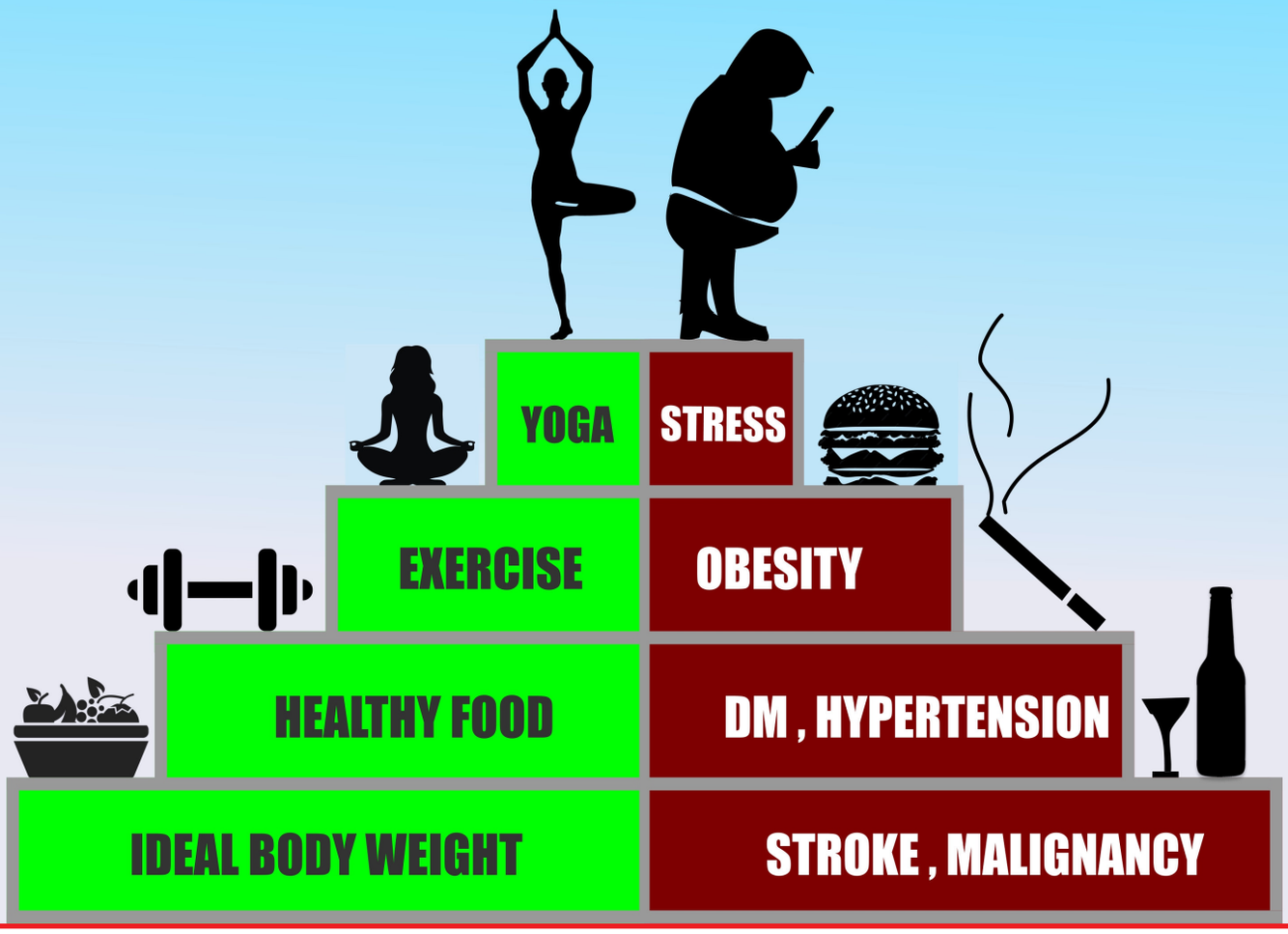
Pulse

Volume 26 December 2024

LIFESTYLE DISEASES



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Pulse

Volume 26 December 2024

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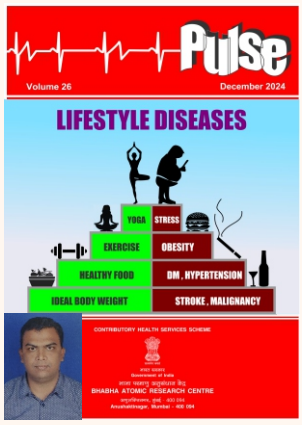

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विषयवस्तु / CONTENTS

संपादक की कलम से — View 01
Editor's Note
डॉ. श्रीविद्या चेल्लम Dr Shrividya Chellam

अध्यक्ष, आयुर्विज्ञान प्रभाग के डेस्क से — View 03
From the HMD's Desk
डॉ. स्नेहल नाडकर्णी Dr Snehal Nadkarni

अतिथि आलेखः तंबाकू का सेवन एवं कैंसर – एक अवलोकन — View 04
Guest Article: Tobacco Usage and Cancer – An Overview
डॉ. अभिषेक देशपांडे Dr Abhishek Deshpande

पॉलीसिस्टिक डिम्बग्रंथि संलक्षणः आगे क्या? — View 08
Polycystic Ovary Syndrome: What Next?
डॉ. संतोषी प्रभु Dr Santoshi Prabhu

जीवन शैली एवं पीठ दर्द — View 11
Lifestyle and Low Back Pain
डॉ. जलपा काटे Dr Jalpa Kate

जीवन शैली बीमारियां एवं कमज़ोरी — View 15
Lifestyle Diseases and Frailty
डॉ. उदय ठाकरे Dr Uday Thakre

हाइपरयूरिसीमिया - एक बढ़ती चिंता — View 18
Hyperuricemia- A Growing Concern
डॉ वैशाली वाढे Dr Vaishali Wadhe

बालपन का मोटापा — View 20
Childhood Obesity
डॉ. उमेश नाईकनवरे Dr Umesh Naiknaware

कीटों से लड़नाः नैदानिक सूक्ष्मजीवविज्ञान के माध्यम से संक्रमणों का पता लगाना — View 25
Battling Bugs: Unravelling Infections through Diagnostic Microbiology
डॉ. सुनयना जांगला Dr Sunayana Jangla

बालपन के मोटापे में आहार की भूमिका — View 35
Role of Diet in Childhood Obesity
श्रीमती अंबिका आर. Smt. Ambika R

उपलब्धियाँ : शैक्षणिक एवं पाठ्येतर — View 38
Achievements: Academic and Extracurricular

इंटरनेट के अधिक उपयोग पर वीडियो — View 41
Video on Internet Overuse
डॉ. अदिति चौधरी Dr Aditi Chaudhari

प्रिय पाठकगण,

हमारी जीवन शैली से जुड़ी बीमारियाँ काफी आर्थिक कठिनाइयों के साथ एक विश्वव्यापी समस्या है/ कोविड महामारी का समय अत्यंत चुनौतीपूर्ण रहा जिसने जीवन शैली के व्यवहार को वैश्विक स्तर पर प्रभावित किया है/ स्क्रीन-टाइम में वृद्धि और शारीरिक गतिविधियों में कमी के साथ-साथ मानसिक स्वास्थ्य पर प्रभाव इसके प्रमुख परिणाम रहे हैं/

मोटापा, हृदय रोग, अतिरक्तचाप, मधुमेह, वसीय यकृत, कैंसर आदि जैसी स्थितियाँ बढ़ रही हैं/ विश्व स्वास्थ्य संगठन (डब्ल्यूएचओ) के अनुसार, ये दीर्घकालिक बीमारियाँ, बीमारी के वैश्विक बोझ के 49% के लिए उत्तरदायी हैं जो वर्ष 2030 तक बढ़कर 56% प्रतिशत होने की प्रत्याशा है/

सामान्यतया, जीवन शैली एक व्यक्तिगत पसंद है परंतु प्रचलित सामाजिक प्रथाओं से बहुत अधिक प्रभावित है/ यहाँ तक कि स्वास्थ्यकर प्रथाओं में कुछ मामूली समायोजन भी सकारात्मक प्रभाव डाल सकते हैं, विशेषतः जब जीवन के शुरूआती दौर में ही इन्हें प्रारंभ किया जाए/

पल्स का यह वर्तमान अंक न केवल बालपन के मोटापे, पॉलीसिस्टिक डिम्बग्रंथि रोग, पीठ दर्द आदि जैसे प्रमुख विषयों पर चर्चा करता है, बल्कि कुछ पोषक रेसिपी भी उपलब्ध करता है/ साथ ही, व्याख्या सहित एक अंतर्दृष्टिपरक कविता आपको मनन करने पर विवश कर देगी/

मुझे आशा ही नहीं बल्कि पूर्ण विश्वास है कि सुधी पाठकगण इस अंक में प्रकाशित लेख को अवश्य पढ़ेंगे और इनका लाभ उठाएंगे/ ऐसी कामना करते हुए.....

आप सभी को नव वर्ष 2025 शुभ हो/

Shrividya

डॉ. श्रीविद्या चेल्लम
मुख्य संपादक, पल्स

अपनी प्रतिक्रिया/सुझाव pulse@barc.gov.in पर प्रेषित करें/

Dear Readers,

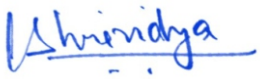
Diseases linked to our lifestyle are a world-wide problem with considerable economic implications. The Covid pandemic has been a watershed moment which has influenced lifestyle behaviour globally. Increased screen-time and reduced physical activities along with impact on mental health has been its major repercussion.

Conditions like obesity, heart diseases, hypertension, diabetes, fatty liver, cancer etc are on the rise. According to WHO, these chronic diseases are responsible for 49% of the global burden of disease which is expected to increase to 56% by 2030.

Generally, lifestyle is a personal choice but is hugely influenced by prevalent social practices. Even a few modest adjustments to healthier practices can have positive impact especially when started early in life.

Current issue of pulse not only discusses key topics like childhood obesity, polycystic ovarian disease, low back ache etc. but also provides a few nutritious recipes. An insightful poem on internet overuse with illustrations will compel you to introspect.

Happy reading!



Dr Shrividya Chellam
Chief Editor, Pulse

Dear Readers,

Greetings! Once again it is the time to connect to you from HMD's desk of PULSE.

The focus of health care has shifted from 'curative' to 'preventive'. This is true more so in relation to diseases linked with the manner in which a person leads life and are called 'lifestyle diseases'. Lack of physical activity, unhealthy eating habits, substance use, and behavior factors like unemployment, poor social environment, working conditions, and stress lead to lifestyle diseases. These in turn have an impact on work force and cost of health care of an organization.

Lifestyle diseases, sometimes called diseases of civilization, are non-communicable diseases which include Alzeimer's disease, arthritis, chronic obstructive pulmonary disease, heart disease, diabetes, asthma , obesity, to name a few. In the past, these diseases were considered diseases of longevity, however, these are now seen in the young.

Lifestyle diseases can be prevented at the individual, family and societal levels. Early life influences can impact manifestation of lifestyle diseases in adulthood. Diet and lifestyle modifications are the main stay of prevention. Avoiding tobacco, alcohol, processed foods along with eating well balanced food are all methods of prevention. Behavior changes like "dos and don'ts" need to be implemented through interactive discussions and need to be reviewed and supported in order to reap in the full benefits of these changes. The person needs to feel "ownership" of the changes suggested and agree to make those changes.

Although making changes is hard and the results are often unpredictable, it is advisable to make small changes in lifestyle at a time and maintain the same before proceeding to make the next change.

The community too can impact lifestyle diseases in a number of ways. Peer pressure can influence behaviors like eating healthy, avoiding alcohol, tobacco; marketing strategies can create a culture of healthy eating especially through responsible food advertising. Promoting outdoor activities by providing outdoor space and opportunities to participate in competitions through educational institutes can reduce screen time. Active family life in the form of outings and celebrations in real vis a vis virtual can have positive effect on prevention of lifestyle diseases.

Here's to a healthy and happy living!

Dr Snehal U Nadkarni

Head Medical Division

Tobacco Usage And Cancer – An Overview

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Introduction

Cancer ranks as the second leading cause of mortality with 9.7 million cancer deaths and 19.98 million new patients with cancer globally.[1] In 2022, India ranked third in the total new patients with cancer across all sites, genders, and age groups, reporting 1,413,316 patients (with an age-standardized rate [ASR] of 98.5 per 100,000 population). The prevalence of patients with cancer is estimated to rise by 12.8% in 2025 compared with 2020. Multiple risk factors contribute to cancer formation, including tobacco use, lifestyle choices, obesity, exposure to infectious agents, family history, dietary habits, and alcohol consumption.[2]

Tobacco use is a major risk factor for many chronic diseases, including cancer, lung disease, cardiovascular disease and stroke. India, ranking second in both tobacco production and consumption worldwide, faces an alarming toll of over 1.35 million tobacco-related deaths annually. The most prevalent form of tobacco use in India is smokeless tobacco and commonly used products are khaini, gutkha, betel quid with tobacco, zarda etc. Smoking forms of tobacco used are bidi, cigarette, hookah etc.[2]

Smokeless Tobacco

SLT is defined as all commercial or non commercial products that contain tobacco, but are not ignited at the time of consumption. Smokeless tobacco is a complex chemical mixture that contains a variety of chemicals and additives, including flavors, areca nut, and slaked lime, and used with betel leaves. Smokeless Tobacco (SLT) products are extremely complex, containing almost 4000 compounds, many of which are hazardous, mutagenic, and carcinogenic in nature.[3]The most harmful compounds in smokeless tobacco are tobacco-specific nitrosamines (TSNAs) and their levels are directly related to the risk of cancer. [3]The alkaloid nicotine, the primary addictive

Dr Abhishek R. Deshpande

substance in tobacco exists in protonated and un-protonated forms. The addition of slaked lime in the preparation of SLT enhances nicotine bioavailability. Areca nut, which is combined with tobacco in several SLT products, is also a confirmed carcinogen. Areca nut contains alkaloids, the most abundant among them being arecoline, from which areca nut–specific nitrosamines, known carcinogens, are formed.[3]

Few of the most common smokeless tobacco products are as follows,

1. Paan (Betel quid) - The consumption of paan (betel quid) with or without tobacco, has been a socially acceptable practice in India. A betel quid typically contains areca nut, catechu (kattha), slaked lime, cinnamon, cardamom, sweeteners and exotic spices wrapped in a betel leaf. Some tobacco derivatives, such as zarda (flavoured, spiced tobacco) and kiwam (fermented thick paste made with tobacco leaf extract, spices and other additives), may also be added to a betel quid. It has been observed that the traditional betel is gradually being replaced by manufactured products such as paan masala and gutkha.[4]

2. Khaini - Khaini (a mixture of sundried, flaked tobacco and slaked lime) is the most commonly used SLT product in India.[4]

