ISSN 0975-0894

The Child and newborn

The Journal of West Bengal Academy of Pediatrics

RNI Registration No. : RNI/68911/97

West Bengal
Academy of Pediatrics

Volume 25 No.1 & 4, January – December 2021

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ISSN 0975-0894 RNI Registration No.:RNI/68911/97

West Bengal
Academy of Pediatrics

Vol.25, No.1 - 4, January - December 2021

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Editorial

Till 2019 people hardly considered Coronavirus a threat to the mankind. The Coronavirus family comprises of two subfamilies, Coronavirus and Torovirus. The Coronavirus subfamily is divided into four genera, Alpha, Beta, Gamma and Delta. Human Coronaviruses (HCoV) belong to alpha and beta genera. Corona virus causes a wide variety of disease in various animal species. The first HCoV isolation was reported in 1965. It is known to cause innocuous respiratory infections and occasional viral diarrhea in human. The first epidemic of HCoV, Severe Acute Respiratory Syndrome (SARS) was reported in 2002. Pandemic caused by SARS - CoV-2 (a beta corona virus) is a third spill over in last 2 decades of an animal corona virus to humans.

The SARS-CoV-2 virus utilizes Angiotensin Converting Enzyme 2 (ACE2) receptors as its cell surface receptor. SARS 2002-3 virus also acted by similar mechanism. Multiple reports have demonstrated that children and young adults have a milder form of the disease compared to adults. Asymptomatic, mild and moderate infections comprise over 90% of all children who have tested positive for COVID-19. The probable reasons for lesser affection and milder infections in children and young adults include lower exposure to virions, being isolated at home and minimal exposure to pollution and cigarette smoke contributing to healthier respiratory tracts. Viral co-infection may be important in potentially leading to limited replication of the SARS-CoV-2 by direct virus-to-virus interaction and competition. Additionally, the distribution, maturation and functioning of viral receptors such as ACE2 may be important in age dependent susceptibility to severe COVID-19 infection. Another proposed theory is the protective role of Bacillus Calmette-Guerin (BCG) vaccine in COVID-19. BCG vaccination has been associated with heterologous immunity to other pathogens, by a phenomenon called 'trained immunity' involving innate cells such as macrophages, monocytes and epithelia.

Severe COVID-19 disease is characterized by three phases. The first phase being the viral phase; the second being the cytokine storm; and the third phase presents with acute respiratory distress syndrome (ARDS), impaired cardiac function and may lead to death. The cytokine storm appears to be driven by a dysregulated host immune response and might contribute to mortality. The profile of the cytokine storm associated with severe COVID-19 disease is similar to that of secondary hemophagocytic lymphohistiocytosis (HLH), which is a rare complication of other viral infections. Children with severe infection may also have abnormal coagulation parameters, perhaps related to high expression of ACE2 receptors in vascular endothelial cells.

On the other hand Multisystem inflammatory syndrome in children (MIS-C) also called Pediatric multi-system inflammatory syndrome (PIMS) is a newly recognized temporally related delayed manifestation of florid or unapparent COVID-19infection. It is characterized by persistent

fever, increased inflammatory markers and multisystem organ involvement. MIS-C usually present with Kawasaki disease like or toxic shock-like symptoms. Both MIS-C and Kawasaki disease share similarities regarding mucocutaneous inflammation. Maculopapular rash, conjunctivitis, and cheilitis are the most common clinical mucocutaneous features found in children with MIS-C.

In response to the COVID-19 outbreak, the Government ordered a nationwide school closure as an emergency measure to prevent spreading of the infection. For all practical purposes this decision was inevitable. Massive efforts were made by schools and teachers at all levels to create online courses and deliver them through different platforms as virtual classes. This novel approach was done in record time. By and large it was well organized. These actions definitely helped to alleviate many parents' concerns about their children's educational attainment by ensuring that school learning is moderately undisrupted. But there are reasons to be concerned because prolonged school closure and home confinement during a disease outbreak might have negative effects on children's physical and mental health. Evidence suggests that when children are out of school for some time, like winter and summer holidays, they tend to be physically less active, have much longer screen time, irregular sleep patterns, and less favorable diets. This results in excess weight gain and a loss of physical and cardiorespiratory fitness. Such negative effects on health are likely to be worse when children are confined to their homes without outdoor activities and interaction with peer group during the outbreak.

Perhaps a more important but often neglected issue is the psychological impact on children and adolescents. Even more problematic and enduring effects on children and adolescents are the stressors like prolonged duration of confinement, fears of infection, frustration and boredom, inadequate information, lack of in-person contact with classmates, friends, and teachers, lack of personal space at home, and even family financial loss can have a negative effect on them.

Schools have a critical role in these situations. Their responsibility is not only to deliver educational materials to children, but in offering an opportunity for students to interact with teachers and obtain proper psychological counseling. Schools can actively promote a health-conscious schedule also. Good personal hygiene, structured physical activities, appropriate diet, appropriate and good sleep habit. Such health promotion materials may be integrated into the school curriculum. In addition to innovative courses for a better learning experience, promotional videos can be useful to motivate children to have a healthy lifestyle at home by increasing physical activities, having a balanced diet, regular sleep pattern, and good personal hygiene.

Children have hardly any voice to advocate for their needs. The latest Commission on the future of the world's children urges a holistic strategy in preparing for the uncertainty that all children are facing. It is the responsibility of all stakeholders, from governments to teachers and parents, to ensure that the physical and mental impacts of the COVID-19 pandemic on children and adolescents are kept minimal.

Dr Jaydeep Choudhury
Editor-in-Chief

Multisystem Inflammatory Syndrome in Children (MIS-C)

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Introduction

Multisystem Inflammatory Syndrome in Children (MIC-C) is a new phenomenon in COVID -19 infection in children which was first reported from UK[1]. COVID 19 till date has caused less severe disease manifestations in children compared to its adult population. But as the pandemic progressed severe manifestation of COVID-19 infection though rare became evident in the form of Pediatric ARDS and different form of multi-organ dysfunctions due dis-regulated immune system. Researchers form UK first noticed some form of hyper inflammatory syndrome mimicking streptococcal toxic shock syndrome in children with evidence previous COVID-19 children. Some of them had manifestation quite similar to Kawasaki Disease. They named these condition as ''Pediatric Inflammatory Multisystem Syndrome-temporally associated with SARS-CoV-2" (PIMS-TS). Later similar disease manifestations were reported from different part of the word and WHO came forward with universally acceptable definition and named the condition as Multisystem Inflammatory Syndrome in Children (MIS-C). Clinical reports of this condition has been published from different countries like UK[2], USA[3], Italy[4], France and Switzerland[5].

Demography

According to the CDC report October 2020[6], 1097 confirmed cases and 20 deaths has been reported from USA due to MIS-C. 98% of them were tested positive for COVID 19 antibodies. Mean age was 8 yers and Male to female ratio 54: 46. MISC has been reported from different part of the world even from India. A report from India also described 23 cases of MIS-C with median age of 7.2 yrs[7]. Though some reports have shown acute COVID 19 coursing serious illness in infant age group[8] but MIS-C usually involves older children.

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Pathophysiology

MIS-C is hyper inflammatory syndrome associated with COVID 19 infection by epidemiological evidence. Different countries reported cluster of similar disease after being heavily affected by COVID 19. Interestingly cluster of cases lag behind the pick of COVID-19 infection. Majority of cases were positive for COVID antibody rather than RT-PCR indicating syndrome may be post infectious rather than related to acute infection. Exact pathogenesis of MISC still unknown but there are different hypothesis to explain this condition.

Immune dysregulation hypothesis:

Early infection activates macrophages followed by T-helper cell stimulation leading to release of cytokines. In addition, B-cells and plasma cells are activated leading to antibody production resulting in a hyper-immune state (phase III). That is why most of the children suffering from MIS-C are RT-PCR negative.

Molecular mimicry:

Some studies have been reported auto antibody against self immune and endothelial cells in children with MIS-C[9]. Probable explanation is that T cell recognizing self antigens and producing auto antibodies and circulating immune complexes.

Neutrophil extracellular traps (NETs) involvement:

Viruses stimulate the formation of NETs whose main function is to entrap virus. This virus induced NETs are known to elicit uncontrolled immunological and inflammatory reactions resulting in hyper-

inflammatory state.

Genetic basis:

There are also evidences which imply that genes also a play role in MIS-C and some racial groups like Africans have strong association with MISC[10].

Definition

WHO developed a preliminary case definition for MISC depending on clinical and laboratory picture of the affected children reported till date[11].

Diagnostic Criteria for MIS-C

All 6 criteria must be met

1. Age 0- 19 years
2. Fever for 3 days
3. Clinical signs of multisystem involvement (at least 2 of the following):

Rash, bilateral nonpurulent conjunctivitis, or mucocutaneous inflammation signs (oral, hands, or feet)
- Hypotension or shock
- Cardiac dysfunction, pericarditis, valvulitis , or coronary abnormalities (including echocardiography findings or elevated troponin/ BNP)
- Evidence of coagulopathy (prolonged PT or APTT; elevated D-dimer)
- Acute gastrointestinal symptoms (diarrhoea, vomiting, or abdominal pain)

4. Elevated markers of inflammation (e.g, ESR, CRP, or procalcitonin)
5. No other obvious microbial cause of inflammation.
6. Evidence of SARS-CoV-2 infection(Any one of the following)

– Positive SARS-CoV-2 RT-PCR

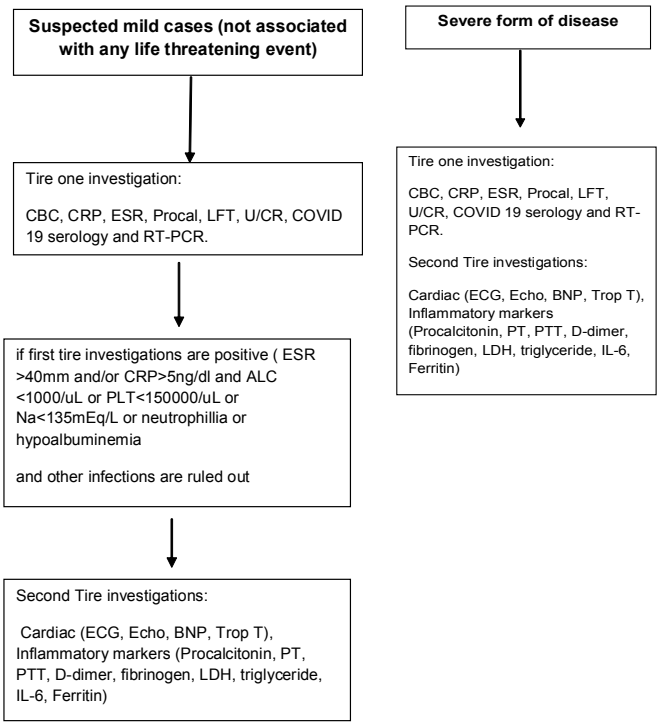
Positive serology(SARS CoV-2 IgG positive)
– Positive antigen test

– Contact with an individual with COVID-19

Clinical features

MIS-C can be self limiting febrile illness to severe multisystem involved disease. Initially it was described as disease mimicking Kawasaki disease, streptococcal septic shock syndrome or macrophage activation syndrome. At least 72 hrs fever is essential to suspect MIS-C. it can be associated with GI symptoms like diarrhea, vomiting abdominal pain, rash, conjunctivitis, musculoskeletal symptoms, hypotension, shock, bleeding. Before making diagnosis of MISC one should exclude bacterial septicemia and locally prevalent tropical fevers like dengue, malaria and scrub typhus.

