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World Heart Day 2023

'Use Heart, Know Heart'

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YOUR HEALTH

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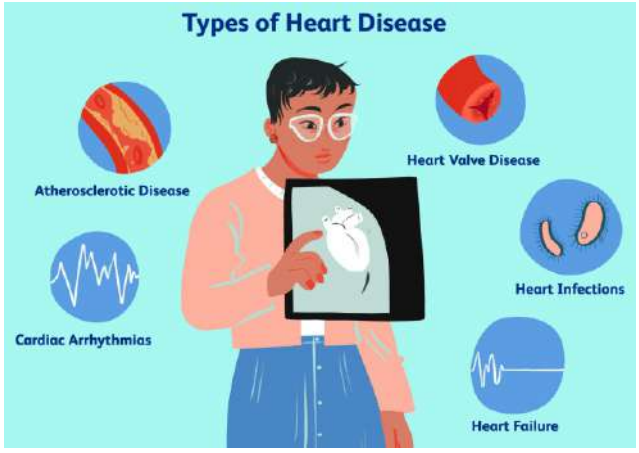
Stop the World's Biggest Killer

Editorial

Dr. Kakali Sen
Hony. Editor, Your Health

Cardiovascular disease (CVD) is the world's number one killer. Combined, conditions affecting the heart or blood vessels – such as heart attack, stroke and heart failure – kill more than 20.5 million every year. The majority of these deaths happen in low- and middle-income countries.

We want to bring this number down – way down. And there's hope: 80% of premature deaths from CVD are preventable. By making small changes to our lifestyle – what we eat and drink, how much we exercise, and how we manage stress – we can better manage our heart health and beat CVD.



World Heart Day is a Global, Multi-Lingual Celebration

Spanning six continents, our hundreds of World Heart Federation (WHF) member organizations, the countless schools, universities, sports clubs and the vibrant cardiology community make World Heart Day (WHD) a truly global celebration.

Every year these groups and individuals bring their local flair, favor and colors to festivities, marking the day by sharing heart healthy regional specialties, leading a dance to get the whole community moving, and sharing life-saving lessons far and wide.

Campaign Support for Companies, Schools, Organizations & Clubs

Part of the fun of celebrating World Heart Day is making the day your own –deciding how you or your organization wants to contribute to making a real difference. This could be a 5K run for everyone at your company, a heart-healthy menu in the school cafeteria or a CPR class for your sports club. No matter what you decide, you can find help and resources on the World Heart Day website, including a toolkit to make communicating and organizing your event a success.

From the Desk of Secretary

Dr. Samarendra Kumar Basu
Hony. Secretary, Your Health

The World Heart Day provides an opportunity to raise awareness about heart health and accelerate actions to prevent, detect and manage cardiovascular diseases.

The WHO South-East Asia Region is home to a quarter of the world's population. The region is experiencing a very high burden of noncommunicable diseases (NCDs), and cardiovascular diseases (CVDs) are responsible for 3.9 million annual deaths, making up 30% of all deaths. Alarmingly, almost half (48%) of these CVD-related deaths occurred prematurely, affecting individuals aged 30 to 70 years and imposing significant socioeconomic burdens on families, communities, and countries.

Main causes to the burden of CVD include modifiable lifestyle factors such as tobacco use, alcohol consumption, unhealthy diets especially high salt intake, and lack of physical activity. Raised blood pressure and raised blood glucose levels are key drivers and they can be detected, diagnosed, and managed adequately in primary care. One in four adults in the region has raised blood pressure, while one in ten has diabetes, and less than 15% are on effective treatment coverage. Additionally, high levels of lipids in the blood and suboptimal management of acute cardiovascular events further worsen CVD mortality.