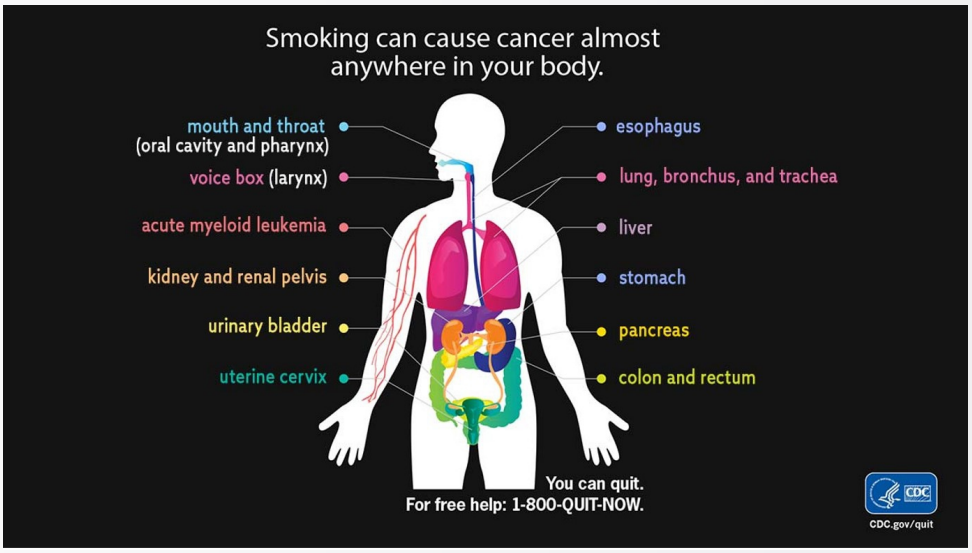

Fig. 1: Smoking and the various organs where cancer is caused

3.Gutkha - Gutkha is the second most commonly used SLT product in India. It is a commercially prepared mixture of tobacco, slaked lime, catechu, areca nut and condiments, and is typically available in small plastic/aluminium sachets. Numerous varieties of paan masala, with or without tobacco, are consumed in India.[4]

Smoking

Cigarettes contain many dangerous chemicals. Some occur naturally in tobacco, and others are formed when processing tobacco into cigarettes. When a cigarette is burnt, it releases thousands more chemicals in tobacco smoke. Many of these chemicals are harmful to people who smoke and also to people who breathe in second-hand smoke. We know that at least 70 of these chemicals cause cancer. Roll-up tobacco cigarettes are not safer. They contain the same cancer-causing chemicals as manufactured cigarettes. Smoking using a filter, and smoking 'low-tar' or 'light' cigarettes also doesn't reduce the risk of disease from smoking. These are not safer or healthier options.

Cancer causing agents:

1. Tobacco-specific N-nitrosamines - Long-term use of some products having smokeless tobacco show increased risk of oral cancer and mainly this elevated risk is due to the presence of tobacco-specific N-nitrosamines (TSNAs). 4-methyl-N-nitrosamino-1-(3-pyridyl)-1-butanone and N-nitrosonornicotine (NNN) are the two TSNAs which are major cancer causing agents in smokeless tobacco. The International Agency for Research on Cancer (IARC) classified them as carcinogenic to humans.[3]

2. Cadmium - The International Agency for Research on Cancer considered cadmium as a cancer causing agent due to increased cancer of lungs in industrial workers through inhalation of high concentrations of it or animal research.[3]

3. Formaldehyde - Formaldehyde is used widely in industry as a chemical and in manufacturing. The International Agency for Research on Cancer classified it as a cancer causing agent in humans, basically in workers who work in industry and are continuously exposed to it or in animal research[3]

4. Polycyclic aromatic hydrocarbons - Incomplete combustion of organic compounds produces polycyclic aromatic hydrocarbons (PAHs). Benzo[a]pyrene (BaP) is the most common and intensively studied PAH. Smokeless tobacco contains BaP mainly from fire-curing, and thus its concentration depends on the amount of tobacco used that is fire cured[3]

5. Lead - Based on animal research, IARC classified lead as a cancer causing agent to humans.[3]

6. Tar - Tar is a sticky-brown substance that collects in the lungs when tobacco smoke is breathed in. It can stain fingers and teeth a yellow-brown colour.Tar contains cancer-causing chemicals. It also increases the risk of lung diseases, such as emphysema and chronic obstructive pulmonary disease (COPD).[4]

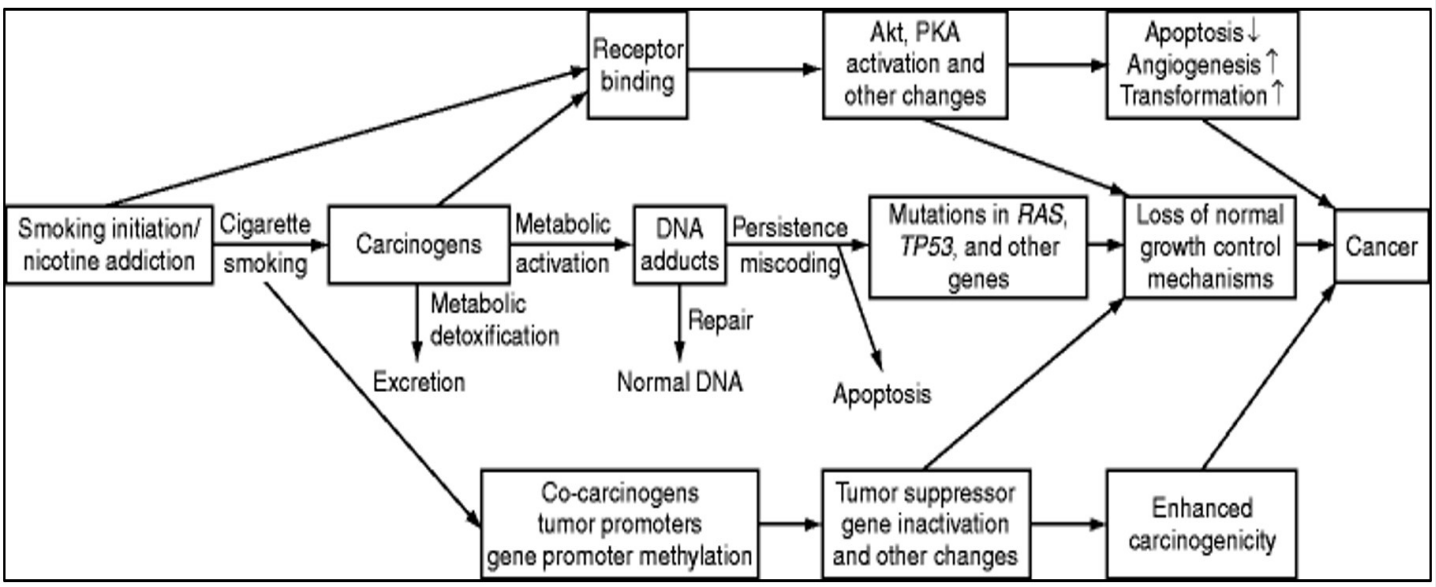

Fig. 2: Tobacco induced carcinogenesis

Hecht, S.S. (2011). Tobacco Carcinogenesis. In: Schwab, M. (eds) Encyclopedia of Cancer. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-642-16483-5_5846.

7. Carbon Monoxide-Tobacco smoke contains a poisonous gas called carbon monoxide.

Carbon monoxide stops the blood from carrying as much oxygen.

This means organs of the body don't get the amount of oxygen they need, and the heart must work harder to supply the body with oxygen. This increases the risk of heart disease and stroke.[4]

How does Tobacco cause cancer?

Among the various carcinogens, tobacco-specific nitrosamines (such as NNK and NNN), PAHs (such as benzo[a]pyrene) and aromatic amines (such as 4-aminobiphenyl) are the prominent carcinogens that have been verified in animal models and positively identified in cigarette smoke.[4]

How to quit tobacco in India?

It is established that a majority of smokers (as many as 70%) desire to quit, but only 30% actually try each year, and only 3%- 5% actually succeed in quitting. Tobacco dependence is a chronic condition that often requires repeated intervention.[5] However, effective treatments exist that can produce long-term or even permanent abstinence. Because effective tobacco dependence treatments are available, every patient who uses tobacco should be offered at least one of these treatments:

1. Patients willing to try quitting tobacco use should be provided with treatments identified as effective.

2. Patients unwilling to try quitting tobacco use should be provided with a brief intervention

designed to increase their motivation to quit.[5]

Numerous effective pharmacotherapies for tobacco cessation now exist. Except in the presence of contraindications, these should be used with all patients attempting to quit tobacco use.

Pharmacological interventions when used with behavioural strategies can produce quit rates of about 25 –30 %. Pharmacotherapies that reliably increase long-term smoking abstinence rates include:

a. Agents that appear to decrease craving - Bupropion, Selegeline, Nortryptiline etc.

b. Agents, which are used to substitute the nicotine, obtained from tobacco - Nicotine gum, Nicotine patch, Nicotine inhaler or Nicotine nasal spray. Nicotine replacement Therapy (NRT) is useful and associated with quit rates of about 23% as against 13% with placebo.[5]

The National Tobacco Control Programme (NTCP) has created various helpline facilities to provide counselling

Quitline: Counselling on telephone by expert counsellors in all languages (Tollfree 1800 11 2356)

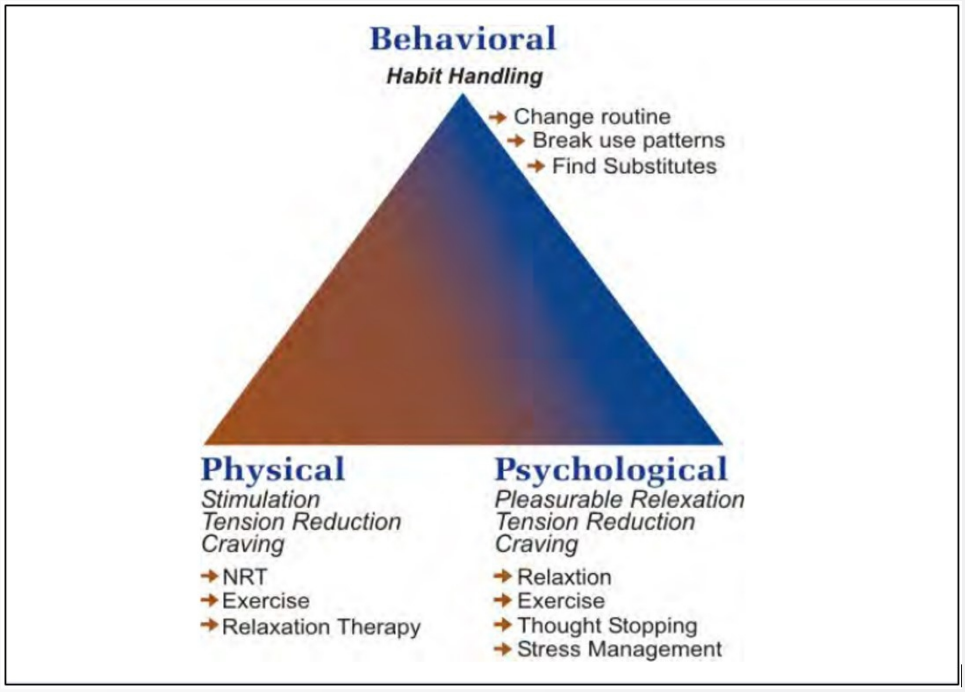

Fig. 3: The Addiction Triangle to assess treatment needs
Tobacco Cessation Programme Facilitator Guide.
http://chppmwww.apgea.army.mil/dhpw/Population/Tobacco 2020

mCessation: Counselling through mobile SMS (011 22901701)

Tobacco Cessation Centre (TCC): Counselling at district level[5]

Conclusion

Tobacco smoking is the main known cause of cancer–related death worldwide. Similarly smokeless tobacco also is the major cause cause of oral and oropharyngeal cancers.Tobacco induced carcinogenesis is a well established fact and yet the number of cancer patients due to tobacco continue to rise.All efforts should be made not only individually but also at the community and national level to promote habit cessation and prevent this avoidable form of disease

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Polycystic Ovary Syndrome : What next?

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Introduction

Polycystic ovary syndrome (PCOS), is a global health concern affecting 8-13% of women in the reproductive age group. The impact of PCOS goes beyond its reproductive repercussions and often challenges both metabolism and psychological health.

The reproductive impact of PCOS include irregular menstrual cycles, hirsutism, infertility and pregnancy complications like diabetes and hypertension. Metabolic features of PCOS include insulin resistance (IR), prediabetes, type 2 diabetes (DM2) and cardiovascular risk factors; whereas psychological features can present as anxiety, depression etc.

Diagnosis

The international consensus for diagnosing PCOS is given by Rotterdam's criteria. According to this, two out of three of the following criteria should be met with after ruling out other aetiologies like thyroid disorders, hyperprolactinemia and adult-onset congenital adrenal hyperplasia

- Clinical and/or Biochemical Hyperandrogenism
- Oligo or Anovulation
- PCO Morphology on Ultrasound

Clinical and/or Biochemical Hyperandrogenism

It is characterised by clinical signs like acne, alopecia and hirsutism.

The Ludwig visual score helps one to assess the degree and distribution of alopecia. Modified Ferriman Gallwey score is used for assessing severity of hirsutism, with a level ≥ 4 - 6 indicating hirsutism. Sudden, severe progression of hirsutism prompts one to look out for androgen secreting tumour (ovarian or adrenal origin). Another characterising feature of this condition is biochemical hyperandrogenemia.Free testosterone; free androgen index is estimated by using liquid chromatography mass spectrometry, mass spectrometry and extraction, chromatography immunoassays etc. Androstenedione, dehydroepiandrosterone sulphate and 17-hydroxyprogesterone levels are advised to rule out adrenal related condition. As Anti-mullerian hormone (AMH) levels change in different phenotypes of PCOS, it is not a diagnostic indicator. However, AMH can help to differentiate between clinical phenotypes, as it is strongly related with PCO morphology but not with hyperandrogenism. AMH changes substantially with age, but is found to be stable in PCOS patients under 30 years old.

Dr Santoshi Prabhu

Oligo or Anovulation

Menstrual irregularity in first 12 months from the onset of menarche is disregarded from evaluation point of view. Drug history, contraception usage: especially injection Depot Medroxy Progesterone acetate (DMPA) in such presenter is to be ruled out.

According to European Society of Human Reproduction and Embryology (ESHRE) guidelines (2018), cycles are considered irregular in following circumstances:

- > 1 to < 3 years post menarche: cycle duration of < 21 or > 45 days
- > 1 year post menarche, cycle duration of > 90 days for any one cycle
- > 3 years post menarche to perimenopause: cycle duration of < 21 or > 35 days or < 8 cycles per year.