Investigations



If in initial echocardiography myocardial dysfunction or coronary dilatation detected then repeat Echocardiography indicated every 2-3 days until myocardial dysfunction or coronary artery size stabilizes, then at 2 weeks and again at 6-8 weeks.

Classification of MIS-C and their treatment[12]

A. MIS-C with life threatening manifestation/ MISC with MODS/MISC with shock:

Presentation

It may present with shock and signs of other organ dysfunction, coagulation profile may be deranged, echocardiography may show cardiac dysfunction, chest X-Ray may show pneumonic patch.

Treatment

Appropriate measures to be taken to resuscitate the patient and patient should be shifted to PICU and supportive measures like bolus, inotropes and

ventilation should be provided as required. Broad spectrum antibiotics should be started.

Intravenous immunoglobulin – 2gm/kg (maximum 100gm) should be given over 8-12 hrs. In case of myocardial dysfunction more slower administration may be required to avoid fluid over load.

Methylprednisolone – 10-30mg/kg/day should be given IV for 3-5 days and it should be followed by oral prednisolone 2mg/kg/day and to be tapered over 4 weeks.

Though treatment failure is rare but in refractory cases a second dose of IV Ig and/or Biologics to be considered as rescue therapy [Tocilizumab 4 to 8 mg/kg/dose or Infliximab 5mg/kg IV or Anakinra2-10 mg/kg/dose (max 100 mg/dose) SQ/IV q6-12h]

Aspirin 3-5 mg/kg(max 75mg) to be given for 8weeks but contraindicated in case of bleeding or platelet count <80,000.

Low molecular weight heparin (LMWH) in the form of Enoxaparin 1mg/kg twice daily to be given by subcutaneous route if there is any documented thrombotic event or left ventricular ejection fraction is <35% or d-dimer level >2000ng/ml (it should be individualized as evidence is not that strong).

B. MIS-C with predominant Kawasaki disease like feature:

Presentation:

Predominantly presents with mucositis, conjunctivitis and cutaneous features and fulfils diagnostic criteria of complete or incomplete Kawasaki disease. Usually does not associate with any life threatening event. ECHO may include mild Left ventricular dysfunction and coronary artery dilatation (>=2.5SD).

Treatment:

Intravenous Immunoglobulin should be given 2gm/kg(max 100 gm) over 8-12 hrs.

Low dose methyl prednisolone 1-2mg/kg /day for 3-5 days followed by oral prednisolone 2mg/kg /day should be started and will be tapered over 2weeks duration.

Aspirin 3-5 mg/kg(max 75mg) to be given for 8weeks but contraindicated in case of bleeding or platelet count <80,000.

LMWH in the form of Enoxaparin 1mg/kg twice daily to be given by subcutaneous route if there is any documented thrombotic event or if there is any giant aneurysm of coronary artery (Z score >10).

C. Mild MIS-C or febrile inflammatory state:

Presentation:

Patient will be febrile for more than 72 hrs and will fulfill other criteria of MIS-C but will not have any shock, organ dysfunction or life threatening event. Patient will not fulfill criteria of Kawasaki disease. Inflammatory markers will be elevated.

Treatment :

As there is no life threatening event so we can weight for another 48 hrs to fever to subside. In between we can exclude other prevalent infections. But if high grade fever persists and inflammatory markers are still high after 48 hrs (five days from the onset of fever) we have to start therapy.

First line treatment will be low dose methyl prednisolone 1-2mg/kg /day for 3-5 days followed by oral prednisolone 2mg/kg /day should be started and will be tapered over 2weeks duration.

If patient does not responds with first line treatment within 48 hrs then we have to start second line therapy in the form of Intravenous Immunoglobulin 2gm/kg.

Low dose aspirin can be given only if there are any coronary artery changes and LMWH will be used only in case of documented thrombotic event.

Discussion

Understanding of pathophysiology of MISC is portent for formulation effective management and prevention strategies. Studies involving measurement of cell-mediated and cytokine mediated immune response are needed to determine actual pathogenesis. It can also determine usefulness of different drugs like tocilizumab and anakinra in the treatment of severe form of MIS-C. Most of recent published case series of MIS-C patients documented increased IL-6 levels[13,14,15]. A case series from France and Switzerland reported use of tocillizumab and anakinra along with conventional immune modulators used in MIS-C[16]. theoretical risk of MISC is also associated with COVID-19 vaccine as it is a immune mediated phenomenon. Till now

COVID-19 vaccine are not widely used in children and adolescents. Effect of immunization on the circulating different strains of COVID-19 may be another concern. Heterotypic immunologic responses may result in severe immunologic manifestations after subsequent infection as similar thing we have seen in case of dengue vaccine. India is in the verge of introducing vaccination drive for the children and adolescent and we have to keep a close eye on incidence and characteristics of MISC as the vaccination drive progresses. It is high time to maintain a national registry for MIS-C as it will help in future research to identify vulnerable population and determine preventive strategies.

Conclusion

COVID 19 is a new disease and we have very little knowledge about it. As till now children are not immunized they may the most valuable target for the upcoming waves of these pandemic. MIS-C is the one of newest complication of COVID-19 infection. As our knowledge is not sufficient new research initiatives have to be taken to evaluate different aspects of MISC and that may help us to formulate more

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India Heading Towards COVID Vaccine for Under 18

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Exactly 100 years after 1920 Spanish Flu pandemic where 5 crore people died, history repeated itself with 2019 Covid pandemic taking approximately 45.5 Lacs lives globally as of now (17th Oct.2021).Over the span of one century a lot has been advanced in the field of medical science and vaccinology towards prevention of infectious disease. Nevertheless it seems before almighty even today having sky high e-knowledge, molecular biotechnology, online treatment all are powerless before a invisible enemy.

After first wave (by alfa strain) we understood little bit about this Covid 19 but our staggered exit and relaxation at the end brought a volcanic disaster by second wave (delta strain). Kumbhmela, election, marriage, birthday party, sports events, conference, rally….all those were the root cause of second wave explosion. The experience from this changed our behavior, attitude, work culture towards new normal. Thus showing our preparedness to face third wave blast.

Its our Covid appropriate behavior (CAB), mass vaccination, SMS (social distancing, mask, sanitization) particularly using mask once we are stepping out from home till we return back will decide about amplitude of third wave. In our country Covid vaccination started from 16th January 2021 with adeno viral vector Covishield vaccine followed by Bharat Biotech Covaxin. Since then round about 97.65 crore (heading towards record number 100 crore) doses administered in India so far. "Announcements will be made at seaports, railway stations, bus stops, metro stations and airports at the time INDIA completes 100 crore jabs", Union Health Minister said.

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About 30% adult population got two doses and 73% got single dose. By this 2021 end Govt.of India is planning to inoculate 2 doses to all beneficiaries above 18 years. If on daily basis its possible to give 1crore doses then only we can achieve our goal. In a country with 139 crore population this mass vaccination is a big challenge. But our vaccination drive is largest in the world and its effort is definitely praiseworthy.

Director of PGIMER, Chandigarh Professor Jagat Ram on 13 september said that india is at the beginning of covid third wave. "A serosurvey conducted by PGIMER, among 2700 children shows 71% of them have developed antibodies. It shows children won't be affected disproportionately during the third wave," he said[1].

Despite a decrease in new infections Indian health experts are warning of a likely third wave during winter months. Approximately 86% of new infections have been reported from just 5 states: Kerala, Maharastra, TN, AP and WB. Waning alertness combined with festive season could create the ideal breeding ground for third wave[2].

Because under 18 are not vaccinated till now, that's why this group becomes more vulnerable towards Covid infection during third wave. Under10, 3%, from 10 to under18, 8% almost same percentage children got infected in both first and second wave. But unless and until a new mutation happens the chance of predominantly kids getting infected seems to be theoretical. Contrary to this for last 20-25 days we are getting 14-20% of covid positive cases among under18 in high covid scoring states in India. Now the time has come for kids vaccination.

ZyCov-D

Zydus Cadila has received approval for EUA from

DCGI for ZyCov D on 20 August 2021, the World's first and India's indigenously developed DNA vaccine and it will be administered to people 12 years and above. Ahmedabad based Zydus Cadila has conducted the largest clinical trial (28,000 volunteers) for the vaccine in india in over 50 centers so far.Phase III clinical trial showed primary efficacy of 66.6% for symptomatic RTPCR positive cases. ZyCov D is to be administered intradermally using a needle free injector as 3 doses (0, 1, 2 mo)[3]. The biggest hurdle with this vaccine is its slow production. ZyCoV-D is going to be introduced in the country's Covid vaccination programme. It is planned to roll over soon in 12-18 age group.

Covaxin

The Subject Expert Committee(SEC) on Covid-19 has granted emergency use approval to Bharat Biotech's Covaxin for children in the 2-18 years age group on 12 october 2021. Hyderabad-based Bharat Biotech had completed Phase-2 and Phase-3 trials of Covaxin on children under 18 in September and submitted the trial data to the DCGI at the start of this month.

"After detailed deliberation, the committee recommended for grant of market authorization of the vaccine for the age group of 2 to 18 years for restricted use in emergency situation," the subject expert panel said in a statement.

The made in India vaccine will be administered in two doses, with a gap of 20 days between the first and second dose[4].

This represents one of the first approvals worldwide for Covid-19 vaccines for the 2-18 age group. This vaccination is likely to happen through pre-filled syringes for better dose accuracy..

According to the experts involved in the clinical trials, the safety and efficacy of Covaxin in the paediatric age group has been found to be almost similar to that in adults, which was 77.8%, as per the phase 3 clinical trials data on adults.

Covovax

The phase 2/3 trials of the Covovax COVID-19 vaccine in children between ages 7 and 11 began in Pune's Bharati Vidyapeeth Medical College and Hospital. Covovax is the Indian version of the Novovax vaccine brought in India by the Serum Institute of India for children.

Corbevax

Vaccine candidate BE Corbevax have recently got approval for clinical trials for children.

Covid vaccination for kids may start in full fledge only by March 2022 even as 3-4 vaccines are likely to be approved for under18 by December, Government official sources said. Many vaccine are in the race of trials in India like Pfizer, Astra Zeneca and Moderna. These vaccines have been tested on children below 18.The efficacy of some of covid 19 vaccine for children is out as 100%.

Global status of Covid vaccine for kids

In May 2021, US and Canada regulators were the first to approve either Pfizer or Moderna jab for use in children from 12 year and older.The rollout started immediately at sites across the US with 2 doses given 3 weeks apart. Pfizer has already started testing its covid vaccine on 5-11 year age group followed by 6mo to 4 year children.

In UK children between 12 to 15 are set to be able to get a doses, following youngsters receiving one dose of Pfizer vaccine. After European Medicines Agency (EMA) approval Denmark, Spain, France, Germany, Sweden, Norway and many more EU countries have moved at different speeds.

