidine is widely used in children under 2 months of age. Alternative agents, such as povidone-iodine or alcohol, can be used in this age group.
- Ensure and document adherence to aseptic technique during CVC insertion using a checklist.
- Perform hand hygiene prior to catheter insertion or manipulation (Use of gloves does not obviate hand hygiene)
- Use maximal sterile barrier precautions : A mask, cap, sterile gown, and sterile gloves are to be worn by all healthcare personnel involved in the catheter insertion procedure.
- The patient is to be covered with a large ("full-body") sterile drape during catheter insertion
- Before catheter insertion, apply an alcoholic chlorhexidine solution containing more than 0.5% CHG to the insertion site The antiseptic solution must be allowed to dry before making the skin puncture.
- Avoid jugular vein catheterisation in children with tracheostomy.
- Ensure appropriate nurse-to-patient ratio (1:2) and limit the use of float nurses in ICUs .
- Before accessing catheter hubs, needleless connectors, or injection ports, vigorously apply mechanical friction with an alcoholic chlorhexidine preparation, 70% alcohol, or povidone-iodine. Alcoholic chlorhexidine may have additional residual activity compared with alcohol for this purpose
- Assess the need for continued intravascular access on a daily basis . Remove catheters not required for patient care.
- Change transparent dressings and perform site care with a chlorhexidine-based antiseptic every 5–7 days or immediately if the dressing is soiled, loose, or damp; change gauze dressings every 2 days or earlier if the dressing is soiled, loose, or damp

- Replace administration sets not used for blood, blood products, or lipids at intervals not longer than 96 hours.
- Use antimicrobial ointments for hemodialysis catheter-insertion sites. Mupirocin ointment should not be applied , povidone iodine or Polysporin “triple” may be used.

Reference:

Health care associated infections fact sheet. World health organization

Strategies to Prevent Central Line–Associated Bloodstream Infections in Acute Care Hospitals: 2014 Update Infection Control and Hospital Epidemiol.Vol. 35, No. 7, July 2014

Strategies to prevent ventilator associated pneumonia in Acute Care Hospitals: 2014 Update. CDC guidelines

Malaria – Essentials for Management

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Malaria is one of the major public health problems of the country, accounting for 75% of all malaria cases in South East Asia.

Core principles of malaria control & elimination strategies include

1.Early Diagnosis & prompt effective management
2.Rational use of antimalarial agents
3.Combination therapy
4.Appropriate weight based dosing

The symptoms of malaria can be non-specific and mimic other diseases like viral infections, enteric fever etc. Malaria should be suspected in patients with fever, residing in endemic areas or who have recently visited endemic area,without any apparent cause for the fever.

All clinically suspected malaria cases should be investigated immediately by microscopy and/or Rapid Diagnostic Test (RDT).Malaria RDTs should be used if quality assured malaria microscopy is not readily available.If the initial blood film examination is negative in patients with manifestations compatible with severe malaria a series of blood films should be examined at 6-12h intervals or a RDT(preferably 1 detecting PfHRP2) should be performed. If both the slide examination& the RDTs are negative, malaria is extremely unlikely& other causes of the illness should be sought &treated.

All fever cases diagnosed as malaria by RDT or microscopy should promptly be given effective treatment. Confirmed P. vivax cases should be treated with chloroquine in full therapeutic dose of 25 mg/kg along with primaquine0.25 mg/kg body weight daily for 14 days under supervision. Artemisinin Combination Therapy (ACT) should be given to all the confirmed P. falciparum cases found positive by microscopy or RDT. This is to be accompanied by single dose of primaquine (0.75 mg/kg body weight) on Day 2.

The ACT recommended in the National Programme all over India except northeastern states is artesunate (4 mg/kg body weight)daily for 3 days and sulfadoxine (25 mg/kg body weight) -pyrimethamine (1.25 mg/kg body weight) [AS+SP] on Day 1.

Mixed infections with P. falciparum should be treated as falciparum malaria.

Severe malaria is an emergency, and treatment should be given promptly. Parenteral artemisinin derivatives or quinine should be used as specific antimalarial therapy.Once the patient can tolerate oral therapy or after at least 24 hours of parenteral therapy, further follow-up treatment should be with full course of oral ACT.Severe malaria caused by P. vivax should be treated like severe P. falciparum malaria, however, primaquine should be given for 14 days for preventing relapse as per guidelines after the patient recovers from acute illness and can tolerate primaquine.