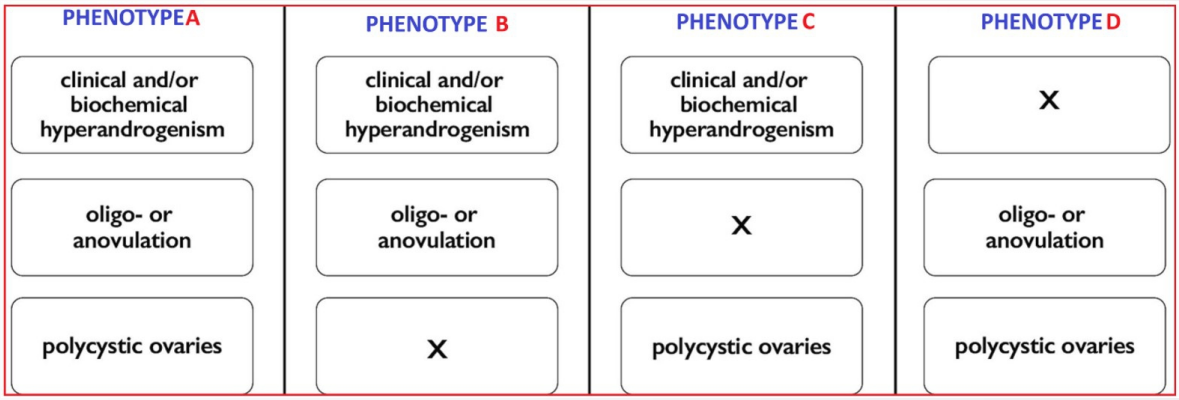

Fig. 1: Modified Rotterdam Criteria for PCOS phenotype

PCO Morphology (PCOM) on Ultrasound

PCOM is ≥20 follicles per ovary measuring 2-9 mm diameter and/ or an ovarian volume ≥ 10ml, after ruling out corpus luteum, cysts of other origins, or dominant follicles.PCOM is considered normal findings on USG in adolescents, perimenopausal women, thyroid disorders and in women on combined oral contraceptives (COCs).

PCOS: What is next?

Role of Genetics and Epigenetics in PCOS

Genome-wide association studies have indicated the role of the following genetic contributors for PCOS, based on similarities in their promoters: luteinizing hormone/chorionic gonadotropic receptors (LHCGR), DENN domain containing 1A (DENND 1A) and thyroid adenoma-associated gene (THADA).

LHCGR are found normally in theca and mature granulosa cells of the adult ovary. Some women with PCOS present with polymorphisms of these receptors, which is responsible for excessive androgen production. Overexpression of gene, DENND 1A, leads to excess ovarian steroidogenesis. Perturbed levels of THADA alter insulin secretion and normal functioning of pancreatic beta cells, thereby causing a subsequent IR.

Emerging research suggests that epigenetic changes that affect gene expression without altering the DNA sequencemay also play a significant role in the development of PCOS. Environmental factors, such as diet and stress, could potentially influence these epigenetic changes, offering new avenues for prevention and treatment.

One such environmental factor includes increased fetal exposure to androgens as a predisposing factor to PCOS. A recent study proposed that insulin resistance in the mother (causing hyperinsulinemia) can trigger the ovaries of the foetus to kick in steroidogenesis with excess androgen production. Under normal circumstances, the aromatase activity of the placenta resolves this excessive androgen. In PCOS pregnancies however, previously reported placental dysfunction combined with hyperinsulinemia induced androgen production, results in a strongly hyperandrogenic foetal environment. Correlative evidence from many models indicates that such an in-utero environment leads to epigenetic changes like methylation of DNA and development of PCOS phenotypes.

This epigenetic re-programming leading to changes in the methylation status of genes regulating folliculogenesis and/or steroidogenesis, glucose metabolism, insulin regulation, etc results in the syndromic outcomes of PCO. Other avenues such as microRNA and mitochondrial DNA which are maternally inherited are also hypothesised to be potential candidates for causing PCOS.

Microbiome Research

Both gut as well as vaginal microbiomes have been found to play a role in the pathophysiology of PCOS. Alterations in gut bacteria have been linked to insulin resistance and inflammation, which are key components of PCOS. This has led to an increased interest in probiotics, prebiotics, and dietary interventions aimed at modulating the gut microbiome as potential therapies for PCOS. Research is also exploring the role of the vaginal microbiome in PCOS, as imbalances here could contribute to reproductive and metabolic symptoms.

Diagnosis of PCOS by Phenotypes

The modified Rotterdam's criteria which define 4 different phenotypes of PCOS A, B, C and D are further categorized into classic PCOS (phenotype A/B) and non-classic (phenotype C/D) PCOS. The classic PCOS is associated

with more obvious menstrual dysfunction, higher prevalence of obesity, hyperandrogenism, deranged lipid profile, increased insulin levels, higher rates of insulin resistance, high level of AMHs, increased body mass index (BMI), risk of metabolic disorders, endometrial cancer and cardiovascular disorders than others. The non-classic PCOS has the mildest degree of endocrine and metabolic dysfunction and the lowest prevalence of metabolic disorders. Differentiation between phenotypes of PCOS is essential for the prognosis of the severity of the disease, the outcome of fertility, and planning individualised treatment.

Preventive Strategies for Long Term Complications

Classic PCOS are more prone for developing long term metabolic and cardiovascular risks such as type 2 diabetes, hypertension, and cardiovascular disease. More emphasis is being placed on early intervention and continuous monitoring of these risks in women with PCOS. Lifestyle interventions focusing on diet, physical activity, and weight management remain the cornerstone of preventing these complications. However, the integration of these strategies with pharmacotherapy tailored to the individual's metabolic profile is becoming more common.

Personalized Treatment Approach and Newer Agents

Identification of phenotype helps to offer personalized treatment approach. This includes specific combinations of medications, lifestyle changes, and alternative therapies based on a patient's unique hormonal profile, metabolic status and genetic makeup. Research is ongoing into new pharmacological treatments for PCOS, particularly those targeting insulin resistance, hormonal imbalances, and inflammation. Newer agents like myo-inositol, D-chiro-inositol, and other insulin-sensitizers are being studied for their effectiveness in managing PCOS symptoms.

Innovations in Fertility Treatment

Technological advancements in ovulation monitoring, including wearable devices and apps are helping women with PCOS to track their cycles more effectively and optimize their chances of conception by timing ovulation.

New approaches in fertility treatment, including more tailored ovulation induction protocols and advancements in in vitro fertilization are improving outcomes for women with PCOS who are trying to conceive avoiding risk of ovarian hyperstimulation syndrome.

Mental Health Focus

Increasing research highlights the significant impact of PCOS on mental health, including higher rates of anxiety, depression, and eating disorders among women with PCOS. As a result, mental health screening and support are becoming integral parts of PCOS management.

Complementary therapies such as mindfulness, cognitive-behavioural therapy and stress management techniques are being increasingly recognized for their role in managing the psychological aspects of PCOS

Conclusion

In summary, the field of PCOS research and treatment is evolving with a greater emphasis on personalized medicine, the role of genetics and the microbiome, mental health and holistic care. These advancements are paving the way for more effective and comprehensive management strategies for women with PCOS.

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Lifestyle and Low Back Pain

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Introduction

Low back pain (LBP) is a commonly encountered musculoskeletal disorder. In India, it is ranked second in causing disability after iron deficiency anaemia. Earlier considered as disease of elderly but due to urban life style, its prevalence is increasing in all age groups. The estimated prevalence of LBP in India is reported to range between 42% and 83%.

Depending upon the origin of low back pain it can be divided in to Spinal (Spondylosis)/mechanical /degenerative pain originates from the various spinal components such as the spinal nerve roots, facet joints , paraspinal muscles and ligaments, sacroiliac joints, and intervertebral discs. LBP of spinal origin can be kept under control with lifestyle modifications.

Non mechanical /non degenerative pain result from pathologic conditions of spine such as neoplasm, infections, such as tuberculosis, inflammatory spinal disorders such as ankylosing spondylitis,traumatic or pathologic fractures etc.

Chronic LBP of extraspinal and visceral etiology LBP can also be referred from other organs, particularly retroperitoneal structures such as pelvic viscera (prostatitis, endometriosis, or pelvic inflammatory disease), kidney (nephrolithiasis, pyelonephritis), gastrointestinal disease such as pancreatitis, cholecystitis, or perforated bowel

In this article, focus will be on LBP of mechanical or degenerative origin and associated lifestyle factors.

Risk Factors

Biomechanical risk factors are determined by abnormal spinal loading and poor posture. Psychosocial factors include psychogenic stress. Personal risk factors are physical, familial, anthropometric (e.g., obesity), and gender-related.

Age and back pain

The risk of LBP tends to increase with age but there is a smaller peak among individuals aged 15–19 years. Several factors such as body posture, gender, psychological status, and prolonged use of electronics may contribute to low back pain in adolescent age group. It is to be noted that adolescent low back pain can become persistent low back pain in adulthood. Therefore it is important to treat it so as to minimize disability risk. Low back pain in middle age group may be due to poor posture in physically demanding jobs for extended periods. In elderly individuals, age related or disease related degenerative conditions lead to LBP.

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Socio- economic factors and low back pain

With high income, there is a surge in obesity, which has become a major and growing health problem. Rapid urbanization and improved living conditions has led to sedentary and less physically active lifestyle. These factors possibly result in low back pain. Contrastingly, due to lack of awareness and less healthcare facilities, low-and-middle-income class of people may also face significant burden of the disease.

Gender and low back pain

LBP is seen more frequently in women than men. Hormonal changes and anatomical alterations during pregnancy as well as hormonal fluctuation during menstrual cycle are chief causes for LBP in women. Socio-cultural factors such as women's responsibilities in household chores, child-rearing, and care giving increase the risk of musculoskeletal strain and injuries, leading to low back pain. Moreover, societal pressures and cultural norms may discourage women from seeking appropriate medical attention and self-care, resulting in delayed treatment and progression of the condition.

Occupation and low back pain

Occupations that require moderate to vigorous physical activity or prolonged sedentary period may increase the likelihood of developing LBP. Suffering from persisting LBP may result in functional impairment in patients, affecting their quality of life and work productivity. The loss of productivity and increased sick leave caused by low back pain can further increase the economic burden on society.

Diet and back pain

Increased levels of pro-inflammatory mediators in the body can be involved in the pathogenesis of chronic LBP. Higher adherence to the unhealthy diet characterized by refined grains, red meat, processed meat, high saturated fat, trans-fatty acids, sugary foods, and caffeine lead to release of pro-inflammatory mediators and hence back pain. Studies have found that adherence to the Mediterranean and plant-based diet, associated with consuming vegetable oils, especially olive oil, effectively reduces musculoskeletal pain. A healthy dietary pattern comprising of an adequate and balanced intake of all food groups, can moderate the inflammatory conditions of the body. Apart from unhealthy diet, calcium deficiency can also lead to back pain. Vitamin D deficiency is an additional factor aggravating other causes of back pain.

Clinical evaluation

A comprehensive history and physical examination are important components in the diagnosis of low back pain. A detailed history should be elicited to know location (axial with or without radiation to lower limbs), quality, factors affecting pain. Associated neurological symptoms should be noted. Assessment of social and psychological factors that may affect patient's pain are to be considered. In addition to pain score, its impact on activities of daily living should be noted. Red flag pain features such as nocturnal pain, bony tenderness, fever with chills, gait disturbances, lower limb weakness, over flow in cotinence need urgent surgical evaluation. In addition to general examination, specific examination include assessment of gait, range of spinal motion, spinal and paraspinal tenderness. Specific tests for the clinical diagnosis of various LBP syndromes, including those for nerve root irritation,facet syndrome, and sacroiliac joint dysfunction etc are to be carried out. Imaging studies are mainly used for diagnosis. However, they typically reveal abnormal findings in both symptomatic and asymptomatic individuals. It is therefore necessary to correlate imaging studies with a patient's clinical findings.

Following imaging studies are typically ordered for LBP evaluation:

a) Plain radiograph - Revels anatomical problems like fractures, deformities, spondylolisthesis, and spondylolysis. Major limitation is inability to show soft tissue pathologies like herniated disc, neural compression etc.

b) MRI scan- MRI is currently considered the gold standard in spinal imaging. It offers excellent images of the spinal canal, the neural foraminae, nerve roots, and the size and shape of the intervertebral discs. MRI may frequently detect findings in asymptomatic individuals therefore clinical correlation is necessary.

c) CT scan- It is most valuable in diagnosing fractures, tumors involving spine. However it cannot reliably distinguish between herniated disc and epidural scar tissue and amongst various spinal canal lesions such as neoplasms of the spinal cord or the nerve roots. Therefore it is not routinely used.

Management can be broadly divided in to non invasive and minimally invasive and surgical (invasive) treatments.

a) Non invasive modalities include the following [Table 1]

Table 1. Non -invasive therapies for chronic pain

Rest	- Strict bed rest was historically the mainstay of acute LBP treatment. Bed rest of more than 1 week is not recommended - Heating pads can help to relax painful muscle spasms. - Continuation of daily activities and early return to work is associated with a decrease in chronic disability
Pharmacological therapy	- NSAIDS: moderately ef fectivef for acute LBP - Opioids: Short term therapy for acute and exacerbation of chronic LBP. (Risk of tolerance and addiction) - Muscle relaxants: modestly reduce pain, muscle tension, and immobility in patients with LBP
Physical therapy	The treatment goals of various physical therapy modalities include the following: - Pain relief - Reduction in muscle spasm - Improved range of movement - Improved strength - Postural correction - Improvement in functional status
Biofeedback	- Involves monitoring the physiologic muscle activity of the patient (often using EMG) and communicating the state of contraction or relaxation to the patient via visual or auditory signals, the patient can train painful or spastic muscle groups to relax on command. - Limited benefit in chronic LBP patients.

b) Minimally invasive therapies [Table 2]

This include a range of diagnostic and therapeutic local anesthetics and steroid injections which target spinal nerve roots, facet joints, sacroiliac joints, and the various muscles and ligaments. If diagnostic injections of these neural components result in short-term relief of pain, a nerve ablation technique may be employed to achieve longer lasting results.

Table 2. Minimally invasive therapies

Injection therapies	- Transforaminal injection - Facet joint injection - Sacro-ilac joint injection - Trigger point injections
Neuroablative procedures	- Radiofrequency ablation - Cryoablation - Chemical ablation
Intradiscal procedures	- Percutaneous disc decompression - Intradiscal electrothermal therapy - Intradiscal bioculoplasty - Stem cell based therapies

c) Spinal surgery [Table 3]

Aims to relieve LBP by neurologic decompression and/or fusion

Table 3. Spinal surgeries

Decompression surgery	- Discectomy - Microdiscectomy - Endoscopic discectomy - Decompression for fixed osseous stenosis
Fusion	- Anterior fusion - Posterior fusion - Circumferential fusion

Prevention of LBP

- Sit in chairs with straight backs or low-back support. Keep your knees a little higher than your hips. Adjust the seat or use a low stool to prop up your feet.
- Turn by moving your whole body rather than by twisting at your waist.
- When driving, sit straight and move the seat forward. This helps you not lean forward to reach the controls.
- If you must stand for long periods, rest 1 foot on a low stool to relieve pressure on your lower back. Every 5 to 15 minutes, switch the foot you're resting on the stool.
- Maintain good posture: Keep your ears, shoulders and hips in a straight line, with your head up and your stomach pulled in.
- The best way to sleep is on your side with your knees bent. You may put a pillow under your head to support your neck. You may also put a pillow between your knees.
- If you sleep on your back, put pillows under your knees and a small pillow under your lower back. Don't sleep on your stomach unless you put a pillow under your hips.
- Use a firm mattress. If your mattress is too soft, use a board of 1/2-inch plywood under the mattress to add support.
- Don't lift by bending over. Lift an object by bending your knees and squatting to pick up the object. Keep your back straight and hold the object close to your body. Avoid twisting your body while lifting.
- Push rather than pull when you must move heavy objects.
- If you must sit at your desk or at the wheel of a car or truck for long hours, break up the time with stops to stretch.
- Wear flat shoes or shoes with low heels (1 inch or lower).
- Exercise regularly. An inactive lifestyle contributes to low back pain.