In June, China began to allow some children from 3-17 year to be offered Sinovac, making it the 1st country to approve a jab for such a young age group.

Israel expanded its vaccination drive to 12-15 year in June.

UAE, Dubai started offering Pfizer vaccine to 12-15 year in mid may.

Singapore opened up its vaccination to adolescents 12-18 from June 1.

Japan on 28 May approved the use of Pfizer vaccine for those aged 12 years and above.

Philippines on May 26 decided to allow Pfizer vaccine for emergency use in children aged 12-15.

Covid vaccine prompts strong immune response in younger children (5 -11), Pfizer says 20.09.2021. Pfizer vaccine has been shown to be safe and highly effective in young children. The news sets the stage for EUA before the end of October. Results from

Moderna vaccine trial in children under 12 are also expected around that time.

Indian status of COVID vaccine for kids

In India 44 crore people belongs to age under 18, where as 12 crore between 12 – 17. India seems to be not prepared well for covid vaccination among under 18. Govt. plans covid vaccines for kids aged 12-17.

Around 20-30 lakh children with comorbidities will be eligible for the first round of vaccination, according to Govt. estimates.

Currently only those who are 18 or above are eligible for Covid vaccination. Union health minister Mansukh Mandaviya had told that covid vaccination for children will start soon.

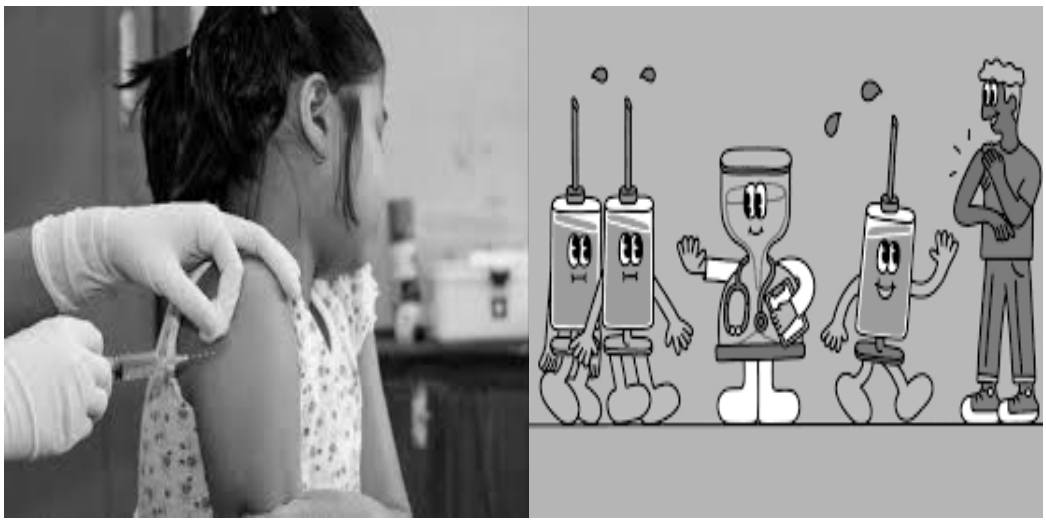
Dr. NK Arora, Chairman of the COVID-9 working group of NTAGI said on 19.09.2021 that vaccination of children under12 is expected to begin in the first quarter of next year. The results of vaccine trial for the younger children are expected by November.Out of 12 crore children between 12 -17 we will initially target children with comorbidities. The country will begin vaccinating healthy children once the 94 crore adult population has been vaccinated.

At last all children should have got at least single dose. Then only we can go for school reopening, sports activities, dancing, singing, group learning etc. with minimal risk. But it is not feasible in present scenario.

The possibility of third wave will be driven by complacency and lowering of guard by the community on one side and faster vaccination on other side. Though 70% of our population is exposed to covid virus, 30% is still unexposed and we still need to be careful.

Until high levels of global vaccine mediated protection are achieved across the world , it could be catastrophic if measures such as mask wearing, physical distancing and hand hygiene are relaxed prematurely. Countries, communities and individuals must be prepared to face this new phase of the pandemic.

More realistic epidemiological end point might arrive not when herd immunity is achieved but when covid19 can be managed as an endemic disease. The biggest overall risk would likely then be the emergence of a significant new variant of concern. As the immunity wanes gradually there will be need of annual booster dose of covid vaccine……But the time will say the ground reality. Till then stay safe. Take care of yourself, family and at last community as well.



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Iron Deficiency States in Children: A Review

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Abstract: *Iron deficiency states and iron deficiency anemia (IDA) are not synonymous. IDA is the most severe form of iron deficiency. Iron deficiency can affect cognitive function, immune function and work capacity of an individual. The cognitive impairment caused by deficiency of iron in infancy is not reversed by supplementation of iron later. Hence, the identification and treatment of iron deficiency is essential. Anemia, or hemoglobin, is used as a screening tool for iron deficiency in the population. Though it is a cheap and easy method, it does not differentiate IDA from other causes of anemia, like thalassemia. So, a complete hemogram and iron studies should be done wherever possible to further pinpoint the diagnosis. Anemia is defined based on hemoglobin < - 2 SD in different age groups from 6 months to 19 years. Anemia should be promptly treated with iron supplementations in correct doses and inhibitors of iron absorption should be taken care of. Hemoglobin levels should be reassessed after completion of treatment. Patients with severe anemia and no response to iron therapy should be investigated for other causes of anemia. Iron prophylaxis should be given to the masses starting from 6 months of age in order to take care of mild and moderate iron deficiency states without anemia. But a thorough clinical examination is essential before this, so that children with thalassemia are not given iron supplements. Thus, careful mass prophylaxis with iron and prompt treatment of IDA can help in reducing the burden of iron deficiency and its unwanted consequences.*

Introduction

The prevalence of anemia in children in the age group 6 – 36 months is 74%. About 50% of all anemia is IDA[1]. It is high time we realized that iron deficiency and IDA are not entirely synonymous. Rather, iron deficiency states are a continuum. In mild iron deficiency, the iron stores are mobilized to maintain a normal serum iron. So, only serum ferritin is decreased. All other indices of iron status are normal and there is no anemia. In moderate deficiency, the serum iron and serum transferrin saturation start decreasing. The total iron binding capacity increases. All tissues respond in a similar way to a decreased supply of iron. They express increased numbers of transferrin receptors. So, there is an increase in the serum transferrin receptors , one of the best indices of iron deficiency. Till this stage the hemoglobin remains unaffected. But, this mild to moderate iron deficiency progresses to IDA if left untreated. Thus, IDA (iron deficient erythropoiesis) is the most severe form of iron deficiency.

2/3 of the total body iron is utilized for hemoglobin production. 14% is used for other vital physiological functions. The remaining iron is deposited in bone marrow as hemosiderin and ferritin. Only a small amount of iron is in transit in plasma[2].

Iron deficiency can result in irreversible impairment of cognitive function[3]. In a study, it was found that school going children who received iron in infancy fared better in mathematics than those who didn't. Iron supplementation at the school going age did not result in any improvement of cognitive function[4]. Also, iron deficiency affects the immune system[5] and there is increased morbidity from infections[6]. This is because in the presence of iron deficiency, the leukocytes have an impaired ability to kill intracellular microbes[7] and the lymphocytes have an impaired ability to react to mitogens. Physical growth and work performance[8] are diminished.

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Iron has an important role in the synthesis of triiodothyronine (T3). Hence iron deficiency is implicated in the temperature dysregulation in hypothyroid children(9). DNA replication and repair are affected as these processes involve iron containing enzymes. Iron deficiency is apparently associated with an increased risk of heavy metal poisoning. Iron deficiency results in an increased gastrointestinal iron absorption. This response is not specific for iron, and other heavy metals, lead in particular, will also be absorbed. Hence the risk of lead poisoning may increase.

IDA in pregnancy increases the perinatal risk for mothers and infants and overall infant mortality[10]. Iron supplementation to pregnant women and ligation of umbilical cord after the pulsation stops can help in reducing iron deficiency in neonates. Exclusively breastfed full term infants usually have adequate iron stores, because though breast milk is relatively low in iron, its bioavailability is far better than cow's milk. Iron deficiency can set in after 6 months of age, with introduction of complementary food, if complementary food offered is poor in absorbable iron. The RDA of iron in the first 6 months is only 0.27 mg/ day, whereas after 6 months, at the time of introduction of complementary food, the RDA increases to 11mg/ day. The RDA of iron in adolescent girls is 15 mg/ day[1].

Iron deficiency states can occur due to nutritional deficiency, schistosomiasis, hookworm infestation, hemorrhage in childbirth, preterm delivery, adolescent females with inadequate iron intake (specially girls who have had their menarche) and Vitamin A deficiency, probably due to altered metabolism of iron .

Anemia is the commonest indicator used to screen for iron deficiency (Table 1). Anemia is defined as hemoglobin < -2 SD of the distribution mean in an otherwise normal population of same age and gender, residing at same altitude[11]. Since only hemoglobin is used for screening for iron deficiency, it must be kept in mind that there are plenty of causes of anemia other than iron deficiency like malaria, chronic infections, chronic hemolytic anemias, G6PD deficiency, childhood malignancies, which cannot be differentiated from IDA only on the basis of hemoglobin.

Table 1: Anemia defined by hemoglobin levels in different age groups[11]

Age Group	No Anaemia	Mild	Moderate	Severe
Child 6-59 month	≥ 11	10–10.9	7–9.9	<7
Child 5-11 year	≥ 11.5	11–11.4	8–10.9	<8
Child 12-14 year	≥ 12	11–11.9	8–10.9	<8
Non-genetic women (15 yrs and above)	≥ 12	11–11.9	8–10.9	<8
Pregnant women	≥ 11	10–10.9	7–9.9	<7

Free erythrocyte protoporphyrin (FEP) is elevated in IDA[12], but it is not a specific index of iron deficiency. It is also elevated in infection, chronic hemolytic anemia etc. Mentzer's index (MCV/ RBC count) is >13 in IDA and <13 in thalassemia, both of which are microcytic hypochromic anemias. Bone marrow staining for ferritin is a reliable index of iron deficiency, but it is not practical to perform an invasive investigation for population screening. Another retrograde method of diagnosis may be used in resource poor settings. In response to iron supplementation, if there is a rise in hemoglobin of at least 1 g/dl after 2 months of therapy, we can consider the subject to have been iron deficient prior to therapy[13]. But this method may occasionally miss a thalassemia trait who is iron deficient at the same time.

While approaching a young child with anemia, a good clinical examination along with a complete hemogram can give us a lot of information and help us in differentiating thalassemia from IDA. The hemoglobin will be low in both. The Mean Corpuscular Volume (MCV) and Mean Corpuscular Hemoglobin Concentration (MCHC) will be low in both. So, the causes of macrocytic and normocytic normochromic anemia can be ruled out. Pancytopenia is seen in aplastic anemia, abnormal cells are seen in leukemias, malaria parasite is seen in malaria. So these causes of anemia can be ruled out to some extent.

The red cell index that helps in differentiating between thalassemia and IDA is Red cell Distribution Width (RDW). It is elevated in IDA. Also, RBC count and reticulocyte count are elevated in thalassemia, but they are low in IDA. But CBC will not be able to differentiate between IDA and

thalassemia trait, and will also not be able to identify patients who have both.