H1N1-UPDATES 2017

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*In the late March and early April 2009, an outbreak of H1N1 influenza A virus infection was detected in Mexico, subsequently in many other countries including the USA

*In June 2009,the WHO raised its pandemic alert level to the highest phase 6,indicating widespread community transmission on at least two continents

*The pandemic was declared to be over in August 2010.

CDC case definitions

***ILI [Influenza like illness]**

Fever[>or =100 degree F or 37.8 deg] with cough and sore throat in the absence of a known cause other than influenza.

***A confirmed case of pandemic H1N1 influenza A**

An individual with an ILI with lab confirmed H1N1 influenza virus A detection by real time Reverse transcriptase PCR or culture.

Transmission

- Swine influenza viruses can be transmitted to humans via contact with infected pigs or environments contaminated with swine influenza viruses.

- An infected human being can spread the virus to other humans presumably in the same way as seasonal influenza is spread.

- Droplet exposure of mucosal surfaces[nose,mouth and eye] by respiratory secretions from coughing and sneezing

- Contact usually of hands with an infectious patient or fomites.
- Small particle aerosols in the vicinity of an infectious patient.
- **Incubation period**—1 to 7 days
- **Peak viral shedding** occurs on day 1 of symptoms
- **Communicability** of illness lasts from 1 day before to 7 days after the onset of symptoms.
- If the illness persists for >7days, communicability may persist till resolution of illness
- Children may spread the virus for a longer period

CLINICAL MANIFESTATIONS

- ASYMPTOMATIC - confirmed by rt-RT PCR of nasopharyngeal specimens or from serology

- UNCOMPLICATED INFLUENZA.
- COMPLICATED OR SEVERE INFLUENZA.

Uncomplicated influenza[as per WHO]

- ILI symptoms - fever, cough, sorethroat, nasal congestion,rhinorrhea,headache,myalgia/malaise

  [NO DYSPNOEA]

- GI symptoms may be present- diarrhoea/vomiting but [NO DEHYDRATION]

- Atypical symptoms without any fever,in elderly or immunocompromised people.

Complicated/severe influenza

-Dyspnoea on exertion or at rest

-Tachypnoea/radiological e/o pneumonia

-CNS involvement - encephalitis/encephalopathy

-Severe dehydration/shock----with secondary MODS

Myocarditis,Rhabdomyolysis,AKI,sepsis

-Exacerbation of underlying chronic disease such as Asthma,COPD,C/c liver disease,C/c renal disease

-Co existent bacterial pneumonia

Continued…..

Any of the signs and symptoms of progressive disease listed below

1] symptoms and signs s/o oxygen impairment/cardiopulmonary insufficiency

a] breathlessness,cyanosis,acute chest pain,bloody or coloured sputum

b] in children----worsening tachypnoea

c] Hypoxia as evidenced by pulse oximetry,ABG

2]Symptoms and signs s/o CNS involvement-

altered mental status,unconsciousness,drowsiness,recurrent or persistent seizures,confusion ,focal neurological deficits such as weakness/paralysis

3] Evidence of sustained viral replication or active secondary bacterial infection based on lab testing/clinical signs.[persistent fever and other symptoms beyond 3 days]

4]Severe dehydration manifested by decreased activity,dizziness,decreased urine output.

High risk groups

- Children less than 5 yrs of age,particularly < than 2 yrs
- Adults more than 65 years of age
- Pregnant women and women upto 2 weeks post partum
- Individuals with certain medical condition[already mentioned]
- Those on immunosuppressive therapy for malignancy/post transplant .
- Health care workers and emergency medical personnel.

Newer classification:as per DHS.

ILI WITH 3 CATEGORIES

*Category A --- mild , self limiting --- NO OSELTAMIVIR

*Category B ----

B1---significantly ill----treat with Oseltamivir

B2---mild, but comorbid conditions present ----- treat with Oseltamivir

*Category C----VERY ILL----treat with Oseltamivir.

H1N1 testing

- For Category A and B----NO TESTING NEEDED ROUTINELY,ONLY FOR DISTRICTS SENTINEL SURVEILLANCE

- For Category C---Test may be needed in some circumstances,BUT DO NOT WAIT FOR THE TEST RESULTS.