BARC hospital data

A total of 700 patients were seen in the Chronic Pain Clinic, in last 10 years. Out of these, 295 patients presented with low back pain and 280 patients underwent interventional pain management procedure. Of these 295 patients, 193 were females and 102 were males. Age wise distribution of patients is shown in figure 1. Majority of the patients underwent transforaminal epidural steroid injection as the treatment while 9 patients received sacro-iliac joint injections, 7 patients received caudal epidural steroid injections, 20 patients received facet joint steroid injection. 10 patients were treated with radiofrequency ablation of facet joint nerve. Multiple pain management interventions were needed in 48 patients. With increasing awareness about Pain Clinic and better diagnostic modalities in BARC hospital, many patients with low back ache are being identified, treated and educated so that they do not progress to a greater severity.

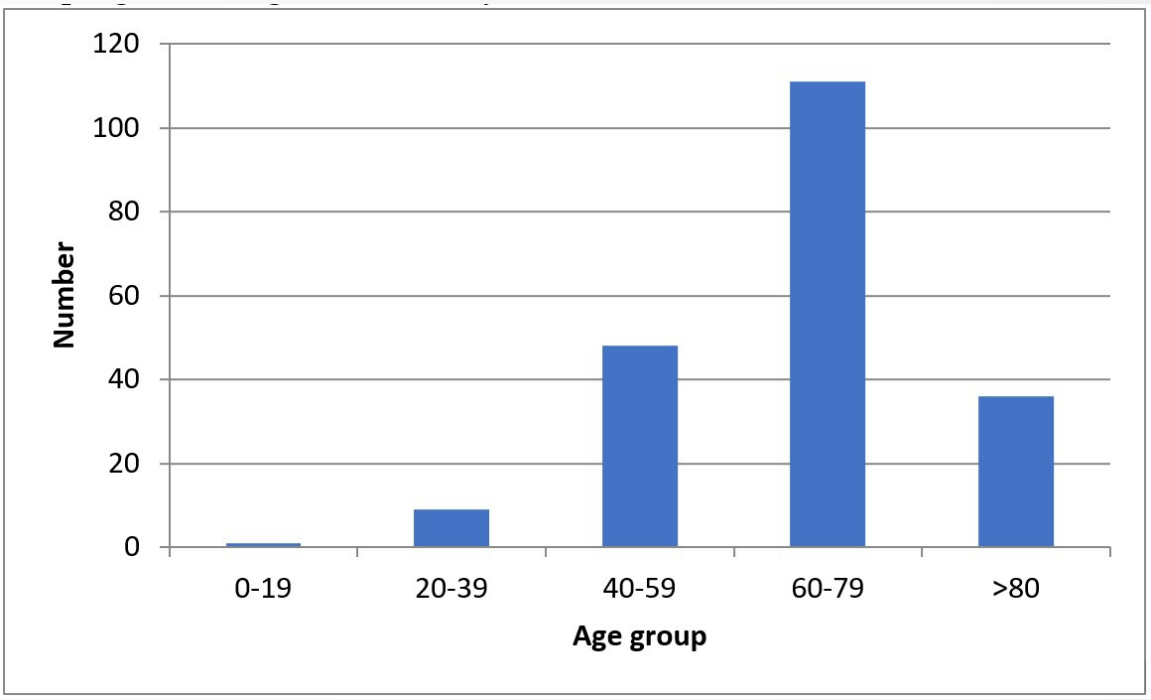

Fig. 1: Age wise distribution of LBP seen in BARCH pain clinic

Conclusion

Common causes for low back pain are degenerative process and life style. Preventative measures such as weight loss, regular exercise, discontinuation of tobacco products, avoidance of excessive or repetitive spinal stress, and maintaining appropriate posture can be adopted for a pain free life. An exercise program directed to increase spinal strength and flexibility will minimize the risk of recurrent LBP.

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The Association between Lifestyle Diseases and Frailty.

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Background

The elderly population is growing worldwide and is nearly 140 million in India which is expected to increase by around 56 million by 2031. With increasing elderly population, concomitant comorbidities will also rise. Frailty among the elderly leads to increased dependency, adverse health outcomes and mortality.

Introduction

Frailty is a multidimensional syndrome characterized by a reduction in the reserve capacity that results in an increased risk of disability and death from minor external stresses. It results in a spiral of decline with an increased risk of worsening disability, increased incidence of admission to hospital, long-term care facility, and death.

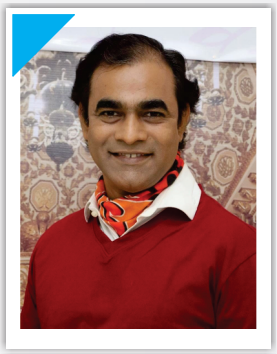
Frailty encompasses unintentional weight loss, weakness, exhaustion, low hand strength, and decreased physical activity. Psychological dimension includes cognitive frailty, low mood, and anxiety. Social dimensions of frailty include a lack of social support and social interactions. Frailty is an aggregate of subclinical reserve losses across multiple systems. Thus, it occurs parallel with multi-morbidity which is the concurrent occurrence of two or more chronic lifestyle diseases.

Lifestyle is referred as the characteristics of inhabitants of a region at a specific time and place. Malnutrition, unhealthy diet, unhealthy habits, substance abuse and sedentary activities leads to lifestyle diseases. Frailty and lifestyle diseases are both associated with increased risk for disability, health care utilization, and mortality among older adults.

Assessment of Frailty

Frailty assessed by using the Frailty Screening Index (FSI), which is comprised of five items answerable with yes/no responses.

- Unintentional weight loss >4.5 kg in last 1 year or BMI <18.5
- Self-described exhaustion
- Weakness
- Slowness
- Low physical activity.



Dr Uday Thakre

The Inter-Related Risk Factors for Frailty and Lifestyle Diseases [Fig. 1]

Not all old individuals are frail, and not all frail individuals are old. The aging process represents a significant risk factor for lifestyle diseases, including insulin resistance, hypertension, and cancers. Obesity, high waist-hip ratio, smoking or previous smoking, sedentary behaviour, and stressful living are significant risk factors for frailty.

Frailty and lifestyle diseases have been a predictor and an outcome of each other, as well as predictors of both disability and mortality.

Stressors in the Implication of Frailty and Lifestyle Diseases

Stressful environments result in a glucocorticoid excess that disrupts normal cellular function, causes cellular damage, system dysfunction, and accelerates aging and frailty. Also, development and progression of stress-related conditions and lifestyle diseases, such as hypertension, coronary heart disease, insulin resistance, cerebrovascular disease, anxiety, and depression occurs.

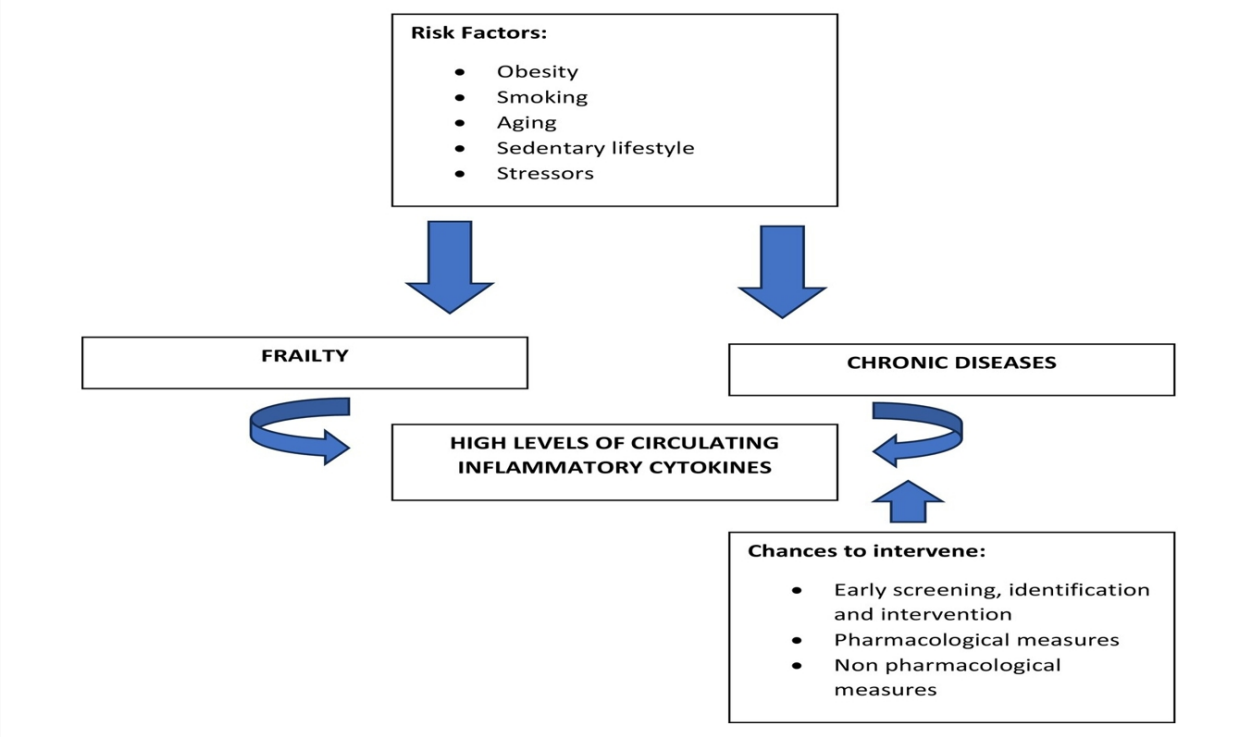


Fig. 1: Risk Factors for Frailty and Lifestyle Diseases

Inflammaging and Lifestyle Diseases

Inflammaging is an age related chronic pro-inflammatory state characterized by high circulating levels of pro-inflammatory markers, such as interleukin- IL-1, IL-6, IL-8, IL-13, IL-18, CRP, IFNα,IFNβ, TNF, TGFβ and serum amyloid A. which are involved in the pathogenesis of frailty and lifestyle diseases.

The Interplay between Key Lifestyle Diseases and Frailty

Hypertension

Arteriosclerosis, inflammation, and oxidative stress are processes involved in the pathophysiology of hypertension and frailty. Orthostatic hypotension in frail individuals can induce falls and fractures, leading to disability.

Cardiovascular diseases

Lifestyle risk factors and normal aging pathophysiological mechanisms are associated with increased risk of cardiovascular diseases and frailty. Conversely, cardiac and cerebrovascular diseases are associated with increased risk of frailty. Frail heart failure patients have more co-morbidities, inflammation, sarcopenia, and global dysfunction. In older adults with Acute Coronary Syndrome (ACS), frailty was associated with increased risk of cardiovascular diseases (CVD), major bleeding and hospital readmission.

Kidney disease

Frailty increases the risk of acute kidney injury and progression to end-stage renal disease and Electrolyte imbalance, volume-depletion, decreased salt and water intake. There is increased risk of harm from the effects of polypharmacy and use of NSAID. Factors that increase the progression of frailty in CKD include the presence of anaemia, uraemia, vitamin D deficiency, hypocalcaemia, undernutrition, and anorexia.

Diabetes mellitus

There is a shared pathophysiology among frailty and diabetes mellitus. Frailty is an independent risk factor for morbidity and mortality in older diabetes mellitus patients. Similarly, high blood sugar levels predict a transition to higher frailty levels. Also, vascular complications of diabetes mellitus and malnutrition lead to functional decline in frail older adults.

Anaemia

Chronic inflammation and frailty, Interleukin-6 correlates best with anaemia in several chronic disease states. Anaemia is associated with an increased risk of falls, impaired activities of daily living (ADL) and reduced strength, physical performance, mobility and cognition. Hence, reversing anaemia is an integral part of management in older adults living with frailty.

Interventions for Frailty and Chronic Diseases

Physical activity

Exercise prevents sarcopenia and in turn, helps to prevent frailty and its progression in old age. Exercise is associated with neurogenesis, release of vascular growth factors, reduction of oxidative and psychological stress, improvement in glycaemic control and reduced vascular risk factors. It is beneficial for maintaining health and reversing chronic lifestyle-related disorders.

Nutrition

Mediterranean-type diet, vegetarian and vegan diets are beneficial in prevention of lifestyle diseases. Protein intake of approximately 1 gm/kg/day beneficial for older adults to counteract age related sarcopenia. Caloric restriction and intermittent fasting have been shown to slow cell growth and metabolism, fat loss and weight loss, thus reversing the process of lifestyle disorders, chronic diseases, and frailty.

Interventions for Frailty in a Patient with Lifestyle Disease

Pro-active care

Frail older adults with lifestyle diseases present late and in crisis with acute issues. Timely identification of pre-frailty and frailty is therefore essential. Pro-active preventative care includes screening and treatment of chronic diseases as well as physical, nutritional, and psychological interventions. Treatable chronic causes of fatigue like depression, sleep apnoea, anaemia, hypothyroidism, vitamin B12 deficiency, and hypotension should be identified and treated.

Reduction of harmful polypharmacy

Lifestyle diseases leads to polypharmacy. Inappropriate prescription could be an exogenous stressor precipitating frailty status.

Conclusion

The causality and pathophysiological mechanisms behind frailty and lifestyle diseases share a common pathway and their progression is interlinked. Individuals must be screened to identify frailty and facilitate discussion around future treatment plans. Non-pharmacological lifestyle interventions play an important role in preventing these conditions.

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Hyperuricemia- A Growing Concern

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Kharghar Dispensary

Hyperuricemia or elevated serum uric acid (UA) has created a lot of interest among us. It is not just correlated with the traditional disorders like gout and nephrolithiasis but associated with numerous other chronic diseases like metabolic syndrome, hypertension, coronary artery disease etc.

Hyperuricemia is defined as serum uric acid level greater than 6.8 mg/dl. Beyond this level, there is supersaturation of bodily fluids and risk of precipitation.

UA is metabolized to allantoin and other metabolites via nonenzymatic oxidation and thus can function to neutralize prooxidant molecules such as hydroxyl radicals, hydrogen peroxide and peroxynitrite. Uric acid has both antioxidant effect on native low-density lipoproteins (LDL) and a pro-oxidant effect on mildly oxidized LDL. It has been suggested that the antioxidant effect of UA in humans contributes to neuroprotection in several neurodegenerative and neuroinflammatory diseases. However, despite the potential antioxidant effect of UA, numerous studies have revealed close association of serum uric acid concentration and various disorders of metabolic syndrome category. UA metabolism is a double-edged sword as regards to the inflammatory and/or oxidative response in many organs, perhaps its harmful effects appear to outweigh the benefits.