Here comes the importance of iron studies. In IDA, serum ferritin, serum iron and serum transferrin saturation will be low, whereas, transferrin receptors and TIBC will be high. In thalassemia, serum ferritin will be high[14]. Ferritin is a very sensitive index of iron deficiency, but it is an acute phase reactant also. So, in the setting of infection or chronic inflammation, ferritin may be elevated, thereby masking iron deficiency. Hence, in patients without any evidence of infection, serum ferritin < 15 mcg/l is considered to be a marker of iron deficiency. Whereas, in a patient with infection, serum ferritin < 30 mcg/l is considered to be a marker of iron deficiency. Serum transferrin receptor is not affected by infection, and hence one of the best indicators of iron deficiency[15,16]. But, it is not widely available. To differentiate IDA from thalassemia trait, or to identify traits with iron deficiency, an HPLC will also be required. But HPLC in a very young child can raise confusion due to high levels of fetal hemoglobin. In such cases, HPLC of both the parents should be done.

For treatment of IDA, both oral and parenteral iron preparations may be used. In most cases, oral iron preparations are required. The ferrous salts are better absorbed than ferric. Ferrous ascorbate is most commonly used as ascorbate enhances the absorption of iron from the gastrointestinal tract. So bioavailability of iron in ferrous ascorbate is high (30 – 40%). Though ferrous ascorbate can cause teeth staining, it is less affected by inhibitors and its gastrointestinal adverse effects are less. Colloidal iron and iron polymaltose complex are also used but their bioavailability is less. The following oral and parenteral iron preparations are commonly available[1]:

Oral

(a) Ferrous salts (ascorbate, sulfate, fumarate, gluconate, succinate, aspartate)

(b) Ferric salts (hydroxide polymaltose complex, iron polysaccharide etc.)

Parenteral

(c) Iron dextran

(d) Iron sucrose complex

(e) Iron sodium gluconate complex

The absorption of iron from the gastrointestinal tract is affected by many factors . The factors which increase the bioavailability of iron (enhancers) are breastmilk (bioavailability of iron is 50%), animal source or heme iron eg. Fish, poultry, meat etc. (bioavailability 30%), and vitamin C. The factors which decrease the bioavailability of iron (inhibitors) are formula milk (bioavailability 4 – 6%), vegetable or non heme source of iron (bioavailability 10%), calcium, tannates, phytates. So, it is important to advice the patient not to take iron with milk or food. Also, patients must be counseled about dietary habits that maximize iron absorption eg. Tea or coffee must not be consumed with food.

In the age group 6 months to 5 years, and also 5 to 10 years, mild to moderate IDA is treated with supplementation of iron at the rate of 3 mg/kg/day for 2 months[11]. The child should be assessed every 14 days to look for the clinical signs of response, e.g. increased appetite, decreased irritability, increased activity. At the end of 2 months, hemoglobin is assessed again to see if it has increased to more than the threshold for that age group. If it has increased, prophylaxis should be instituted. If there is no rise in hemoglobin after 2 months, investigations should be done to identify and treat the cause of anemia. Severe anemia should be investigated thoroughly at the outset, and if necessary, blood transfusion should be done.

Table 2 : Treatment of iron deficiency anemia in 6 months – 5 years old children[11]

Mild Anemia (10-10.9 gm/dl)	3 mg of irox/kg/ day for 2 months	Follow-up every 14 days by ANM Hb estimation after completing 2 months of treatment to document Hb>11 gm/dl
Moderate Anemia (709.9 gm/dl)	3 mg of iron/Kg/ day for 2 months	Follow-up every 14 days by ANM Hb estimation after completing 2 months of treatment to document Hb>11 gm/dl

In the age group 10 – 19 years, mild to moderate IDA is treated with supplementation of elemental iron at the rate of 60 mg/day for 3 months[11]. Regular clinical assessment and hemoglobin estimation after 3 months should be done. Severe and unresponsive anemia should be investigated thoroughly.

As already stated, iron deficiency and IDA are not exactly synonymous. IDA is the severest form of

Table 3: Treatment of iron deficiency anemia in 5 – 10 years old children[11]

Level of Hb	Treatment	Follow-up
Mild Anaemia (11–11.4 gm/dl)	3 mg of iron/Kg/ day for 2 months	Follow-up every 14 days Hb estimation after completing 2 months of treatment to assess if Hb estimates are >11.5 gm/dl.
Moderate Anaemia (8–10.9 gm/dl)	3 mg of iron/Kg/ day for 2 months	Follow-up every 14 days Hb estimation after completing 2 months of treatment to assess if Hb estimates are >11.5 gm/dl.
Severe Anaemia (<8 gm/dl)	Refer urgently to DH/FRU	

Table 4: Treatment of iron deficiency anemia in 10–19 years old [11]

Level of Hb	Treatment	Follow-up
Mild Anaemia (11–11.9 gm/dl)	60 mg of elemental iron daily for 3 months	Follow-up every month Hb estimation after completing 3 months of treatment to assess if Hb estimates are >12 gm/dl.
Moderate Anaemia (8–10.9 gm/dl)	60 mg of elemental iron daily for 3 months	Investigate Follow-up every 14 days Hb estimation after completing 3 months of treatment to assess if Hb estimates are >12 gm/dl.
Severe Anaemia (<8 gm/dl)	Refer urgently to DH/FRU	Severely anaemic adolescents would be line listed by ANM

Table 5: Iron prophylaxis in the public sector[1]

Age group	Intervention/Dose	Regime
6–60 months	1 ml of IFA syrup containing 20 mg of elemental iron and 100 mcg of folic acid	Biweekly throughout the period 6–60 months of age and de-worming for children 12 months and above.
5–10 years	Tablets of 45 mg elemental iron and 400 mcg of folic acid	Weekly throughout the period 5–10 years of age and biannual de-worming
10–19 years	100 mg elemental iron and 500 mcg of folic acid	Weekly throughout the period 10–19 years of age and biannual de-worming
Pregnant and lactating women	100 mg elemental iron and 500 mcg of folic acid	1 tablet daily for 100 days, starting after the first trimester, at 14–16 weeks of gestation. To be repeated for 100 days post-partum.
Women in reproductive age (WRA) group	100 mg elemental iron and 500 mcg of folic acid	Weekly throughout the reproductive period

Table 5: Iron prophylaxis[1]

AGE	Elemental iron	FROM -TO
Full term (1)	1 mg/kg/day	4-6 months
Preterm	2-4 mg/kg/day	2 weeks-1 yr
1-3 yrs	7 mg /day	
4- 8 yrs	10 mg/day	
9-13 yrs	8 mg/day (2)	
Adolescent children	20 mg /day	weekly

iron deficiency. In fact, individuals with mild to moderate iron deficiency will not exhibit pallor. But, if iron supplementation is done in these individuals, then progress to IDA and its adverse effects on health can be averted. Here lies the importance of iron prophylaxis in children, pregnant women and lactating mothers. Also, children with undiagnosed congenital cyanotic heart diseases, e.g. Tetralogy of Fallot, have high iron demand and will be benefited by this mass prophylaxis. Iron prophylaxis in the public sector is different from the private sector as the iron preparations available in these two sectors are different in composition. The following chart describes iron prophylaxis in the public sector[11].

Full term exclusively breastfed infants have good iron stores in their bodies and do not require iron prophylaxis. But they need iron prophylaxis from 6 months of age when complementary feeding is introduced. Preterm infants need iron prophylaxis from 2 weeks of age[1].

The only disadvantage of giving iron prophylaxis to the masses is that thalassemia patients may inadvertently be given iron, leading to iron overload. Also patients with latent infections or states of chronic inflammation e.g. obesity may endure harm from iron supplementation. So, a thorough clinical examination should be done before starting iron prophylaxis.

The common adverse effects of iron preparations are gastrointestinal adverse effects, like epigastric pain, nausea, vomiting, diarrhea, constipation. Black stool is a common occurrence which raises alarm among parents, but it is not harmful and parents must be told that this should not be a reason for

stopping iron preparations. High dose ascorbic acid, if taken with iron, may cause epigastric pain.

All iron preparations reduce the absorption of tetracyclines, trimethoprim, sulphonamides. So care must be taken while prescribing these drugs.

Parenteral iron is indicated in severe iron deficiency with chronic bleeding, intolerance to oral iron, malabsorption or inflammatory bowel disease, or patients on erythropoietin therapy. Sensitivity test must be done before administration of parenteral iron, as hypersensitivity reactions are not uncommon[1].

Other health measures along with iron prophylaxis and treatment of IDA that help in reducing the prevalence of iron deficiency include malaria prophylaxis, hookworm control, immunization, environmental health measures, fortification of food and community based primary health care.

Conclusion

Iron prophylaxis in the general population is essential for preventing the progress of mild to moderate iron deficiency states to IDA, and also to reduce cognitive impairment and morbidity from infection due to iron deficiency. But, this should be coupled with regular health checkups so that children likely to endure harm from iron supplementation can be identified, and iron prophylaxis can be avoided in them. Anemia based on hemoglobin levels is an excellent tool for population screening of iron deficiency, and those found to be anemic should be given iron supplements in therapeutic doses. But, these patients again should be regularly followed up for adverse reactions and response to therapy. Those not responding to iron therapy or those with severe anemia should be thoroughly investigated to rule out other causes of anemia. Complete hemogram and iron study should be done wherever possible for better diagnosis and management.

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Screen Time in Children and the Advent of Online Classes – The Pediatrician's Perspective

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Abstract : *The last decade has seen a tremendous upheaval in usage of digital media by children, and the repercussions of this are manifold. The COVID-19 pandemic has also resulted in digitalisation of education to a significant degree, resulting in increased digital screen exposure among children. The UNICEF, WHO and IAP have formulated their individual action plans to curtail the effects of digital media exposure. Clinicians need to be aware of these guidelines and communicate them effectively to the parents.*

Key words : *Screen time, Online classes, Smartphone, Television, Sedentary behavior*

Screen time or digital screen exposure is defined as the time spent by an individual in utilisation of digital media like television, smart phone, computers and tablets etc[1]. The last decade has seen a tremendous upheaval in usage of digital media by children, and the repercussions of this are manifold. The COVID-19 pandemic has also resulted in digitalisation of education to a significant degree, resulting in increased digital screen exposure among children. A longitudinal study reported that there was a rapid increase in TV-time by one year of age, and children who had screen time <1 hour per day at 14 months of age, started watching screens for >2 hours/day at 30 months age[2].

Screen time does not have solely negative impact on the development of children. Studies show that by 15 months, children can learn words from digital media but have difficulty using them in real world. By 2 years of age, children can learn new words via video chatting.

In case of pre-schoolers, cognitive, social and literacy skills can improve from well-designed television shows. However, higher-order thinking skills and executive functions (task persistence, impulse control, emotion regulation, and creative, flexible thinking) are best taught through unstructured and social (not digital) play, and parent child interactions.

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Prevalence of high digital media exposure

Prevalence of excess screen time ranges between 10% to 93.7% across the high-income countries, and 21% to 98% in the middle income countries[1]. The maximum duration of screen time is observed between the ages of 3-5 years in home based care[3,4]. Amongst the school-aged children and adolescents, television was still the most widely used digital media, and the children clocked an average of 2 hrs/day on cumulative digital exposure. Around 70 percent adolescents used social media applications.