In response to this significant public health importance, NCDs has been identified as a Regional Flagship priority since 2014. In 2022, the region has adopted the 'Implementation Roadmap for accelerating the prevention and control of NCD in South-East Asia 2022–2030.' The SEAHEARTS (WHO HEARTS package adaptation to South-East Asia Region) initiative of the region, brings together measures to reduce risk factors (tobacco control, salt reduction, and trans-fatty acids) with improvements in hypertension and diabetes coverage and control in primary health care. SEAHEARTS resonates with the World Heart Day 2023 theme 'Use Heart, Know Heart' and offers countries a roadmap to scale up their current situations and align their actions within the broader NCD prevention and control efforts.

Tobacco use prevalence in the region is declining due to the implementation of WHO Framework Convention on Tobacco Control measures. Bangladesh, India, Sri Lanka, and Thailand have taken steps to eliminate trans-fatty acids from their national food supplies, potentially benefiting over 1.7 billion people. India's target of reaching 75 million people with hypertension and diabetes under standard care by 2025 is the largest cover of NCDs for primary health care in the world. Accelerating the control of CVDs is a priority and WHO South-East Asia Region is calling for action in four key areas. First, countries need to place CVD high on their agenda and expand their efforts through commitment and leadership at both policy and programmatic levels. Second, continue implementing evidence-based tobacco control laws in line with the WHO Framework Convention on Tobacco Control and its MPOWER package across all countries. Third, promote healthy diets with a specific focus on salt reduction and eliminating trans-fatty acids by implementing WHO SHAKE and WHO REPLACE technical packages. Fourth, scale up programs and service delivery models that improve the detection, diagnosis, and management of hypertension and diabetes in primary health care with referral mechanisms.

Each of us can make a meaningful difference within our capacity and can collectively contribute towards SEAHEARTS that holds the potential to save lives and improve the well-being of millions.

*Extracted from the message of Dr Poonam Khetrapal Singh, WHO Regional Director for South-East Asia

Cardiac Arrest : Resuscitation and after

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Knowledge about Cardiac Arrest and the technique of cardio pulmonary resuscitation is important not only for cardiologist or intensivist but for all branches of medical science. CCU may be the best place to resuscitate a cardiac arrest (CA) but often the event occurs in unmonitored zones . Only a third of cardiac arrest outside , are resuscitated to be admitted in hospital . Although there is no specific survival data about arrest in CCU , as low as 12% of out of hospital cardiac arrest (OHCA) and 24% of in-hospital cardiac arrest (IHCA) are discharged .

Adult chain of survival

- Recognize Symptoms
- Early CPR
- Defibrillate with AED
- Advanced Life Support
- Post Cardiac Arrest Care

Post cardiac Arrest Syndrome (PCAS)

PCAS is a pathophysiological complex arising from after effects of cessation of circulation during CA and consists of 4 components :

- Anoxic Brain Injury (BI)
- Systemic Ischemia- Reperfusion Injury (IRI)
- Post Arrest Myocardial Dysfunction (PAMD)
- Persistant Precipitating Pathology

The PCAS consists of the following phases :

Immediate phase – 0-20 mins after return of spontaneous circulation

Early Phase : 20 min - 12 hours

Intermediate Phase : 12 hours – 3 days

Late Phase : 3 days onwards

The components of Cardiac Arrest care

- During Arrest before Restoration of Spontaneous Circulation (ROSC)
- Cardiopulmonary Resuscitation (CPR)
- Defibrillation / electrical management (temporary pacing)
- Airway Management and Ventilation
- Drug therapy – Antiarrhythmic and vasopressors

After Arrest after ROSC

- Cautious Fluid repletion
- Vasopressors for maintaining MAP 70 mmHg
- If comatose -Temperature Targeted Management (TTM)/ hypothermia
- Optimize Organ perfusion – Assessing use of Dobutamine
- Mechanical Circulatory Support : IABP , VA-ECMO Appropriate diagnostic tests : ECG , early Angiography if STEMI suspected
- Appropriate Prognostic tests : EEG , CT Brain for BI evaluation .
- De-escalation of therapy in selected cases

The principles of CPR

- Closed-chest cardiac massage (chest compressions) can produce enough forward blood flow to the brain and organs to decrease the extent of ischemic injury and to delay metabolic deterioration.