Hyperuricemia and Diet

Dietary purines are responsible for about 1/3RD of the body's daily serum uric acid production; the rest is synthesized from endogenous sources. Raised uric acid level can also be seen with increased purine degradation in high cell turnover states (e.g. tumor lysis, hemolysis, rhabdomyolysis) and with decreased excretion (e.g. genetic disorders, renal insufficiency, metabolic syndrome). About 2/3rd of its excretion is through the kidney and 1/3rd through the gastrointestinal tract. Studies have shown that prevalence of hyperuricemia increases proportionally with alcohol consumption in the male drinkers. It did not differ much among drinkers and nondrinkers in the female population probably due to estrogenic inhibition of renal

Dr Vaishali Wadhe

urate reabsorption with an increased renal urate clearance. Total protein intake is not independently related with hyperuricemia and surprisingly purine rich vegetables (peas, mushroom, lentils, spinach) and low-fat dairy products milk and yogurt are found to be negatively associated with uric acid levels. Sugar sweetened beverages (SSB) such as high fructose corn syrup used as an industrial sweetener has emerged as the contributory factor towards hyperuricemia. A recent area of study also revealed the interaction of SSB and specific genetic variations (SLC2A9 - Urate transporter) accounting for increased risk of developing hyperuricemia. The other consistent important association has been found is body bulk of an individual, which is independent of dietary intake. Body weight, height, BMI, BSA are all powerful predictors of the serum urate levels of an Individual.

Hyperuricemia and Its Role in Inflammation

There is increased xanthine oxidase (XO) expression in response to the amount of its substrate purine. Aging is another factor associated with elevated XO activity. Increased XO leads to increased superoxide free radical generation, activation of inflammasome like NLRP3(Nod Like Receptor 3), apoptosis associated speck like protein containing caspase recruitment domains (CARD), Caspase I and maturation of IL1, IL8. Chronic hyperuricemia can cause subtle renal injury including renal vasoconstriction, tubular ischemia, interstitial inflammation, persistent salt sensitivity, sodium retention and increased blood pressure. UA also activates vascular endothelial cells where it blocks

nitric oxide (NO) release, inhibits endothelial proliferation and stimulates C-reactive protein production. All these inflammatory changes have been demonstrated to be linked to the onset and progression of human diseases including Gout, Atherosclerosis, CKD and NASH.

Uric Acid and Lifestyle Disorders

High plasma uric acid levels are positively associated with increased incidences of hypertension in adults. However, this association decreases as patients age and is not found in elderly people. Damage of small renal vessels leads to irreversible salt sensitive hypertension. This hypertension persists regardless of uric acid levels. When hypertension develops in the elderly, other pathophysiological mechanisms such as decreased arterial compliance may play a larger role in hypertension than hyperuricemia. One study conducted on elderly participants, stated that undiagnosed hyperuricemia is a strong risk factor for resistant hypertension (>140/90) despite appropriate therapeutic approach and lifestyle modification. Hyperuricemia is also associated with insulin resistance and progression of type II diabetes mellitus. Persistent chronic hyperuricemia can manifest as a plethora of diseases if left untreated. Most patients are asymptomatic and do not need medical therapy. Regular screening will help establish appropriate timely intervention, early diagnosis and treatment. Dietary intervention should be prioritized and patient should be educated about a low salt, low fructose diet E.g. Mediterranean or DASH diet. Symptomatic patients should be treated for hyperuricemia with medical therapy and lifestyle modifications with defined end points.

Different Pharmacological Strategies to Reduce Serum Uric Acid

- Reducing uric acid production by inhibiting Xanthine Oxidase (Allopurinol and Febuxostat)
- Inhibiting UA reabsorption by targeting urate transporter 1 (such as Probenecid)
- Metabolizing circulating UA to allantoin by uricase (Rasburicase)
- Angiotensin receptor blocker losartan has been shown to lower UA level by inhibiting URAT1
- Anti-diabetic SGLT2 inhibitors (such as canagliflozin and empagliflozin) lower uric acid by interfering with GLUT 9(glucose transporter type 9).

Conclusion

Lifestyle disorders are on the rise although being easily preventable.Their early redressal is the need of the moment to avoid a global burden. Serum uric acid reflects the lifestyle choices we make with regard to our diet and alcohol consumption. The risk of hyperuricemia is positively correlated with intake of alcohol, red meat, seafoods, or fructose and negatively with dairy products or soy foods. High purine vegetables showed no association with hyperuricemia and gout. Considerable evidence suggests that people who adhere to a healthy lifestyle, such as avoiding obesity, moderate drinking, no smoking, a healthy plant based diet and regular physical activity have markedly reduced risk of hyperuricemia. Adopting a healthy lifestyle is the most cost-effective way to prevent hyperuricemia and related disorders in future.

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Childhood Obesity

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Introduction

The Covid-19 pandemic and the lockdown have changed the usual active lifestyle habits of children and adolescents. These changes in lifestyle behaviours have short and long-term cardio-metabolic and psychological health outcomes. The most observable outcome seen in children in post Covid times, is obesity.

Definition

Obesity is defined as an abnormal and/or excessive accumulation of fat that can impair health. Body mass index (BMI), defined as a person's weight in kilograms divided by the square of height in meters (kg/m2), is the most commonly used measure of overweight and obesity in adults and children.

The BMI categories for defining obesity vary by age and gender in infants, children and adolescents. For adults, WHO defines overweight and obesity as follows. Overweight is a BMI greater than or equal to 25 while obesity is a BMI greater than or equal to 30. For children, age needs to be considered when defining overweight and obesity.

For children under 5 years of age, overweight is weight-for-height greater than 2 standard deviations above WHO Child Growth Standards median; and obesity is weight-for-height greater than 3 standard deviations above the WHO Child Growth Standards median. For children aged between 5–19 years, overweight is BMI-for-age greater than 1 standard deviation above the WHO Growth Reference median; and obesity is greater than 2 standard deviations above the WHO Growth Reference median.

Although BMI is a reliable and easy-to-use tool, it may not correspond to the same degree of fatness in different individuals. For example, Indian adults are known to be more prone to abdominal obesity and a much higher rate of metabolic complications at lower BMIs. Hence, lower BMI cut-offs of 23 kg/m2 for overweight and 28 kg/m2 for obesity have been suggested for Indian adults. Similar findings of higher body fat for the same BMI were replicated in Indian children and adolescents when body fat

Dr Umesh Naiknaware

percentage was measured by alternate methods like bio-electrical impedance and dual emission x-ray absorptiometry.

Facts & Figures

Among children between 5-19 years, the prevalence of overweight and obesity rose from just 4% in 1975 to over 18% in 2016. These increased rates were similar in both boys and girls. In 2019, an estimated 38.2 million children under the age of 5 years were overweight or obese, and almost half of them lived in Asia.

According to the National family health survey-5 conducted during 2019-2021, the rates of under-five overweight (weight for age) increased from 2.1% in 2015-2016 to 3.4 % (urban- 4.2%, rural 3.2%) in 2021. It is predicted that with the current rate, India might be home to 27.48 million obese children between 5-19 years only second to China by 2030.

Causes of Obesity

Obesity can be classified into nutritional (or exogenous) and pathological obesity which occurs with underlying monogenic disorders or hormonal disorders.

Exogenous obesity is the commonest and is due to an imbalance between energy intake and energy consumption. There is normal growth, development and puberty. Secondary causes of obesity include drugs, neuroendocrine diseases (hypothalamic, pituitary, thyroid and adrenal disorders) and monogenic disorders. In monogenic obesity there is early-onset obesity (before 5 years of age) with extreme hyperphagia. Hypothalamic obesity is associated

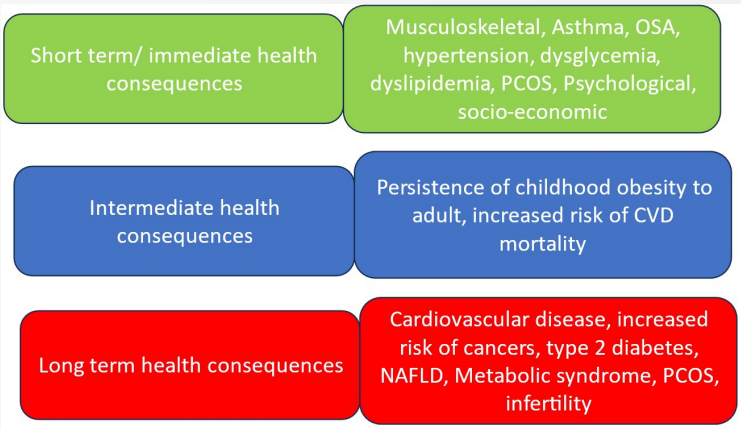

Fig. 1: Health Consequences of Childhood Obesity.

with neurological features like headache, irritability, seizures and/or neurological insult with rapid weight gain and hyperphagia. Neuroimaging is essential to identify a hypothalamic lesion. Drug-induced obesity is associated with glucocorticoids, antipsychotics (risperidone and olanzapine), and antiepileptic drugs (valproate and carbamazepine). Endocrine causes like hypothyroidism, Cushing syndrome, pseudohypoparathyroidism is associated with short stature which is the hallmark of underlying endocrine cause. Obesity causes mild elevation of thyroid-stimulating hormone (TSH) that is usually its effect and not the cause.

Effects of obesity on childhood [Fig. 1]

Health effects in an obese child have significant impact on their health as an adult. Being an obese child increases the likelihood of being an obese adult and there by leading to obesity related complications like increased risk of developing the metabolic syndrome, cardiovascular disease, type 2 diabetes, non-alcoholic fatty liver disease, obstructive sleep apnoea, polycystic ovarian syndrome, asthma, orthopaedic complications, psychiatric disease, etc. These complications are now becoming increasingly prevalent in children with obesity.

Psychological Consequences

Disorders of mood (anxiety), somatoform and eating disorders are detected more among children with obesity. There is a widespread stigmatization of children with obesity, which causes behaviour abnormalities like eating disorders, social isolation, declining academic performance and decreased physical activity in these children.

Physical Health Consequences

Physical complications of childhood obesity may not be immediately evident but they take decades to manifest. There is an increasing prevalence of gallstones, hepatitis, sleep apnoea and increased intracranial pressure among obese children.

Musculoskeletal Complications can manifest as Slipped capital femoral epiphysis and Blount's disease (Tibia Vara)

Pulmonary Complications

Increased frequency of asthma, decrease in exercise tolerance, abnormal sleep patterns, obstructive sleep apnoea are common.

Cardiovascular Complications

Overweight adolescents are at higher risk for developing hypertension during adulthood whereas 1/3rd of obese children can develop hypertension during childhood.

GI Complications

Obese children demonstrate evidence of steatohepatitis (by USG or elevated transaminases) and are prone to have gallstones.

Endocrine Complications

Insulin resistance, higher levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides have been noted in obese children. Overweight adolescents developed type 2 diabetes at an early age as compared to their lean counterparts. Hyperandrogenemia and menstrual abnormalities are more common in obese female children. Although early menarche is common in obese girls, delayed menarche, oligomenorrhoea, amenorrhoea is also been observed. Hormonal patterns of polycystic ovary syndrome have been increasingly observed in obese adolescents.

Elevation of C-reactive protein which is an inflammatory marker is observed in obese children which has been linked to increased incidence of cancers, autoimmune disorders and heart disease later in life.

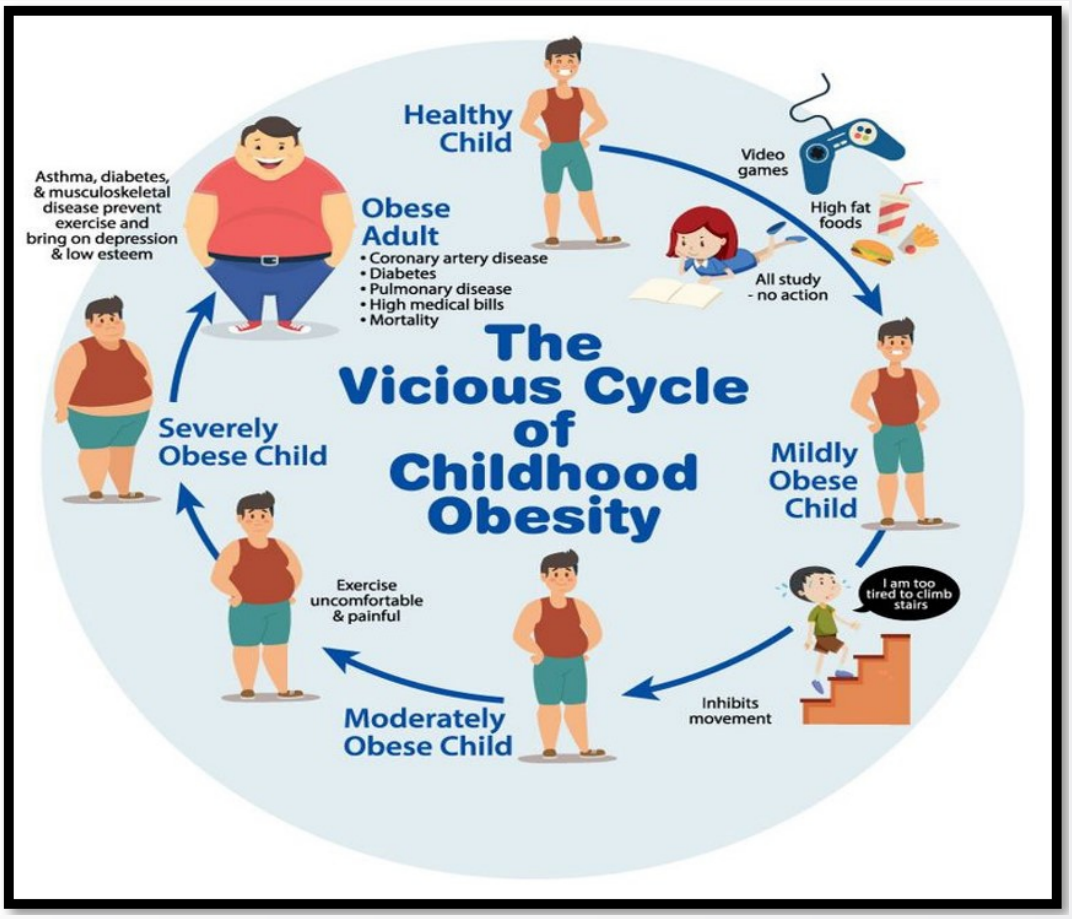

Fig. 2: The vicious cycle of childhood obesity

Intervention, Treatment & Prevention

A multidisciplinary approach with the main objectives of permanent change in the child's eating habits and lifestyle, maintaining mental health, treating/preventing the complications and preventing relapses should be undertaken rather than focussing just on attaining rapid weight loss which may affect nutritional status adversely.