There is a dearth of Indian studies on digital media exposure, but a cross-sectional study shows screen time >1 hour/day in 88.7% and >2 hours/day in 56.5% children between the ages of 15-18 months[5]. Most (72%) parents were not concerned with their child's screen time[5].

Factors associated with increased screen time

Multiple factors contribute to increased screen times in children which can be broadly categorized as per Figure 1.

Child related factors :

Increased age[6], hyperactivity-inattention[7], daily sleep duration, sedentary preferences and male sex were found to be more positively associated with increased

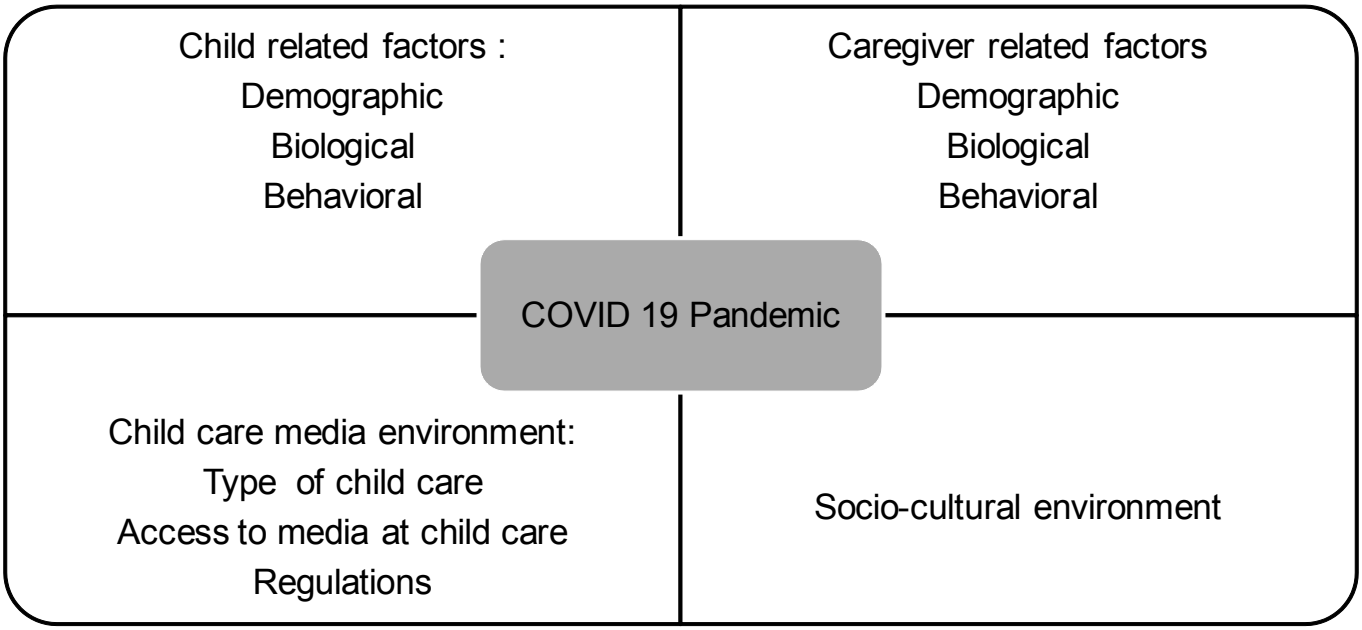

Fig 1: The contributory factors to increased digital media exposure in children

screen times.

Caregiver related factors:

Paternal age, time spent on work by mothers, maternal stress levels , parental screen and shorter breastfeeding were positively related to child's screen time[1]. Children of primi mothers had increased in screen time. Parental income, parent's occupation, socioeconomic status of the family, maternal ethnicity, low maternal body mass index , decreased level of education and physical activity of the parents'/ caregivers were negatively associated with average digital-media exposure of the child[1]. Another study shows high child BMI, maternal distress/depression, television viewing time of the mother and cognitive stimulation in the home environment were associated with screen media use[8]. Having at least one electronic device in toddlers' bedrooms compared to none, was associated with higher screen time among toddlers[9].

Child care media environment :

This includes both the digital micro as well as the macro environment. Micro environmental factors like the access to digital media gadgets, screen time rules at home, home-based care, parenting skills, computers placed outside the child's bedroom and TV on during dinner, infant crying duration and heavy TV use are associated with increased screen times[1].

The digital macro environmental factors include geographical conditions like increased screen time during the winter season, accessibility to media according to socioeconomic strata of the family and the prevalence of media usage in the particular location[1].

Socio cultural environment :

This broadly encompasses the cultural practices regarding media usage and with respect to the current scenario, digitalization of education and a subsequent increment in screen exposures amongst children.

Consequences of increased screen time

Consequences of increases digital exposure in children can be broadly subdivided into early and late effects (Figure 2) . Early effects are those seen in children less than 5 years while late effects deal with those seen in ages above[5].

In older children and adolescents additional problems include exposure to pornography and phishing sites, video game addiction, internet gaming disorders, cyber bullying and sexting, increased exposure to substance abuse and self injury and increased depression due to dissatisfaction as a result of increased social media usage, which may lead to body image problems and eating disorders.

Screen time recommendations

The AAP, UNICEF-WHO and IAP have recently developed their individual guidelines for regulation

Early	Late
■ BMI ■ Parent-child conflict ■ Poor cognitive development[10] ■ Poor eating habits[11] ■ Aggressive and antisocial[12] ■ Sedentary habit	■ Poor motor skill, language skills and cognition[10] ■ Sedentary habits[13] ■ Eating disorders and obesity[6] ■ Poor class room engagement ■ Victimisation ■ Disturbed sleep timing[13] ■ Non communicable diseases[14] ■ Hyperactive inattentive behavior ■ Emotional problems

Fig 2: Early and late consequences of increased screen time

of screen time in children.

The AAP guidelines[14] :

(a) Infants and toddlers experience "video deficit:" difficulty learning from 2-dimensional video representations at younger than 30 months of age.

(b) For children <18 months, avoid use of screen media other than video-chatting

(c) For children ages 2 to 5 years, limit screen use to 1 hour per day of high-quality programs. Parents should co-view media to judge its acceptability for child viewing.

(d) For children ages 6 and older, there should be consistent limits on the time spent using media, and the types of media.

WHO recommends no screen time for children less than 1 year of age, and for 2 years, sedentary screen time be limited to 1 hour/day. Children between 3-4 years of age should also be restricted to 1 hour/day of digital exposure[15].

WHO states16, in order to curtail the side effects of excessive media usage in children, parents should

(a) Empower children with knowledge and information

(b) Create a balance between online and offline activities.

(c) Set clear rules about screen time and how, when and where children can use the Internet.

(d) Communicate the boundaries and rules clearly with children.

(e) Install the latest software updates and antivirus programmes on device(s) used by children.

(f) Encourage children to be active.

School authorities should

(a) Develop or review and update online safety guidelines.

(b) Communicate online code of conduct with students

(c) Maintain and promote online counselling services for students.

Health care professionals should

(a) Disseminate information among families about the risk of excessive screen usage.

(b) Be vigilant about the possibility of excessive screen time or gaming during this period

(c) Provide online psychological support and counselling sessions for people with gaming and gambling disorders.

IAP has also formulated a guideline, based on the WHO recommendations as a part of the Parent Guideline modules[17].

The American Academy of Ophthalmology recommends[18] :

(a) 20-20-20 rule: every 20 minutes, look at least 20 feet away for 20 seconds.

(b) Alternate reading an e-book with a real book

(c) Pre-mark e-books with bookmarks every few chapters to remind the child to look up.

(d) After completing a level in a video game, look out the window for 20 seconds.

(e) Avoid using screens outside or in brightly lit areas.

(f) Adjust the brightness and contrast of the screen.

(g) Use good posture when using a screen.

(h) Encourage the child to hold digital media farther away: 18 to 24 inches is ideal.

(i) Remind them to blink when watching a screen.

The BLINK 20-20-20 is the recent addendum in the guidelines by the AAO with BLINK being an acronym for Blink, Lubricate, Inches away, Near Device Breaks, Know your sources[18].

Online classes – the new challenge

COVID-19 pandemic has vastly altered the mode of living around the world, and needless to say, has altered the way we teach our children as well. Digitalisation of education around the world has been the norm of teaching, with students learning via online classes and lectures. Online education is no more an option, but a necessity. Several studies have researched the accessibility and effect of online education on children and teachers alike (Figure 3), but studies on the health effects of online classes are still undergoing and are yet to be published.

Online classes, despite making education accessible to a vast majority of children isolated at home has the propensity of multiple effects. It increases the duration of digital media exposure in children by a huge extent, increases their sedentary habits and pave way for increment in the incidence ophthalmological problems like refractive errors and eye strain.

The Government of India has recently formed the Pragyata Guidelines[19] for regulation of online education in the country. The guidelines restrict the daily class duration to less than 30 mins for pre-primary classes, two sessions of 30-45 mins on decided days for classes 1-8 and four sessions of 30-45 mins on decided days for classes 9-12. Parent teacher interactions are to be organised once a week and online and offline curriculum must be appropriately balanced by the school authorities. The government has also introduced several digital education programs like PM e-VIDYA program (DIKSHA, SWAYAM , Prabha channel, On-air SHIKSHA VANI, DAISY for hearing and visually impaired, ITPAL fir IIT/JEE)

Conclusion

There is a dearth of adequate studies regarding screen time and its consequences on children in India, which calls for further research. As pediatricians, however, it is imperative that we start clinically judging the effects of digital media exposure amongst children in our clinics, especially between the ages of 1-5 years age, and provide the parents with detailed guidelines and support regarding media usage. The importance and the side effects of online classes, physical activity and sleep guidelines as advocated by the WHO[15] should be effectively communicated to them, to ensure the overall wellbeing of our children.