- Change from ABC (airway , breathing , circulation) to CAB (Compression , airway , breathing) – early initiation of chest compression have improved outcomes . Start CPR within 10 seconds .
- High-quality chest compressions (Fig 1) are defined as a rate between 100 and 120 compressions per minute with a depth of at least 2 – 2.5 inches in adults, allowing full recoil. Compressions faster than 120 per minute may not allow for cardiac refill and reduce perfusion. Chest compressions should be delivered to children (less than one year old) at a depth of one third the chest, usually about 1.5 to 2 inches (4 to 5 cm). Use the heel of one hand on the lower half of the sternum in the middle of the chest , keeping other hand over the top of first hand .Do not lean on chest in between compression as it prevents recoil .
- Interruptions of chest compressions, including pre- and post-AED shocks should be as short as possible.
- Compression to ventilation ratio remains 30:2 for an individual without an advanced airway in place. For those with an airway one ventilation every six seconds .
- Biphasic defibrillators are more effective in terminating life-threatening rhythms and are preferred to older monophasic defibrillators.
- Standard dose epinephrine (1 mg every 3 to 5 min) is the preferred vasopressor .
- After 30 compressions, stop compressions and open the airway by tilting the head and lifting the chin with index and middle fingers to give rescue breaths (except in case of neck injury) . Observe for rise of chest .
- In two rescuer CPR with bag and mask ventilation , the second rescuer holds the bag-mask with one hand using the thumb and index finger in the shape of a “C” on one side of the mask to form a seal between the mask and the face, while the other fingers open the airway by lifting the person's lower jaw .
- If pulse is palpable after initial CPR , one ventilation is given every 6 seconds . If pulse is still not palpable , CPR continues for 2 minutes and then rhythm is assessed and the cycle continues . If etCO2 during CPR < 10 mmHg , vasopressors should be added .

Use of Automatic External Defibrillator

- Oxygen tubing should not be across patient's chest while defibrillating .
- Caregivers must stay away from the patient during shock delivery .
- Adult / paediatric pads are to be placed on bare chest with jelly provided .
- One pad on upper right side and the other on the chest a few inches below the left arm.
- AED reads rhythm then gives : in VF defibrillation (unsynchronized) shock (Fig 2),in VT (synchronized) shock . For biphasic defibrillator 200 joules and for monophasic one 360 joules are usually recommended .
- If no pulse post shock , resume CPR immediately .
- In patients with electronic device , keep pads away from the pulse generator .

The ACLS survey

- Airway : Advanced airway in hospital setting .
- Breathing : Give 100% O2 , keep SpO2 > 94% . High-quality CPR should produce a CO2 between 10 to 20 mmHg. If the ETCO2 < 10 mmHg after 20 minutes of CPR for an intubated individual, then you may consider stopping resuscitation attempts.
- Circulation : Use intravenous or intraosseous route to give drugs .
- Differential Diagnosis : Rapidly diagnose cause of CA .

The drugs used in resuscitation

Amiodarone 300mg (5mg/kg)
Lidocaine 100mg (1.5 mg /kg)
Epinephrine 1 mg
Vasopressin

The principles of airway management

Proper bag mask ventilation in earlier stage of cardiac arrest may be more effective than attempting intubation especially because positive pressure ventilation itself has detrimental effects .

What are the types of airway ?

Changing from bag mask ventilation to intubation is done quickly , hampering CPR as little as possible .

Two types of route may be used :

- Nasopharyngeal Airway (NPA) – by nose
- Oropharyngeal Airway(OPA) - by mouth

If gag or cough reflex is present use NPA .

The main advantage of a NPA over an OPA is that it can be used in either conscious or unconscious individuals because the device does not stimulate the gag reflex.

Attempts at suctioning should not exceed 10 seconds. To avoid hypoxemia, follow suctioning attempts with a short period of 100% oxygen administration.