Targets

Gradual and sustained weight loss with Body mass index (BMI) SD score (SDS) reduction of 5% which roughly translates to 7–10% weight loss over 6 months should be aimed. In any circumstances rapid weight loss of over 1.5 kg per month is avoided.

Diet

A balanced and varied diet is recommended to fulfil the National Recommended Energy and Nutrient Intake Levels, based on sex, age and ideal weight for stature (proteins 1 g/kg/day; carbohydrates 45–60% of total calories; simple sugars < 15% of total calories, lipids 20–35% of total calories starting from 4 years of age, saturated fatty acids < 10% of total calories)

The salient points in the diet management include

- Eat 5 meals a day (three meals and two snacks)
- Do not skip the breakfast.
- Increase intake of fruit, vegetables and fibre content (complex carbohydrates)
- Limit portions
- Avoid eating/grazing/snacking between meals
- Avoid high-energy and low nutrient density foods (eg. Sweetened drinks, fruit juices, fast food, high-energy snacks)
- Reduce saturated dietary fat intake for children and adolescents >2 years of age
- Recognize eating cues in the child's or adolescent's environment by the parent, such as boredom, stress, loneliness and screen time.

It is better to follow the traffic light diet, rather than eating

Table 1. Type of Physical Activity

	Preschool-Aged Children	School-Aged Children	Adolescents
Moderate intensity Aerobics	Follow the leader, Playing on a playground, Tricycle or bicycle, Walking, running, skipping, jumping, dancing, swimming catching, throwing, kicking	Brisk walking, Bicycle riding Swimming Baseball and Softball	Brisk walking Bicycle riding Swimming Baseball and softball, House work such as sweeping
Vigorous intensity Aerobics	Moderate and vigorous intensity are almost the same only the effort matters, Martial arts, Vigorous dancing		
Muscle strengthening	Tug of war Rope or tree climbing, Yoga		
Bone strengthening	Hopping, skipping, jumping, running		

replacement meals, hypocaloric diets with low glycaemic index/ low glycaemic load and very low caloric diet.

Traffic Light Diet Plan

Diet should be rich in 'green foods' or foods with low caloric density (fruits, vegetables, salads, low-fat dairy products, legumes, lean meats) and limited in foods with high caloric density or 'red foods' (fat-rich meats, fried foods, 'fast-foods', sugary beverages, sweets, fruit juices and canned food products). 'Yellow foods' (cereals, pulses, root vegetables, milk products) should be consumed in moderation.

Exercise

Physical exercise reduces cardio-metabolic risk factors. Change in body composition (especially fat reduction) rather than BMI reduction is the more effective way to evaluate the effectiveness of exercise. Obese children usually can't perform vigorous strenuous exercises, hence mild to moderate aerobic exercises can be started with and gradually the intensity can be increased. Children and adolescents should perform aerobic exercises for 60 min or more every day and muscle/bone strengthening exercise at least thrice a week as per the physical abilities of the obese child. Sedentary lifestyle and screen exposure has to be avoided.

Pharmacological intervention & Bariatric surgery

Pharmacological therapy can only be applied after the failure of the multidisciplinary lifestyle intervention only in children above 16 years. Orlistat was the only drug available

Fig. 3: '5-2-1-0 Rule' for Obesity Prevention

for the treatment of children and adolescents with severe obesity. In 2020 US-FDA (United States-food and drug administration) approved Liraglutide for the pharmacological management of obesity as adjunctive to diet and exercise for children above 12 years. However the medication is discontinued after 12 weeks if the reduction in BMI is <4%.

Bariatric surgery is the last resort solution in adolescents with severe obesity and resistant to all other treatments, especially when serious complications are present. The indications for surgery in the adolescent are: a) BMI ≥35 kg/m2 with at least one severe comorbidity (Type 2 Diabetes Mellitus, moderate -severe obstructive sleep apnoea, idiopathic endocranial hypertension, non-alcoholic steatohepatitis with significant fibrosis b) BMI ≥40 kg/m2 with less serious comorbidities.

Prevention

The best approach is to prevent the child from getting obese by adhering to following.

- Exclusive breastfeeding till 6 months of age.
- Regular meal timings, including breakfast.
- At-least 7–8 hours of sleep daily at night.
- Lifestyle intervention should precede and should be maintained lifelong.
- Obesity prevention guidelines from American Academy of Paediatrics recommend Fight Childhood Obesity by '5-2-1-0' rule.

To conclude, the effort to avoid obesity is the way forward beginning from the womb, tiding through the childhood and reaching the adulthood.

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Battling Bugs: Unravelling Infections through Diagnostic Microbiology.

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Introduction

Micro-organisms are ubiquitous. When these cause diseases in humans they are called pathogens. A pathogen can be fungus, bacteria, virus or parasite. From petri dishes to PCR, diagnostic microbiology unlocks microbial mysteries.

Processing of samples for microbiological tests

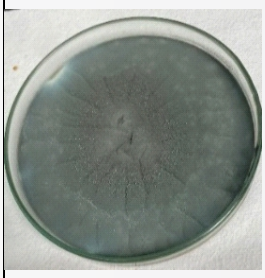
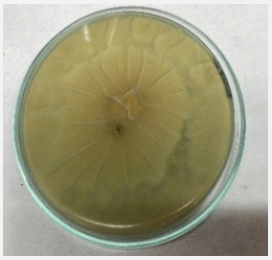
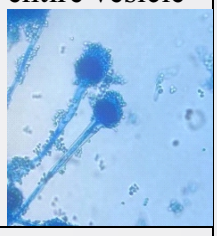
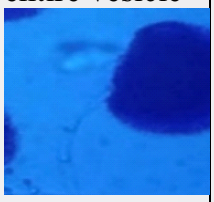
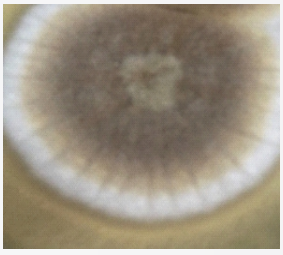
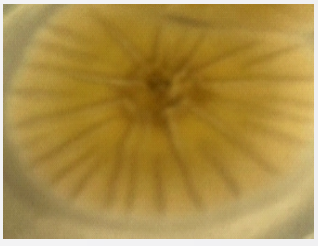
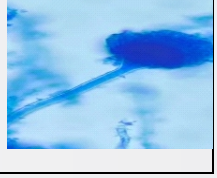
The range of diagnostic microbiological tests performed in our hospital include microscopic examination, serological, molecular and culture along with antibiotic susceptibility testing. Microscopic tests involve staining of a part of the sample and observing under the microscope for presence of cells, presence of organism especially its morphological appearance and staining characteristics. This can give an idea about the type of organism, thus contributing to presumptive diagnosis and start empirical antibiotic eg. presence of subterminal spores of Gram-positive bacilli indicates that the organism could be Clostridium and thus the decision of amputation can be made. [1] Culture helps confirm the findings of microscopy, gives a definite diagnosis and makes available colonies for antibiotic susceptibility . [1,2] Thus, it makes the invisible world of microbes visible. Antibiotic susceptibility leads the way for treatment and gives an idea about resistance pattern of the isolate like multi or extremely drug resistant (MDR/XDR). Serological tests like antigen tests are rapid and easy to perform. [1] They bridge our past immunity with our present health, revealing the footprints of battles fought within. Latest in the spectrum are molecular tests like Xpert/MTB Rif where rapid diagnosis meets drug resistance detection. It detects M. tuberculosis along with rifampicin resistance within two hours and is simple to perform. Rapid molecular testing systems for SARS-COV2 like True Nat have significantly contributed in the fight against COVID-19 pandemic.

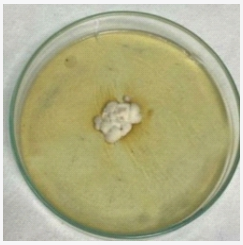
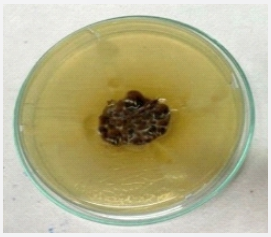
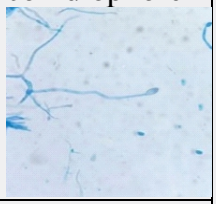
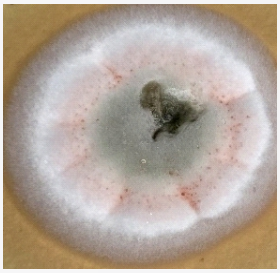
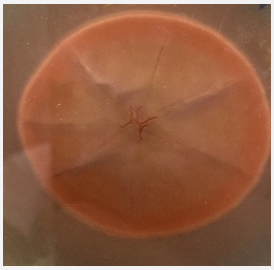
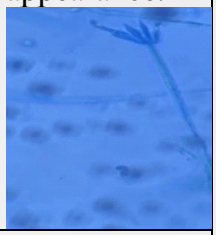
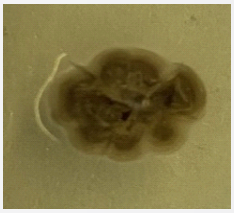
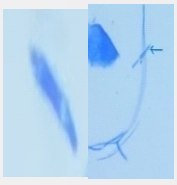
Dr Sunayana Jangla

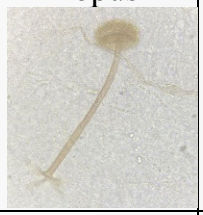
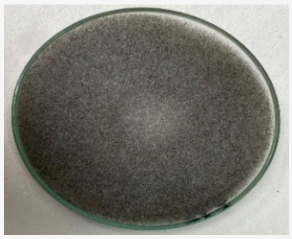
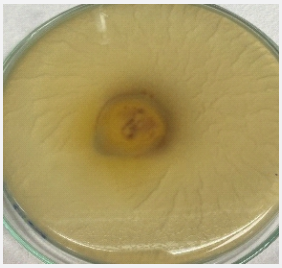
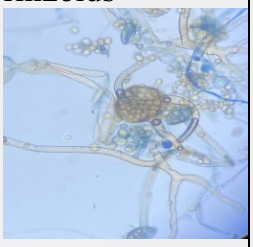
Contribution to patient management

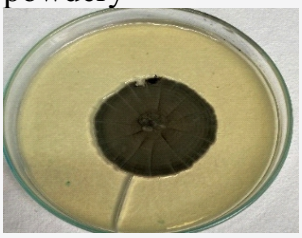
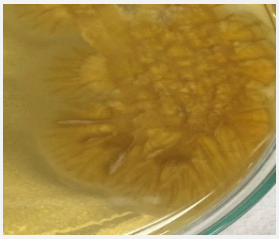
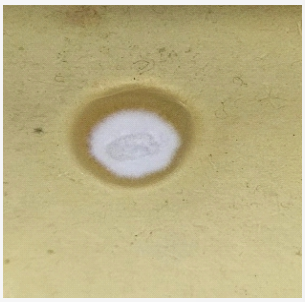
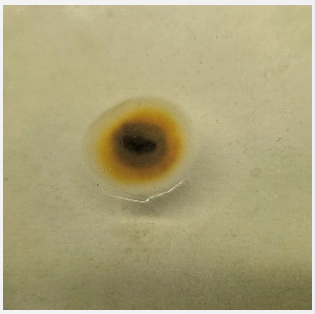
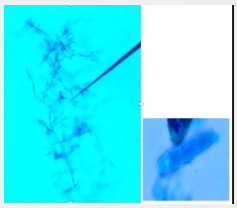
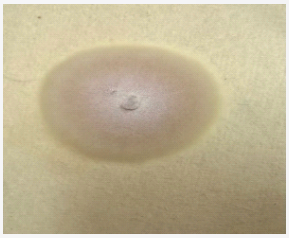
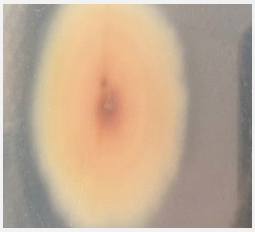
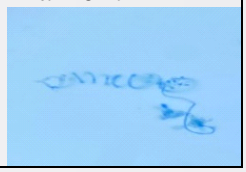
Below are some unusual pathogens reported from various samples of patients of our hospital which were received for microbiological investigations (culture and or microscopy) between January 2015 and May 2024. (Table 1) [1-4] Reporting these has aided in better patient management. Conservative management included administering appropriate antibacterial, antifungal or anti-parasitic drugs and managing the underlying co-morbidity. [1,3,4] eg glycaemic control in diabetics and additionally changing of intra-vascular device in case of perma-cath or shunt. Surgical management included debridement and amputation (in case of gas gangrene).

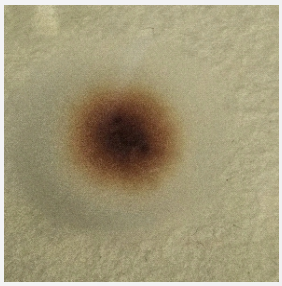
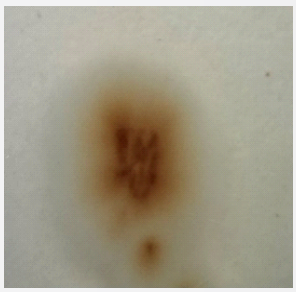
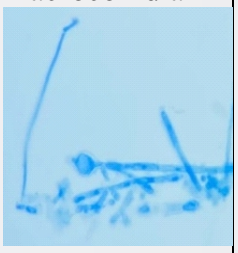
Table 1: Pathogens reported based on microscopy and culture from patient samples received in our hospital from January 2015 to May 2024.