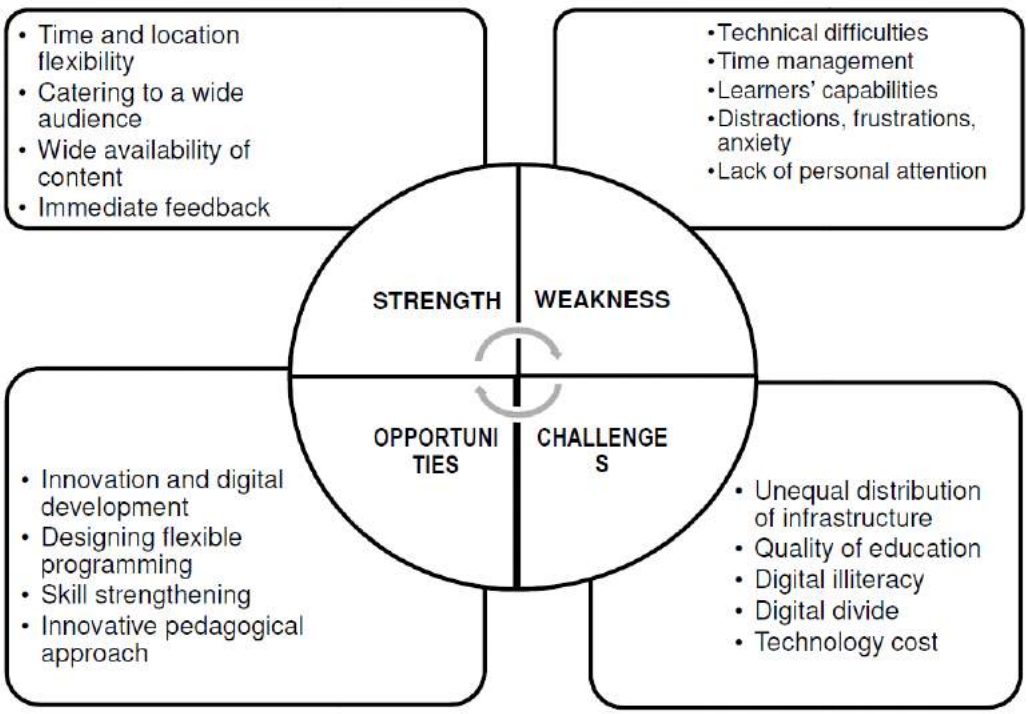

Fig 3: SPOC chart demonstrating the strength, weakness, oppurtunities and challenges of online classes[20]

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Attention-deficit/hyperactivity Disorder (ADHD)

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Pradeep (name changed), a 11 year old boy is brought to his pediatrician by his mother who is too bothered by the restlessness of the child. His mother tells that she is worried by the repeated reporting Pradeep's behavior from school by his teachers due to bothering other children in his classroom and poor academic performance. He is often breaking things at home and suffered a fracture in his right leg while riding his bicycle off the roof. She found out that a boy of similar age in their neighbourhood has been put to some medication for similar problems and is now able to concentrate on his work. Mother relates that Pradeep's father is against medication for the child, who believes that he is quite successful, even after his own behavioral troubles in school during his childhood. The pediatrician after detailed discussion and evaluation reported that the child may be suffering from ADHD which is quite common and treatable behavioural illness.

What is ADHD and how commonly it is seen?

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common neurobehavioral disorders that can profoundly affect children's various domains such as academic achievement, general well-being and social interactions, manifesting in early childhood with symptoms of hyperactivity, impulsivity, and/or inattention having a chronic course within a lifespan.[1]

In a meta-analysis of 175 studies in school going children across a wide geographical distribution, the estimated pooled prevalence of ADHD was 7.2 percent (95% CI 6.7 to 7.8).[2]

In India, the reported prevalence rates vary. A study done in 2013 from southern India reported prevalence of 11.3% in primary school children[3].

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Among children and adolescents with current ADHD, almost two-thirds were taking medication, and approximately half had received behavioral treatment of ADHD in the past year. Nearly one quarter had received neither type of treatment of ADHD[4].

Boys are more than twice as likely as girls to receive a diagnosis of ADHD[5], possibly because hyperactive behaviors, which are easily observable and potentially disruptive, are seen more frequently in boys. The majority of both boys and girls with ADHD also meet diagnostic criteria for another mental disorder[4]. In the 2015 to 2016 NHIS, the prevalence was 14 percent in boys and 6 percent in girls[6].

ADHD often co-exists with other comorbid conditions such as oppositional defiant disorder, conduct disorder, learning disability and anxiety, obsessive compulsive disorders, tic disorder, depression, autism spectrum disorder[7].

What are the factors involved in pathogenesis of ADHD?

Though the pathogenesis of ADHD is not definitively known yet some primary as well as secondary factors play a significant role in it.

Primary factors include – genetic factors, neuroanatomy and catecholamine metabolism. A genetic contribution to the pathogenesis of ADHD is supported by the increased risk of ADHD in first degree relatives with single nucleotide polymorphism and copy variant though they are not specific.

Structural brain imaging shows differences in anterior brain areas like smaller prefrontal cortical volumes, reduced thickness of anterior cingulate cortex, cortical thinning, etc. Whereas functional brain imaging of a group of children with ADHD have revealed reduced global activation, reduced local

activation of neurones in the area of basal ganglia and anterior frontal lobe.

Catecholamine metabolism including nor epinephrine and dopamine appears to have a role in evolution of ADHD. Patients have an increased in dopamine transporter density compared to normal healthy individuals and methylphenidate increases extracellular dopamine in brain[8].

Environmental factors play a secondary role in predisposing ADHD. They include certain food additives and allergens, sleep deficiency, prenatal exposure to tobacco and medications, psychosocial stressors, etc.[9]

What are the signs and symptoms of ADHD?

ADHD is characterised by two categories of core symptoms.Hyperactivity/ impulsivity and inattention. The DSM-5 criteria define 4 dimensions of ADHD:

1. Attention-deficit/hyperactivity disorder primarily of the inattentive presentation (ADHD/I)
2. Attention deficit/ hyperactivity disorder primarily of the hyperactive-impulsive presentation (ADHD/HI)
3. Attention deficit/ hyperactivity disorder combined presentation (ADHD/C).
4. ADHD other specified and unspecified ADHD.

The criteria of the symptoms required for diagnosis as described by DSM-5 criteria[10] are the following.

(a) Occurrence of symptoms before the age of 12 years

(b) Pervasive nature, symptoms present in 2 or more settings

(c) They should be persistent (for more than 6 months)

(d) For children <17 years, 6 symptoms of hyperactivity and impulsivity or 6 symptoms of inattention

(e) Causing clinically significant Impairment

(f) Unrelated to symptoms for other mental disorder

(g) Assessment for comorbid conditions that might coexist with ADHD

Hyperactivity/ impulsivity is described by excessive fidgetiness. The child faces difficulty remaining seated when required, inappropriate runs around or climbs and is always “on the go/ driven by motor”. There is difficulty in quiet play or engaging in leisure activities. Such type of children are always reprimanded for their excessive talking, difficulty in waiting turns, blurting out answers too quickly and interrupting others. These features are often observed by 4 years of age with a progressive course over next 3 to 4 years, peaking at 7 to 8 years and gradually declining and plateauing thereafter.

Compared to the hyperactive type, the signs of inattention appear late in childhood but are persistent in varying degrees throughout life. This may be due to late recognition of symptoms by the caregiver. The predominantly inattentive subtype of ADHD is characterized by reduced ability to focus attention and reduced speed of cognitive processing and responding, which could not be argued by manifestations of defiant behaviour. Teachers may report that the child fails to provide close attention to detail, performs careless mistakes, not listening or responding, even when directly addressed. Such children face difficulty in completing and organizing tasks, activities, and belongings and are often forgetful.

The disease has a steady course across the phases of life. For preschoolers behavioral disturbance and unintentional injuries are the major setbacks as per their age, while parents may feel themselves incompetent to manage such children. In school going age children manifest behavioral disturbance, aggressive tendencies, academic impairment and difficulty in peer interaction. As the young ones move towards adolescent age group new issues crop up. Non-fulfillment of academic potential clubbed with peer rejection pushes down their morale further. Lack of confidence and low self-esteem leads them to resort to substance abuse and antisocial behavior which is further reverberated in adulthood exhibited by relationship problems, occupational difficulties, injuries and accidents.

How to approach for diagnosing a case of ADHD?

A comprehensive assessment including detailed history, physical and mental status examination is needed to achieve a diagnosis and ascertain the significance of impairment of the disorder on both the patient and caregivers. Developmental, psychiatric, and medical disorders and psychosocial or environmental stressors such as family discord, parental substance abuse that are directly relevant to the management should be assessed as far as practicable.

The assessment of pre-schoolers, school going children or adolescents should be conducted through series of interviews of the patient, parents and extended family members and observations in different settings like that in home, school and within peer group.

Information from teachers must be obtained whether in structured format via rating scales or through unstructured teacher observation reports. Evaluation of the report cards, samples of schoolwork and a detailed summary of classroom behaviour and interventions to be sought for. While recording the history it should be remembered that the information gathered is mostly subjective to the respondents' perceptions hence multi informant technique seeking information across different domains of functioning of the child in multiple settings is the key. The significant aspects to be gathered while asking is outlined in Table 1.[11]

A comprehensive evaluation for ADHD requires complete physical examination, though mostly normal in almost all cases. It should include anthropometry, and vitals, vision and hearing evaluation, assessment of dysmorphic features and neuro-cutaneous abnormalities and a complete neurologic examination.

It is prudent to be equipped with age appropriate play and work materials for better interaction with the child and also engaging them in an age appropriate activity during which he or she can be observed for general intellectual capacity of the child, language and other spheres of development, verbal and nonverbal communication and behavioural abnormalities.

The evidence of over activity, impulsivity, and inattention may not be found during the time of interview as children may perform better when they find tasks interesting or the situation is structured with one to one adult interaction as in the clinic thus making the clinician prone to rule out the diagnosis.Rather observation for co-existing disorders including intellectual subnormality,child–parent interactions and parents' styles of responding to defiance must be given significance. Adolescents

Table 1. Outline of interview for comprehensive assessment and evaluation

Developmental and psychiatric history	For differential diagnosis and comorbidities
Medical history	Comorbidities and contraindications for medications' Comorbid physical disorders: tics, seizures, malnutrition, thyroid abnormalities; Contraindications: Syncope, exercise intolerance
Substance use history	
Family history	ADHD, other developmental, psychiatric, and physical disorders; family history of young, sudden cardiac deaths before drug prescription.
Treatment history	Past treatment type if any, drug dose, formulation, dosing schedule, adherence, response, side effects and perceptions, treatment adherence, premature termination
Personal history	Sleep patterns and appetite, educational evaluation
Psychosocial history	Environmental factors, perceptions and expectations regarding diagnosis and treatment, family psychopathology and family resources
Dysfunction: Domains involved (academic, family, social, interpersonal,legal, peer relationship, occupational)	Severity and impact of illness on the patient: secondary depression, perpetrator of bullying, or a bully victim, stigma, parental abuse (psychological, physical, and neglect). Impact of illness on the life of caregivers: burden, distress, burnout, psychiatric/psychological burden, stigma
Strengths and assessment of current needs	Physical, psychological, cognitive or creative attributes of thechild that can be considered as assets; e.g., good in a sport, coloring or sketching, obedient and respectful of elders

should be separately interviewed from their caregivers.

A mental status examination should include questions for revealing psychosocial stressors, emotional, medical, and developmental events that mayprovide an alternative explanation for the symptoms, a formal assessment of form and content of thought, presence of delusions, hallucinations, depressive cognitions, worries, ruminations, and obsessive symptoms as far as possible.

Rating scales may assist in evaluation of ADHD and recording and rating observations and interviews, which include Child Behaviour checklist, Conner's parent and teacher rating scales, Vanderbilt parent and teacher rating scales, and ADHD rating scale–fourthedition. The major setback is, they are not validated according to Indian standards. The only freely available tool (based on fourth edition of DSM) that can be used for diagnosis of ADHD in the Indian context is theINCLEN Diagnostic Tool for ADHD[12] though is time consuming and not superior to an information gathered via good clinical assessment.

Regular psychological testing is not required except for suspicion of coexisting intellectual disability. In case of specific learning disorder, the psychological assessment should be carried out after adequate control of ADHD symptoms.