- SENTINEL TESTING---

  a] NOW NEEDS TO BE DONE REGULARLY FOR EPIDEMIOLOGICAL PURPOSE

  b] FOR CONSTANT MONITORING OF PREVALENCE IN A REGION/DISTRICT

  C]SWAB POSITIVITY RATE

Testing centres—

*Virology Division,MCVR,KMC Hospital,Manipal,Karnataka

*NIV Unit,Medical college,Alappuzha

General advice treatment

- Home isolation
- Reassessment of cases on home---applicable to category A and B only
- Supportive care

Oseltamivir dosage schedule

weight	dosage
<15 kg	30 mg BD for 5 days
15-23 kg	45 mg BD for 5 days
24-40kg	60 mg BD for 5 days
>40 kg	75 mg BD for 5 days

For infants

< 3 months	12 mg BD for 5 days
3-5 months	20 mg BD for 5 days
6-11 months	25 mg BD for 5 days

SPECIAL DOSE

- If needed ,dose and duration can be modified as per clinical condition, IN CATEGORY C cases ONLY
- For example,if the response to the treatment as assessed by the treating team of doctors is

‘NOT ENOUGH’, the dose may be increased to 150 mg BD on a one to one basis.

Chemoprophylaxis

- Wide spread chemoprophylaxis to family/school/social contacts of a positive case is NOT NEEDED.

- BUT

  *For those contacts with high risk---YES

*For other contacts-----Reassure-----Recommend watchfulness----assess category ---if and when they become symptomatic,then treat as per ABC Guidelines.

General guidelines for schools/educational instituitions if outbreak develops

- Assembly to be limited to once a week or preferably less
- Screening of students in the class by class teachers for symptoms of flu
- Leave for teachers and and other employees if they develop flu like symptoms.
- NO MEDICAL CERTIFICATE to be insisted on from preventive absentees
- Promote frequent handwash with soap and water

continued

- Promote frequent handwash with soap and water
- All to observe strict cough/sneeze etiquette
- Regular cleaning with the cleaning agent they oridinarily use.
- Closure of schools not routinely recommended.Contact DMO/DSO for advice
- Display the “DO s and DON‘T”s for ILI s,H1N1 infection at all important places

VACCINES AVAILABLE AGAINST INFLUENZA

1) Inactivated injectable trivalent

Vaxigrip-TM (Sanofi Pasteur), Agripal-TM (Panacea Biotech/ Novartis Vaccines), Influren-TM (Lupin Lab), HNVAX-TM (Bharat Biotech)

2) Live attenuated nasal influenza vaccine

Nasovac-S-TM (By Serum Institute of India)

- All the vaccines provide protection against the currently circulating swine flu vaccine

- The efficacy of vaccine may be about 70% to 80%, especially in geriatric age group

- Major circulating strain in India is of A (H1N1) virus (also called pandemic swine flu virus of 2009) which is not drifted and antigenically the same that contained in most of the current vaccines.

- So all the available flu vaccines will be effective against currently circulating H1N1 virus, but not against other two viruses (H3N2 & B group viruses)

According to IAP, following group of individuals should be offered the vaccine

1-Children with certain high-risk conditions

2-Health care professional including pediatricians

3-Laboratory personnel and healthcare workers

4-On demand of anxious parents after one-to-one discussion.

Government of India's guidelines on influenza vaccination

Health Care Workers working in close proximity to influenza patients

- In casualty/ emergency,
- Department of identified hospitals treating Influenza cases,
- Health workers working in ICU and Isolation Wards,
- Laboratory personnel testing influenza samples
- Rapid Response Team members investigating outbreaks of Influenza.
- Drivers and staff of vehicles/ ambulance involved in transfer of Influenza patients.

The Importance of HAND WASHING

When to Wash

- Before eating
- Before, during and after food prep
- Before and after caring for someone who is ill
- After blowing your nose, coughing or sneezing
- After touching garbage
- After using the toilet

Global Handwashing Day
October 15

95% of people wash their hands improperly.

Hand washing reduces respiratory infections by 16%

Handwashing with soap could prevent about 1 out of every 3 episodes of diarrheal illnesses.

How to Wash

1. Wet hands with clean, running water.
2. Lather hands with soap. Cover the backs of hands, palms, under fingernails and between fingers.
3. Scrub hands for 20 seconds.
4. Rinse with clean, running water.
5. Dry hands with a clean towel or air dry.

- Sources:
- Centers for Disease Control and Prevention, Journal of Environmental Health

THANK YOU.....