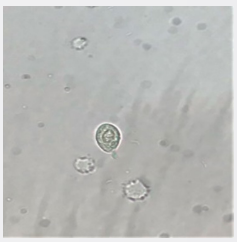
FUNGUS				
HYALINE FILAMENTOUS MOULDS				
Sample/Age (years)/ gender/ history	Name of the organism	Appearance on obverse (On plain Sabouraud Dextrose Agar AND/OR Sabouraud Dextrose Agar with antibiotics)	Appearance on reverse (On plain Sabouraud Dextrose Agar AND/OR Sabouraud Dextrose Agar with antibiotics)	Lactophenol Cotton Blue (LPCB) mount (x40) from growth
Sputum / 74/M /cough with breathles-sness. k/c/o bronchial asthma	Aspergillus fumigatus	Green suede-like 	Tan 	Septate hyphae with long conidiophores and phialides not covering entire vesicle 
Right nasal cavity/81/ M/ nasal crusting with history of COVID-19 positive	Aspergillus flavus	Greenish yellow powdery 	Tan 	Septate hyphae with long conidiophores and phialides covering entire vesicle 
Tissue from foot/58/M in c/o cellulitis/	Aspergillus terreus	cinnamon brown colony 	Tan 	Septate hyphae with phiallides in two rows, compactly columnar 

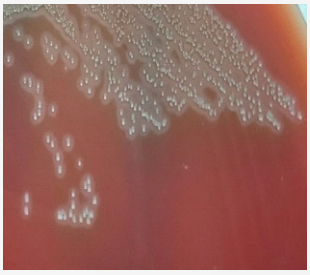
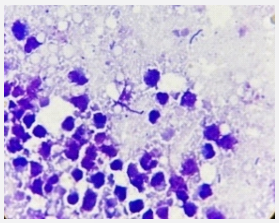
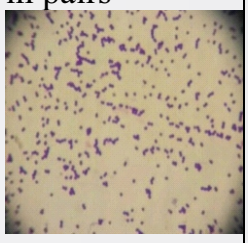
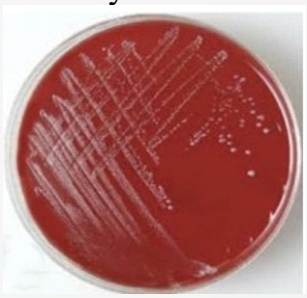
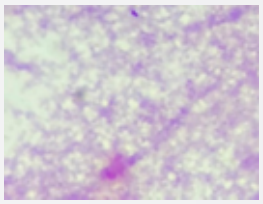
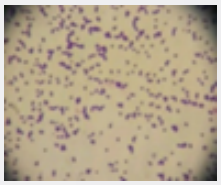
Pus from pre-patellar bursa/ 52/M/ swelling and redness of knee	Scedospor-ium apiosper-mum	Cottony white growth with suede like surface 	Black 	Septate hyphae with single conidium on tip of conidiophore 
Sputum/88 /M/ cough and breathles-ness	Talaromy-ces (Penicill-ium) marneiffii	Green coloured colony 	Red pigment 	Conidiophore s from branched meticulae giving brush-like appearance. 
Nail scraping/ 53/F/Toe nail	Acremo-nium kiliense (Non-Dermatoph ytic mould)	White cottony  No growth on media with cycloheximide	Brown 	Conidia (single, septate straight) Septate hyphae with conidia 

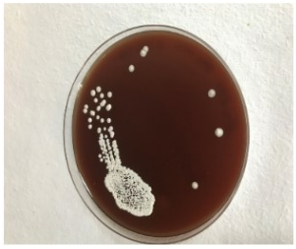
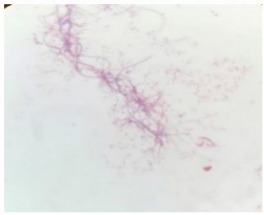
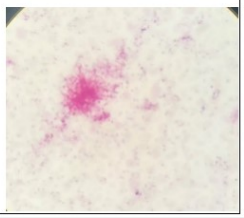
FUNGUS				
ZYGOMYCETES				
Para-nasal sinus tissue/ 76/M/ COVID-19 positive	Rhizopus species. Koh mount from sample showing Rhizopus 	Black greyish brown covering entire plate 	Tan 	Brown aseptate hyphae with root like structures called rhizoids 
Sputum /63/M/ cough with breathless-ness	Mucor along with Aspergillus flavus	Green powdery with black dense woolly growth 	Tan 	Broad aseptate hyphae with indistinct columella (Mucor) 

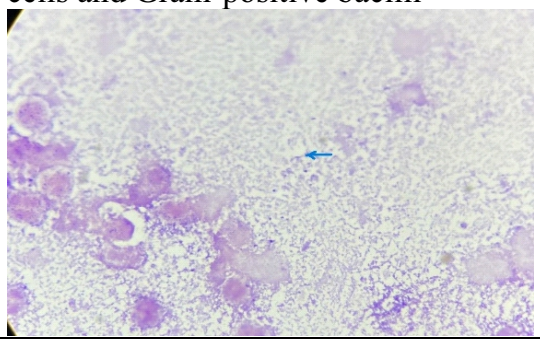
DERMATOPHYTES				
Nail clipping/ 56/M/disc olouration of nail	Epidermo-phyton floccosum	Khakhi coloured powdery 	Tan 	Large smooth-walled club shaped macroconidia 
Skin scrape from foot/ 70/M/ itching	Tricho-phyton mentagroph yte	Cottony white 	Yellow-brown pigment 	Septate hyphae with clusters of microconidia and cigar shaped macroconidia 
Skin scrape/ 63/M/ itching	Tricho-phyton mentagroph yte	Cottony white 	Buff-coloured 	Septate hyphae with clusters of microconidia and macroconidia with rat-tail filament 

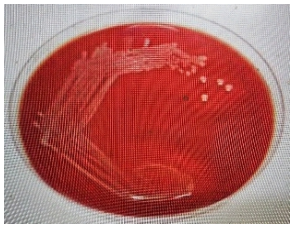
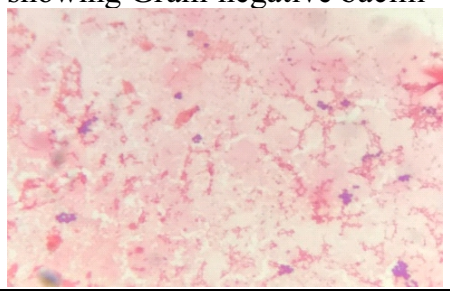
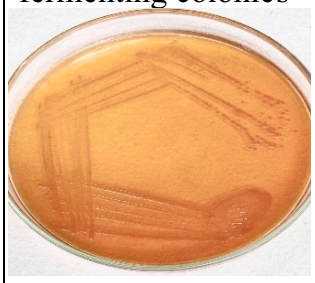
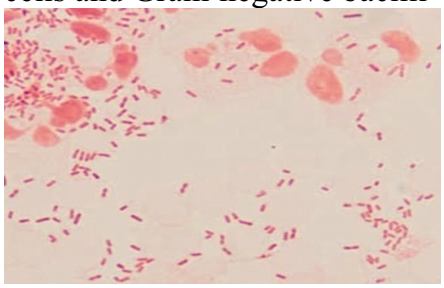
Skin scrape from limbs, back, gluteal region/ 72/M/ itching	Tricho-phyton rubrum	White downy with regal folds 	Velvety, red-pigment 	Septate hyphae with plenty tear-drop shaped microconidia along sides of hyphae and few long pencil-shaped macroconidia 
Skin scrape from face and left foot/27/F/ itching	Micro-sporum audouinii	Velvety brown 	Cinnamon 	Septate hyphae with bizarre shaped macroconidia 

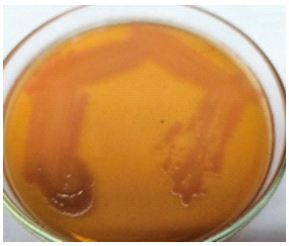
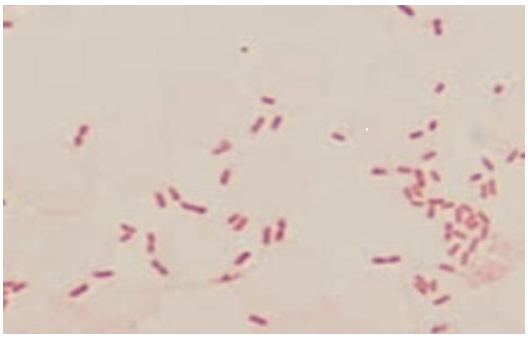
PARASITE (FLUKE)				
Urine/ 51/F /excretion of worms in the urine	Shistosoma (female counterpart)	Worm on Gross appearance	Worm end with sucker 	Egg with terminal spine (x40) 

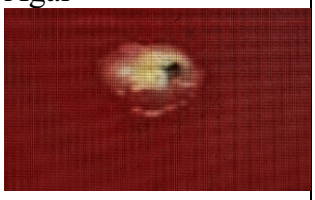
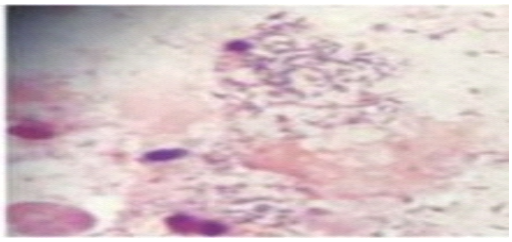
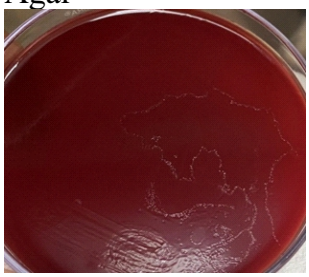
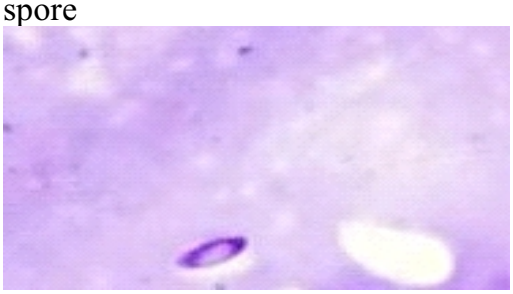
AEROBIC GRAM-POSITIVE COCCI				
Tissue and pus from bed sore/76/M	Enterococcus avium	Beta-haemolytic colonies on Sheep Blood Agar 	Gram stain from sample showing pus cells and Gram-positive cocci in pairs 	Gram stain from growth showing Gram-positive cocci in pairs 
Pus from spine (C2-C3)/20/F/ Neck pain radiating to right shoulder	Enterococcus casseliflavus (along with Xpert positive for MTB)	White beta-haemolytic colonies 	Gram stain from sample showing pus cells and Gram-positive cocci 	Gram stain from growth showing Gram-positive cocci in pairs 

AEROBIC GRAM-POSTIVE FILAMENTOUS BACILLI				
Pus from post-operative wound of right breast 61/F	Nocardia species	Dry white colonies on chocolate agar 	Gram stain showing Gram positive filamentous bacilli 	Modified Z.N stain positive 

AEROBIC GRAM-POSITIVE NON-FILAMENTOUS BACILLI			
Pus from exit site of perma-cath /82/M k/c/o End stage kidney disease on dialysis	Lysisnibacillus sphaericus	Dry white colonies 	Gram stain from sample showing pus cells and Gram-positive bacilli 

AEROBIC GRAM-NEGATIVE BACILLI			
Pus from leg foot ulcer 66/M k/c/o diabetes mellitus	Aeromonas hydrophila	BA: grey mucoid colonies 	Gram stain from sample growth showing Gram-negative bacilli 
Pus from ear discharge and consequent skull base osteomyelitis 72/M k/c/o diabetes mellitus	Achromobacter xylosoxidans	MacConkey agar: Non-lactose fermenting colonies 	Gram stain from sample showing pus cells and Gram-negative bacilli 

NON-FERMENTER GRAM-NEGATIVE BACILLI			
Blood 24/F c/o meningomyelocele with V-P shunt	Burkholderia cepacia group	MacConkey agar: Non-lactose fermenting colonies 	Gram stain from growth showing Gram-negative bacilli 

ANAEROBIC GRAM-POSITIVE BACILLI			
Pus from lower jaw/78/F/lower jaw swelling History of trauma to left side of tooth present	Actinomyces species	Colony with Molar-tooth appearance on Anaerobic Blood Agar 	Gram-stain from sample showing Gram-positive branching filamentous bacilli 
Tissue from left lower limb/ 67/M/ History of trauma present	Clostridium species	Swarming on Anaerobic Blood Agar 	Gram stain from sample showing Gram-positive bacilli with sub-terminal spore 

Conclusion

This article illustrates how tests like routine smear examination by Gram stain or KOH mount (microscopic tests) and culture when ordered have yielded unexpected pathogens and helped to initiate specific treatment in every case. Awareness of these diverse pathogens is essential for better clinicopathological correlation and effective treatment. Timely and accurate microbiological insights enhance patient care and contribute to better outcomes.

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Role Of Diet In Childhood Obesity

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Introduction

Criteria for defining obesity is different for adults and children. According to the Centre for Disease Control (CDC), obesity in adults is defined as a body mass index (BMI) of 30.0 or higher. BMI is calculated by dividing weight in kilograms by height in meters squared.

For children aged between 2 and 19 years, BMI is based on age- and sex-specific percentiles from the CDC Growth Charts and BMI at the 95th percentile or higher is considered obese.

Obesity affects 380 million children and adolescents worldwide. Low and middle-income countries are the most affected regions worldwide. If current trends persist, India will contribute approximately 11% of the global burden of child obesity by 2030. [1] India could be facing an obesity epidemic with alarm bells ringing particularly for the young. A new global analysis, published by The Lancet, found that 12.5 million children (7.3 million boys and 5.2 million girls) in the country, aged between five and nineteen, were grossly overweight in 2022, up from 0.4 million in 1990. [2] Childhood Obesity has thus become a global Pandemic in developed countries leading to a host of medical conditions that contribute to increased morbidity and premature death. [3]

Genetic factors

Genetic inheritance influences 50-70 percent a person's chance of becoming obese. If both parents are obese the chance of obesity in children is 80 percent.

Age and sex

It can occur at any age and in either sex as long as the person is in a positive energy balance.

Total intake of calories rather than the frequency of meals causes obesity. However, eating between meals can also contribute to obesity (except low calorie fruits and vegetables). Bottle fed infants are more likely to be obese than breastfed infants if the formula is prepared with packed or heaped scoops [4]

Smt. Ambika R

Physical activity

When physical activity decreases and food consumption either remains the same or increases with improved economic status can lead to obesity

Endocrine factor

Dysfunction of the thyroid and pituitary may result in obesity.

Trauma

Obesity may follow damage to hypothalamus after head injury due to dysfunction in regulation of appetite or satiety.

Psychological Factors

For people who are bored, lonely, discontented or depressed, food becomes the focal point, leading to obesity.

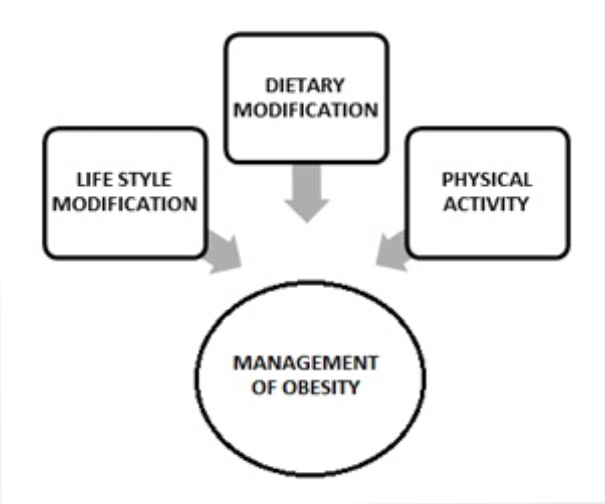

Fig. 1: Management of Obesity

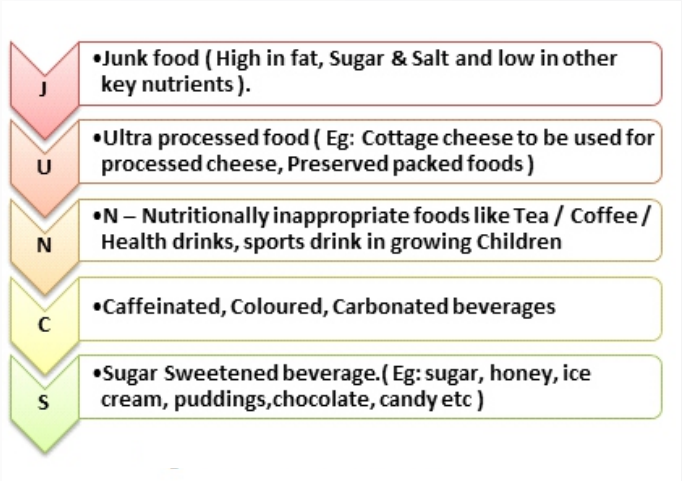

Fig. 2: JUNCS to be avoided

Principles of Dietetic Management

Low calorie, normal protein, restricted carbohydrate, restricted fat, adequate vitamins & minerals, high fiber and adequate intake of fluids.