Routine laboratory investigations are not indicated except for suspicion of any co-existing medical illness or to rule out medical differentials. Neuroimaging and electroencephalogram are also not a part of regular work-up and is necessary only for ruling out neurodevelopmental syndromes and metabolic disorders. Suspicion of substance abuse may require toxicology screens as well as genetic testing required for establishing the diagnosis of syndromic children. Table 2 shows differential diagnosis of ADHD.

Table 2: Differential Diagnosis of ADHD

Condition	Type
Developmental variations	Intellectual disability, Normal behaviour of children
Neurologic or developmental conditions	Learning disabilities, speech and language disorders, autism spectrum disorders, neurodevelopmental syndromes- Fetal alcohol syndrome, Fragile X Syndrome. Seizure disorder, CNS infections, trauma sequelae Metabolic disorders (adreno-leukodystrophy)
Emotional and behavioural conditions	Anxiety disorders Mood disorders Oppositional-defiant disorders/ Conduct disorders Obsessive compulsive disorders, Tic Disorders, Depression, PTSD
Psychosocial/environmental factors	Stressful home environment
Medical conditions	Hearing /visual impairment Lead poisoning Thyroid abnormalities Sleep disorders Drug-induced/substance abuse

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DMS-5) is adhered to for diagnosing ADHD which can be applied to children as young as 4 years of age and has been discussed already. The differential diagnosis for conditions mimicking ADHD symptoms are tabulated in table 2 and can be easily ruled out if the case is approached as described.

How to formulate a plan of management of ADHD?

With no permanent full cure of ADHD things that should be borne in mind at the onset of treatment are the age and developmental stage of the child, the significance of impairment of functioning of the child and the family members, the existing needs of the child which would be changing from time to time and goals targeted for therapy.

At the inception of management, the first foot forward will be from the end of the pediatrician to build up a therapeutic alliance with the child and his or her family, hearing their perceptions and concerns of the disease with a positive regard and assessing related predisposing factors. Communication of diagnosis should include the course of the disease, clearing misconceptions, alleviating anticipations of social stigma, treatment outcome along with side effects of therapy. Psychoeducation individualized to each child should be carried out with the family members in regular extended sessions.

Management of children with ADHD involves pharmacological and non-pharmacological measures including behavioural interventions, environmental and educational modifications. Young ones within 4 to 6 years of age are put for behavioural modification therapy, only resorted to medications if symptoms are uncontrolled. Drug therapy is preferred for older children aged 6 years and above with behavioural interventions act as add on modality.[7]

Behavioral interventions are modifications in the physical and social environment that are designed to change behavior by using rewards and non-punitive consequences. Parents can be trained the principles of **positive reinforcement** (rewarding on desired behavior), **time-out, response cost** (withdrawing rewards or privileges when unwanted or problem behavior occurs), **token economy** (a combination of positive reinforcement and response cost) that can be tried at home targeted at improving parent child relationship[13].

Environmental modifications, both at home and school are important for the children suffering from the disease to wriggle out of the symptoms. These include the following :

i. Formulation of a daily time schedule for the child with highlighters for improving attention
ii. Putting them always under adult supervision that would limit distractions and factors influencing negative behaviour.
iii. Providing specific logical places for keeping toys, schoolwork and clothes
iv. Setting small, reachable goals
v. Finding activities in which the child can be successful and engaging them in attention improving tasks and playing instrumental music.

Parents need to be persistent, patient and also flexible in allowing adequate playtime. They should be trained to give clear specific short instructions and cut down critical comments and maintaining a liaison with the teachers and school.

Educational adjustments include written assignments written on the board with charts, checklists and planners to keep the children "on task". Placing them nearer to the teacher and allowing extended time for task or test completion in a less distracted environment. Teachers should be mindful enough to send private signals whenever the child seems to be "off-task''. Short breaks are allowed with activities during breaks. A "Study Buddy" or "Shadow Teacher" can be assigned for giving extra required attention to these special ones in need. Teachers should be part of diagnosis and assessment of ongoing therapy using behaviour rating scales mentioned above and regular report card monitoring to look for any changes in the treatment plan required.[11]

Pharmacotherapy for ADHD should be tailored to fit individual youngsters according to their age, need for therapy and coexisting neuropsychiatric illnesses. Prior to starting drug treatment history of syncope, exercise intolerance, shortness of breath, chest pain, coexisting seizures or tics, sleeping habits, appetite and feeding patterns, sudden cardiac death in family and previously identified congenital heart disease should be sought for. Anthropometric measurements should be performed along with heart rate and blood pressure measurements. ECG and baseline liver enzyme levels should be done if at all indicated[14].

For a school-aged child or adolescent, a stimulant is the first-line agent, followed by amphetamines. Stimulants are preferred because of their rapid onset of action, safety and efficacy. Methylphenidate is the only stimulant available in India. Dosage titration is done according to improvement of core symptoms

gradually which is marked by appetite suppression, initial few weeks are required though for desired therapeutic effect. Frequency of medications depend on the type of symptoms and target set for the therapy. Monitoring of therapy is initially required weekly or biweekly to note the drug level required for alleviation of symptoms balancing the specific side effects like altered sleeping patterns and poor appetite. Details of drugs used are enlisted in table 3. Continued monitoring may be required at 3 monthly intervals.

Drug holidays are planned breaks, shorter (during weekends) or longer (during vacations) from medications, when there is lesser demands for performance which give span for catch up growth and control of adverse effects along with need for continuing pharmacotherapy. After few years on treatment, clinicians offer trials of withdrawing the drugs for assessing the need of therapy and if successful adjunct psychoeducation and behavioural modifications along with gradual tapering of dosage are attempted.[11]

Combination therapy uses both behavioral interventions and medications. Combination therapy may be initiated in preschool children who do not respond to behavioral interventions, also some studies have shown better outcome even in school children on monotherapy with reduced dosage and frequency of drugs.[7]

Treatment for ADHD can be initiated by primary paediatrician only based on diagnosis using DSM-5 guidelines with the plan as described above. Though in certain cases referral to a developmental paediatrician or a pediatric neuropsychiatrist seems prudent like in conditions with comorbidities (oppositional defiant disorder, conduct disorder, substance abuse, emotional problems); coexisting neurologic or medical conditions (seizures, tics, autism spectrum disorder, sleep disorder); or in cases of less than adequate response to a trial of stimulant or Atomoxetine therapy or combination therapy.

What are the key messages?

Multisite multiple informant approach fulfilling DSM 5 Criteria with obvious social impairment is necessary for the diagnosis of ADHD. Cases without co morbidities can be managed adequately by pediatricians. Behavioural therapy with environmental and educational interventions play the major role in management along with pharmacotherapy. Scope of further research and paving the path for disability certification is warranted

Table 3. Details of pharmacotherapy

Drugs (Type)	Duration of action	Dosage	Side effects
Methylphenidate (Stimulant)	3-5 hours	5mg/day for day 1 then 5mg twice daily.	Appetite suppression, growth retardation, tachycardia, rising BP, cardiac adverse effects of therapy, orthostatic hypotension insomnia, mania, psychosis, suicidal behaviour, labile mood, hepato-toxicity, tics, seizures.
Delayed onset Methylphenidate (Stimulant)	3-8 hours	5 mg/day twice daily. Increments 20 mg per day, every 3-7 days	
Dextro-amphetamine (Stimulant)	4-5 hours	2.5-5 mg 1-2 times daily, increments 2.5-5mg weekly	
Atomoxetine (Non-stimulant)	10-12 hours	0.5 mg/kg per day for 3 days Increase to 1.2 mg/kg per day after at least 3 days	
Clonidine (Non-stimulant)	10-16 hours	0.1mg tablet at bed time	

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Self – Harm

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Abstract - *Self-harm or self-injury encompasses a broad class of phenomenon in which an individual directly and deliberately cause harm to own self. Such behavior can include nonsuicidal self-injury (NSSI) which refers to direct and deliberate destruction of one's own body tissue in the absence of intent to die. NSSI should be differentiated from the stereotypical self-injurious behavior (SIB) found in individual with intellectual developmental disabilities. It is also different from severe form of self-mutilation such as limb amputation seen in psychotic patients. There is a lack of consensus in conceptualization, definition, classification and diagnosis of the problem. There is also a lack of sufficient understanding about the function or etiology of self-injury. The nosology, psychosocial characteristics, functions and diagnosis of the two common types of self-injury in children and adolescents, namely NSSI and SIB are reviewed highlighting their similarities and differences.*

The behavior of deliberate self-harm or self-injury is one of the most perplexing clinical phenomena. This behavior in children and youth may be viewed in two perspectives.

A. Self-injury with intent to die (Suicidal behaviors)
B. Self-injury without intent to die (Nonsuicidal self-injurious behaviors)

The Nonsuicidal self-injurious behaviors may again be conveniently classified in the following categories.

1. Nonsuicidal self-injury (NSSI) occurring in typically normal developing children and youth.
2. Self-injurious behaviors (SIB) occurring typically in children and youth with intellectual developmental disabilities (IDD).

But, nevertheless, it is to be mentioned here that there is a lack of consensus regarding what self-harm is or is not. Moreover, diverse terms like deliberate self-harm[1], moderate self-mutilation[2], self-cutting, self-wounding[3] have been used interchangeably to describe this phenomenon. The term parasuicidal behavior which is most often used to describe suicidal behavior has also been applied to nonsuicidal behavior[4]. In this scenario of diverse conceptualization regarding self-injury, the above-mentioned approach[5] to delineate the problem appears to be most precise and comprehensive. The present article aims to describe the characteristics and features of the two most common presentations of self-injury in children and adolescents.

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Definition

NSSI :

Nonsuicidal self-injury can be defined as the deliberate destruction of one's own body tissue, occurring without any conscious suicidal intent and for purpose not socially sanctioned. Thus it does not include piercing and tattoos. It also should be differentiated from severe forms of self-mutilation seen in psychotic individuals. NSSI occurs among typically normal developing children and adolescents and should be differentiated from the stereotypic self-injurious behaviors (SIB) in children with intellectual development disorders. Common types of NSSI are cutting, burning, scratching and bruising, piercing, hitting, and picking at wounds interfering with healing[6] and digging nails into the skin[7]. Cutting is most common and found to be 70-90% in different studies[8].

SIB :

Self-injurious behavior (SIB) may be defined as physical acts directed to one's own body that result in or produce tissue damage, or have the possibility of producing tissue damage if left unchecked[5]. SIB

is found in children with intellectual development disabilities (IDD) and has profound implications for a child's health and quality of life. SIB also include stereotypic movement disorder (SMD) found among the children with intellectual disability. The most common types of SIB are different from NSSI and include head banging, biting, pinching and rubbing. Each individual with SMD presents with his or her own uniquely patterned behavior. Stereotypic movements may occur many times a day, lasting a few seconds to several minutes or longer. The behavior may occur when the individual is excited, stressed, fatigued or bored.