Assessment of neurologic outcomes

First step is proper assessment of neurologic status (table 1) . A patient with loss of pain stimuli and brainstem reflexes even after 6 hours after resuscitation has grim prognosis . An absent pupillary and corneal reflex 72 hours after arrest implies worst prognosis . A CT scan of brain suggesting grey to white matter ratio (GWR) <1.2 is a marker of adverse neurologic prognosis . CT Brain may show intracranial haemorrhage unexpectedly and appropriate corrective action is to be taken . EEG is important to rule out continuing status epilepticus or undiagnosed myoclonus . Burst –supression on EEG is poor prognostic marker in CA .

Table 1 . Rapid Neurologic Exam

Parameter	Normal Response	Significance
Spontaneous breathing present when in ventilation	Total Breathing rate more than set ventilation rate	Intact respiratory centre function
Pupillary reaction to light	Constriction	Intact brainstem
Corneal Reflex	Blinking in response to brushing	Intact Brainstem
Dolls Eye	Eyes move in opposite direction to the turning of head	Intact Brainstem
Gag reflex	Gag on suction of posterior pharynx	Intact Brainstem
Cough reflex	Cough on suction	Intact Brainstem
Motor response	Withdrawal of upper and lower limb on pain	Intact Pyramidal tract

Targeted temperature management (TTM)

After cardiac arrest three waves of neuronal injury occurs in brain : a) initial hypotension - hypoxia – low and slow flow , b) second wave of ischemia – reperfusion – injury , c) third wave of injury due to temperature dysregulation and the accompanying cytokine induced pyrexia . Severe neuronal injury induces death in 24- 48 hours after ROSC .

TTM or controlled hypothermia by maintaining a temperature of 32- 36 degrees has been shown in some studies to improve neurologic outcomes if started within 6 hours of ROSC .

The contraindications of TTM include refractory shock and bleeding disorders. The relative benefit of 33 vs 36 degree vs normothermia is yet to be determined with certainty . However for those with unstable hemodynamics near normothermia is better while those judged with severe neuronal injury , 33 degrees may be better .

Infusing saline from refrigerator via a central line or using ice packs or cooling blankets are practical but crude ways of using this technique in situations like ER where cooling devices with feedback loop are not available .

Controlling shivering with dexmedetomidine (side effect bradycardia) or propofol (side effect : hypotension) may improve pyrexia control . Using benzodiazepine may hamper neurological assessment . Neuromuscular blockers have also been used for this purpose .

The cardiovascular implications post cardiac arrest

Post arrest the haemodynamics passes through four phases :

- Initial hypertension and tachycardia phase : Due to vasopressors administered
- Honeymoon Period : phase of relative hemodynamic stability as the effect of vasopressors wane .
- Post Arrest Myocardial Dysfunction (PAMD) Phase : PAMD leads to cardiogenic shock at around 6-8 hours after ROSC .
- Phase of Vasoplegia : Between 24 – 48 hours , profound hypotension and shock may may be seen – this is phase has similarities to septic shock . Vasopressor requirement peak during this phase .

Shock , persistant acidosis and hypotension (MAP < 70 mmHg) are poor prognostic markers after ROSC . Reccurent cardiac arrest occurs in 10% . Post ROSC there is a capillary leak syndrome which should be treated with i.v fluids . If hypotension does not improve , noradrenaline is the vasopressor of choice . Aggressive fluid resuscitation may lead to pulmonary edema especially if there is LV dysfunction . In case of PAMD , dobutamine may be helpful in some cases . An ScvO2 <70% indicates requirements of inotrope .

If arrest is due to a treatable cause like STEMI/ACS or pulmonary embolism specific treatment is instituted . STEMI / ACS must be subjected to CAG + Revascularization after quick stabilization even if patient is in coma .