Energy- 20Kcal/Kg IBW is prescribed.

Protein- 1gm /kg/IBW

Fat- 20-35% of total calories in which, <10% of Saturated fatty acid.

Carbohydrate -45-60% of total Calories.

Vitamins: With prolonged restriction of fats, there is likely to be a deficiency of fat-soluble vitamins A and D which should be supplemented.

Minerals: Sodium intake is to be restricted as excess sodium predisposes to retention of fluid.

Fluid: If salt is restricted then fluids can be taken liberally as extra fluids are excreted by the healthy kidneys. A glass of water taken before meals may help to cut down the intake of food.

High fibre: High fibre, low calorie food like green leafy vegetables, fruits, vegetable salads, whole grain cereals and pulses can be included in the diet.

DIETARY GUIDELINES

Thumb rule: JUNCS to be avoided

1. Don't skip breakfast
2. Eat five meals a day. Limit portion intake.
3. Increase intake of fibre, fruits and vegetables inform of soup and salad
4. High calorie fruits and vegetables like mangoes, bananas, potato, beetroot and carrot should be taken in limited amount.
5. Lean mutton/chicken/fish should be steamed and not fried.
6. All fried foods like puries, parathas etc., should be avoided.
7. Be sure your child gets enough sleep. Too little sleep may increase the risk of obesity. Sleep deprivation can cause hormonal imbalances that lead to increased appetite.

PHYSICAL EXERCISE

A low-calorie diet accompanied by moderate exercise is effective in causing weight loss. Aerobic exercises for 15-30 minutes in which speed and resistance is constant for eg. In walking, running and swimming can be performed as it directly increases the daily energy expenditure and is useful in long term weight maintenance. Further 50% of glucose and 50% of fat is metabolized to give energy when such exercise is performed. Exercise also preserves lean body mass and prevents the decrease in basal energy expenditure.

BEHAVIOR MODIFICATION

Behavior modification program must focus on three components: self monitoring, stimulus control and techniques for self-reward.

Self-monitoring: A daily self-dietary history record of food intake to identify physical and emotional settings in which eating occurs.

Stimulus control involves recognize eating cues in the Children by parents, such as boredom, loneliness, stress and screen time.

The last component is self-reward for eating control.

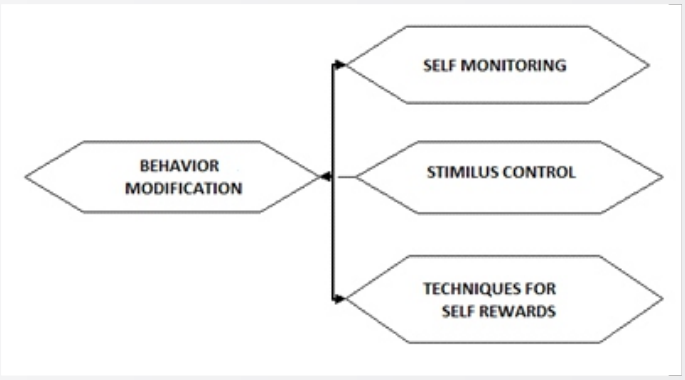

Fig. 3: Behavior Modification

Fig. 4: Replace Junk food by Healthy Diet

LOW CALORIE RECIPES FOR CHILDREN:

1) MILLETS VEG UPMA

Ingredients: -Millets, mixed vegetables (carrot, peas beans, onion, tomato), cumin seed, salt oil

Method: - Roast the millets and soak for 15 minutes, take pressure cooker, heat oil, add cumin seed and add all vegetables sauté for 2-3 minutes, add millets and water with salt. Cook for 2 whistles.

2) SPROUTS CHILA

Ingredients: - Sprouts, green chilli, ginger, salt

Method: -Blend into batter with ginger, Coriander and chili, cook on pan like pancake, garnish with beetroot, carrots etc., serve with green chutney/ Curd.

3) RAGI IDLI / DOSA

Ingredients: - Ragi flour, urad dal, fenugreek seed, salt

Method: -Soak urad dal and fenugreek seed, grind to a batter, mix with ragi flour. Ferment overnight. and Steam

4) OATS KHICHADI

Ingredients: - Oats, moong dal, mix vegetables (carrot, peas beans, onion), salt

Method: -Roast oats, cook moong dal in pan sauté cumin seeds, add vegetable, cooked dal and oats, cook together with water and salt

5) MILLET CUTLET

Ingredients: -Millet, mix vegetables (carrot, peas beans, onion), salt, grinded roasted oats, Gram Flour

Method: - Rinse, clean, peel, chop, steam and smash all mix vegetables. Soak millets and grind it. Mix all ingredients and make a thick patty. Lastly heat it in one spoon of oil for shallow frying in wide pan or Preheat the oven to 180C/ 360C for 15 minutes and placed the patties on greased tray. Serve hot.

6) KETO BURGER

Ingredients: -Paneer, cabbage, onion, tomato, capsicum, cucumber, lettuce, red chili flakes, oregano, green chutney, salt,

Method: -Cut paneer into cubes. Take a hot pan add one teaspoon of oil. Add oregano, red chili flakes. Next add paneer and sauté it and remove from the stove. Take cabbage whole leaves. Add green chutney and place paneer and rest of the vegetables, hung curd and cover it with the cabbage leaves. Serve it.

7) BADAM PISIN LEMONADE

Ingredients: Badam gum (soak over a night), Lemon juice, salt, Pudina leaves

Method: Cut lemon and squeeze the juice in a glass. Add water, salt and chopped or ground Pudina leaves with Badam gum. Serve it.

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3. Dietary Interventions to prevent Childhood Obesity, Ana Rita Pereira et.al Nutrients 2021, PMC.
4. Clinical Dietetics and Nutrition, Fourth Edition, Authors: F.P. Anita & Philip Abraham

ACADEMIC ACHIEVEMENTS

Publications

1) Bhonsle K, Dalal K.S, Bhirud P.H. Role of Ultrasonographic Assessment of Internal Jugular Vein Distensibility in the Prediction of Hypotension Following Spinal Anaesthesia: An Observational Study. Int J Sci Res. 2024;13 (4):16-18.
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4) Shrividya Chellam, Kajal Dalal, Pratibha Toal. Anaesthesia Management of a Morbidly Obese Patient in a Non bariatriac Setup Using HFNO: A Case Report.
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6) Veena R Karkhele, Deepanjali Patankar, Sayali Bhedasgaonkar, Snehal Nadkarni; An Analysis of Tear Film Secretion and Stability in Patients Undergoing Phacoemulsification and the Effect and Need of Lubricating Eye Drops Used Postoperatively; International Journal of Pharmaceutical and Clinical Research
   2023; 15(9); 862-870.

Paper and Poster Presentation

1) **Ranjit Kaur Dhanjal,** Nurse (D) successfully completed Master of Business Administration (MBA) in Health Care Management from Suresh Gyan Vihar University (S.G.V.U.), Jaipur. As a part of the training, she completed a thesis on "Perception of Burden by Caregivers of Dementia Patient and its Association with Severity Of Dementia". This research was conducted at BARC Hospital with Dr Aditi Chaudhari, consultant psychiatrist as project guide. The paper from this thesis titled "Perception of Burden by Caregivers of Dementia patient and its Association with Severity of Dementia" was shortlisted for oral presentation at the 78th National Conference of the Trained Nurses Association of India, held at Chandigarh in December 2024. It was presented by Smt. Ranjit Kaur Dhanjal on 7th December 2024 at the conference.

ACADEMIC ACHIEVEMENTS

2) A paper on 'Health Related Quality of Life of Patients with Advanced Cancer in an Urban Healthcare Center' by Dr Debjani Pal, Dr Sonali Shejul was presented by Dr Debjani Pal at 52nd Annual Conference of General Practitioner's Association, Greater Mumbai at Jamnabai Narse Monjee School, Mumbai on 6th January 2024 which received Best Clinical Paper Award .

3) A paper on 'Leveraging Aspirates for Enhanced Diagnostics: Evaluation of Scrape Cell Blocks as Adjunct to Breast Fine Needle Aspiration Cytology' by **Dr Shifa Khan** won the first prize at MaCyCon January 2024 at Nagpur.

4) A paper on 'Application of Milan System for Reporting Salivary Gland Cytopathology and Histological Correlation' by **Dr Shifa Khan** won the first prize at MAPCON September 2024, Nagpur.

5) A paper on 'Deciphering the Diagnostic Puzzle Of Breast Papillary Lesions by Integrating International Academy of Cytology (IAC) Categorization and Cytomorphological Features' was presented by Dr Shifa Khan at Midyear CME in Pathology May 2024, Pune

6) A paper on 'Retrospective Observational Evaluation of the Brandwein-Gensler Risk Model for Assessing Outcomes in Oral Squamous Cell Carcinoma' was presented by Dr Jyoti Gupta at MAPCON September 2024, Nagpur.

7) A paper on 'Demystifying Grey Zone Lesions in Breast Cytology: Unraveling Diagnostic Ambiguities to Enhance Risk Stratification' was presented by Dr Jyoti Gupta at MACYCON January 2024, Nagpur

8) A poster on 'Presbyopia: Current Scenario of the Amount of Acceptance in Industrialized Societies' by Shri Jeewan Prakash Srivastava, Dr S.U. Nadkarni, Dr S. Bhedasgaonkar, Dr Divya Gupta was presented by Shri Jeewan Prakash Srivastava at the 3rd International Optometry Conference ASCON Novemeber 2023 in Karnavati University, Gujarat.

9) A poster on 'Acceptance, Commissioning and Quality Assurance of a 1.5T MRI Machine' by Shri M. Kumaresan, Dr Ajay Chaubey, Dr Surita Kantharia, Dr Shubhra Gupta was presented by Shri M.Kumaresan at International Conference On Medical Physics December 2023 at DAE conventional centre, Mumbai.

10) A poster on 'Low Grade Intraductal Papillary Mucinous Neoplasm of Salivary Gland: a Diagnostic Challenge on Cytology' was presented by Dr Shifa Khan at MaCyCon January 2024 at Government Medical College, Nagpur

11) A poster on 'Tell Tale of Multiple Myeloma: Unfurled Saga through Cytology of Scalp Swelling' was presented by Dr Jyoti Gupta at MaCyCon 2024 at Government Medical College, Nagpur

12) A poster on 'Postpartum Uterine Inversion' presented by **Dr Pooja Rathod** won the First prize in a poster competition at 27th NMOGS annual conference, Navi Mumbai

ACADEMIC ACHIEVEMENTS

13) A poster on 'Heterotopic Pregnancy' presented by **Dr Sudarshan Lugade** won the second prize in a poster competition at 27th NMOGS annual conference, Navi Mumbai in November 2024.

Dr Sheetal Chiplonkar and Dr Jalpa Kate completed online fellowship in 'Musculoskeletal Ultrasound in Pain Medicine' organised by Daradia Pain Clinic, Kolkata in Oct 2024.

Candidates passing DNB exam in 2024.

Congratulations!!

Dr Palash Bauri
Department of Ophthalmology

Dr Thankuraj Rajan
Department of Anaesthesia

Extracurricular Achievements

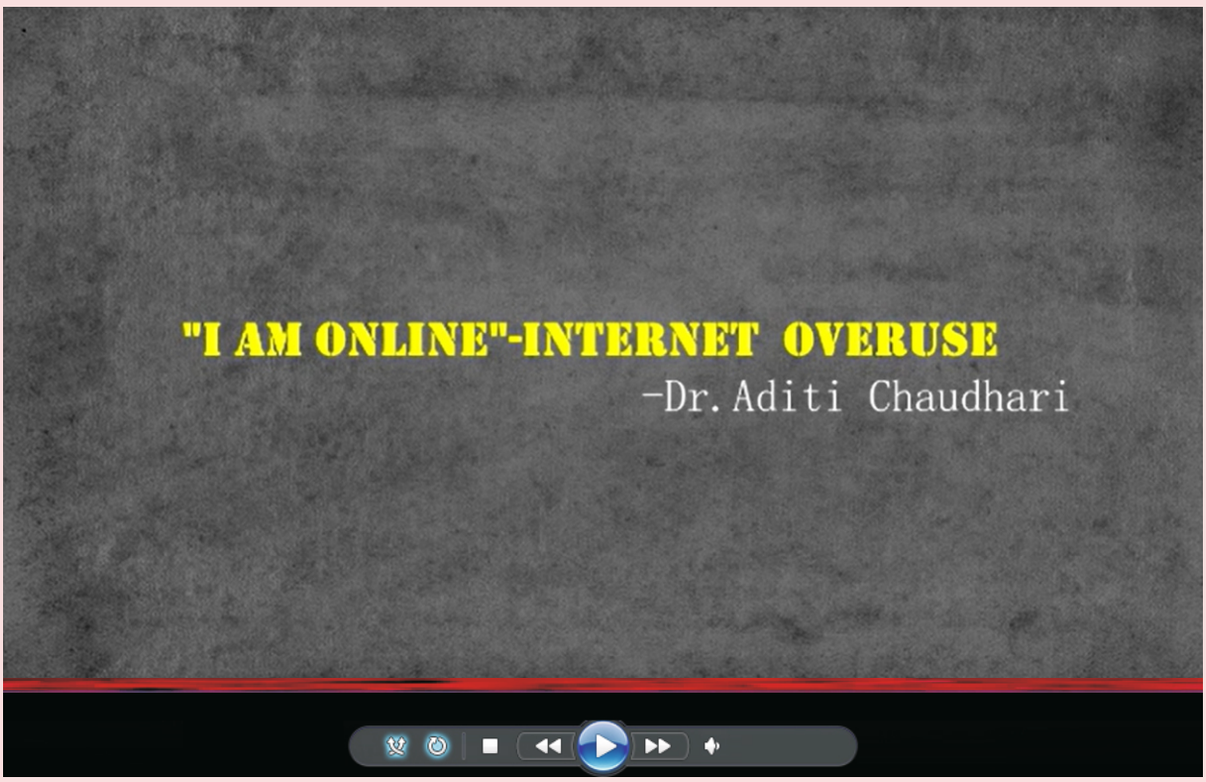
Dr Pratibha Toal, Head, Anaesthesia Unit and MOIC –II, finished Navy Mumbai Half Marathon (21km), 2024.

Dr Shrividya Chellam, Anaesthesia unit and MOIC Casualty unit, received 3rd prize in Chinmaya Mission Bhagavad Gita chanting competition, Mumbai 2024.

Educational Video

A poem by Dr Aditi Chaudhari, Department of Psychiatry, BARC Hospital (Illustrations by Br Jayesh Panchal)

To view, click here.



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