Epidemiology

NSSI :

Most commonly, the age of onset of NSSI is early adolescence (12-14 years). Studies have reported a history of self-injury in 4% of United State population[9], 4% of military recruit[10], and 14% or more of college students[11]. In American society, 14% to 20% of adolescents in community samples report having engaged in NSSI at least once during their life time[5]. One Indian community-based study on NSSI reported a lifetime prevalence of almost 31% in a sample of emerging adults. Around 19.8% of this sample engaged in moderate and severe forms of NSSI. The average age of onset of NSSI behaviors were 15.9 years[15]. For those adolescents and youth who engage in self-injurious behavior for the first time approximately 20% will repeat the behavior within the same year[8].

The incidence of NSSI is becoming increasingly common among pre-pubertal children. Retrospective reports of self-injuries show that NSSI is not uncommon in children. Self-injurious behaviors (particularly cutting) has been documented in children as young as 7 years old[7].

It was originally believed that NSSI is predominantly a female behavior. But studies regarding gender difference in NSSI have revealed divergent results. Some studies have found female predominance in this behavior while others concluded that there is little or no gender difference in occurrence of NSSI.

SIB :

The age of onset and gender difference for SIB are different from those of NSSI. SIB occurs at all levels of IDD across life span. The frequency of SIB increases with the severity of intellectual impairments. The age of onset is not specifically known, but SIB has been documented in 18 months old child[5].

Unlike NSSI, SIB has a clear gender difference with male preponderance by a ratio of 4 to 1 approximately.

Function

One of the most pertinent questions regarding self-harm is- "Why they do so?" Researchers have revealed several motivations underlying self-harm and the various functions that self-harm serves.

NSSI :

The functions of NSSI (i.e. motivating and reinforcing variables) can be categorized into the following models[8]. But the models described are not mutually exclusive and different functions may co-occur and overlap conceptually.

1. *Affect-regulation model* – It suggests that self-injury is a strategy to alleviate acute negative affect i.e. to elicit a calm feeling or to eliminate tension. Emotion regulation difficulties are at the root of this behavior. The individuals engaging in NSSI are less able to manage their affect and therefore prone to use self-injury as a maladaptive affect regulation strategy. They, when asked about the cause of their behaviors endorse reasons like – "to stop bad feeling" ; " to relieve anxiety and despair that I feel I can't otherwise control" ; " distraction from painful feeling" : " to feel relaxed" etc.
2. *Anti-suicide model* – It suggests that self-injury is a coping mechanism for resisting urges to attempt suicide. From this perspective, self-injury may be thought as a means to express suicidal thoughts without risking death or a compromise with the desire to commit suicide[11]. In one study 41% of adolescent self-injurers from a non-clinical population endorsed the reason "it stopped me from killing myself"[12]. In another study, the participant selected the reason "stop suicidal ideation or attempts".
3. *Anti-dissociation model* – Self-injury is a response to periods of dissociation or depersonalization and the individuals who self-injures, use it to decrease dissociative symptoms specially depersonalization and numbing. Depersonalization is a type of disorder that consists of persistent or recurrent feelings

of being detached (dissociated) from one's own body or mental process, usually with feeling of being an outside observer of one's life (one's own thoughts or body). Causing physical injury to oneself may shock the system – perhaps through sight of blood or the physical sensation – and thereby interrupt a dissociative episode – and lead one to regain a sense of self. Reasons endorsed by the individuals who self-injure may be like – "to stop feeling numb or dead "; "to feel something even if it is pain "or "to feel real again".

4. *Self-punishment model* – It suggests that self-injury is an expression of anger or derogation towards oneself. Self-punishment reasons are common in adolescent self-injurers. In one study about 50% of adolescents receiving psychiatric treatment endorsed the reason "punish self for being bad / bad thoughts"[13]. In another study in a community sample of adolescents self-injurers, 70% endorsed the reason "I did not like myself" or "I felt like a failure"[12].
5. *Interpersonal-influence model* – Self-injury may be used to influence or manipulate people in self-injurers environment. It is a cry for help, a means of avoiding abandonment, or an attempt to be taken more seriously. For example, an individual might self-injure to elicit affection from loved one, or to elicit responses from authority figures or peers.
6. *Interpersonal boundaries model* – It suggests that self-injury is a way to affirm the boundaries of self[11]. Self-injurers are thought to lack a normal sense of self due to insecure maternal attachments and a subsequent inability to individuate from mother[14]. Marking the skin, which separates individuals from environment and other people, is thought to affirm a distinction between oneself and others and thereby asserts one's identity or autonomy. In one study , it was found that 26% of psychiatric patients endorsed "ownership of the body" as a reason for self-injury[9].
7. *Sensation-seeking model* – It regards self-injury as a means for generation excitement in a manner similar to sky-diving or bungee-jumping. This model is not common in clinical practice. The reasons cited by adolescent self-injurers are like – "to provide a sense of excitement" or "I thought it would be a fun".

SIB :

The etiology of SIB, in some cases, may be related to an undiagnosed painful medical condition. But, in most cases the specific etiology of SIB is unknown. Communication impairment associated with IDD and severity of intellectual impairment are two important primary risk factors.

Co-occurring disorder

NSSI :

The knowledge of co-occurrence is very important to assess and treat adolescents who engage in self-injury. Some of the most commonly co-occurring disorders are borderline personality disorder, mood disorder, anxiety disorder, impulse control problems, uncontrolled anger, alcohol or substance abuse, eating disorder. Repeated engagement in NSSI may lead to increased pain tolerance and reduction in the fear of death. This in turn, may increase the risk of suicide attempts. Some researchers consider NSSI as a gateway to suicide[16].

SIB :

Some IDD-related syndromes tend to occur with SIB. The most common of these are Lesch-Nyhan Syndrome, fragile X syndrome, Cornelia deLange syndrome, Prader-Willi syndrome and Rett syndrome.

Diagnosis

NSSI :

It is to be noted that NSSI is largely a hidden behavior. Most individuals engaging in self-injury try to keep the behavior secret. Sometimes they reveal it only to friends or peers in online communications. However, physical examination may reveal fresh injury, scars, burns or unexplained bruises. Sometimes there may be pin or razor blade scratching which the injurers explained as 'cat scratching". The signs of self-injury should also be differentiated from the signs of physical abuse. NSSI is often overlooked in male due to the misconception that it is largely a female behavior.

One of the most important parts of the diagnosis is that it is to be differentiated from suicidal behavior. Suicidal attempt and NSSI are distinct behaviors. Suicidal attempts are behaviors that may or may not result in injury. There is intent to die and this behavior expresses to stop living. On the other hand, NSSI is a behavior where immediate tissue injury is present and there is no intent to die, rather it is an

attempt to feel better. Nevertheless these behaviors are not mutually exclusive and some youth engage in both.

SIB :

SIB mostly consists of biting of hands, arms or lips; banging the head on solid surfaces; hitting the head or face with a closed fist or open palm; eye-poking and scratching, pinching or rubbing skin. Mild, moderate or permanent tissue damage and disfigurement can occur. SIB injuries can be mistaken for physical abuse. Nevertheless, the children with development disabilities are at higher risk for abuse and neglect. SIB occurs more often in the presence of certain people, places, materials, demand context and biological states than in others[5].

Risk assessment and referral

NSSI :

The management of NSSI begins with appropriate assessment. It includes a full history of the behavior, including age of onset; incidence over time and also the history of method, frequency, location, number of injury per episode and medical severity of injuries. These variables may change with time reflecting a pattern of waxing and waning. Assessment must include evaluation of risk of suicide and presence of other co-occurring risk factors. Currently, the only treatment approach for NSSI that has some empirical support is dialectic behavioral therapy. The key role of pediatrician is to determine the current risk level and make appropriate referrals.

SBI :

A child with IDD presenting with SBI should be evaluated for any possible illness or the likelihood of an acute or chronic condition that may be painful. Treating the underline condition may lead to reduced SIB. For behavioral assessment, referral to a qualified specialist is recommended.

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Spot the Diagnosis: Ulcerative Skin Lesions over Face, Hand and Foot

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A 5 year old boy, only sibling of consanguineous parents, presented with bizarre looking ulcerative lesions with granulation tissues over his nose and pinnas (Fig 1). Similar lesions were noted over the index and ring fingers of left hand, thumb and middle finger of his right hand and right great toe (Fig 2). A few lesions were secondarily infected. They usually would start as blisters following a minor trauma and appear intermittently over different parts of the body since birth. Enamel hypoplasia,discoloration with excessive carries and dystrophic nails were also noted.

The child was clinically diagnosed as epidermolysis bullosa (EB). For further identification, biopsy was performed after rubbing the skin with an eraser for immunofluorescence antigen mapping. The specimens were stained along with control normal skin for BP antigen, collagen IV, laminin 332, collagen VII. The biopsy revealed sub epidermal split with BP antigen taking the roof of the blister and collagen IV and collagen VII taking the floor of the blister. Also, there was reduced expression of laminin 332 at the basement membrane zone compared to that of control, confirming a diagnosis of generalised, intermediate junctional epidermolysis bullosa (JEB), formerly called non Herlitz JEB.

This is a major form of EB with autosomal recessive inheritance, characterised by skin separation at the level of lamina lucida of dermo-epidermal junction. There are two main types: Herlitz JEB (absent Laminin 332) and non-Herlitz JEB (Laminin 332 present but reduced) which are now called as JEB generalized severe and JEB generalized intermediate respectively. Intermediate or Non- Herlitz JEB is the less severe form with less scarring and a typical distribution of lesions as are present in the index case.

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Figure in the facing page

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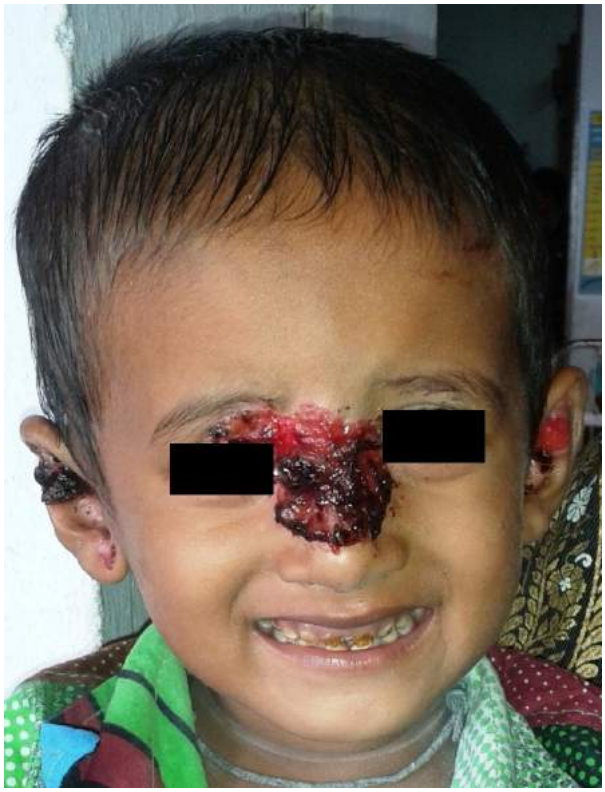

Fig 1: Ulcerative skin lesion with granulation over the nose and pinnas with enamel hypoplasia and discoloration

Fig 2: Lesions over limbs with secondary infection and nail dystrophy

Published by Dr Madhumita Nandi, Hony Secretary of West Bengal Academy of Pediatrics, 15C, Canal Street, Kolkata 700014, and Printed by Roshni Enterprise, Kolkata