When used in young patient with a reversible cause, emergent venoarterial ECMO may of value in two ways : extracorporeal CPR (ECPR) deployed rapidly during CPR itself and secondly during post ROSC phase . Young patients with reversible cause of cardiac arrest and shockable rhythms when initiated on ECPR within 20 minutes from start of CPR and preferably with etCO2 > 20 mmHg are the optimal candidates for this procedure . However randomized controlled trials and outcome data are lacking .

Post ROSC there is myocardial stunning due to combined effects of oxidative stress from ischemia – reperfusion injury , cytokine storm and deleterious effects of vasopressors , leading to an LVEF of 35-40% . Though it is challenging to differentiate this entity from previously existing structural heart disease , serial echo will show improvement in ventricular function .

The respiratory implications of cardiac arrest

Respiratory arrest is often a part of cardiac arrest . ETCO2 guided ventilation may be useful . A tidal volume of 6-8 ml/kg body weight and PEEP of 5-10 mmHg is beneficial in reducing lung injury (Table 2) . Post ROSC hypoxemia (PaO2 < 60 mmHg) or hyperoxemia (PaO2 > 300 mmHg) both are detrimental and hence should be avoided . PaO2 80-150 mmHg (SpO2 94-99%) is acceptable . The injured brain is still responsive to CO2 . PCO2 <35 mmHg can trigger cerebral vasoconstriction and hence precipitate injury . Permissive hypercapnia with PCO2 of 45-50 mmHg is a useful goal in post cardiac arrest situation .

The neurologic complications of cardiac arrest

Two major neurologic complications are : cerebral edema and seizures .

Cerebral edema is common after hypoxic brain injury . A CT scan showing grey to white matter ratio < 1.2 is suggestive . Cerebral edema leading to elevation of intra cranial pressure (ICP) is a poor prognostic marker Elevation of head end of bed , hyperventilation , osmotherapy with mannitol are unproven therapies in cardiac arrest patients with this condition . However maintaining mean arterial pressure (MAP) with vasopressors is of great importance to improve brain tissue oxygen levels .

Seizures occur in 25% patients post arrest and herald a poorer prognosis . Status epilepticus has a particularly bad prognosis . Similarly poor prognosis has been described for status myoclonus especially if it lasts for > 30 minutes . EEG monitoring and brain imaging are necessary . Levarecitam , valproic acid , phenytoin , phosphenytoin and lacosamide have all been used , with no agent being proved better than the rest .

Further Reading

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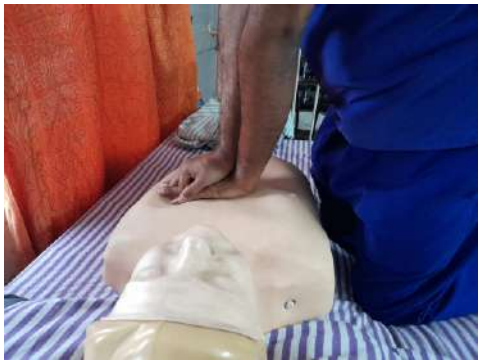

Fig 1 . Technique of chest compression . Note the position of hand .

Fig 2 . Defibrillation. Note the position of the paddles .

Medicolegal Aspect in Precious Pregnancy

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INTRODUCTION :

Medical profession is the most noble but dealing with the most complicated science of human life. It is unpredictable and uncertain, varies from person to person and in the same person from time to time.

It is also varies from drugs to drugs, from dose to dose, from investigations to investigations, from procedure to procedure.

Medical science is not exact science. Due to continuous research experiments newer and newer modalities of management of the Patients coming up. A drug or a procedure well accepted today can be outdated tomorrow. A sincere doctor will try to be updated with newer drugs, technique and procedure but at the same time it is not possible to be aware of each and every research and newer modalities of treatment. Doctor is also under obligation to manage the patient with due reasonable care and skill. In spite of all these limitations, doctor works in emergency twenty four hours a day and seven days a week. But expectations of patients and relatives are so high that, in spite of all effort on part of a doctor, if anything goes wrong or expected result is not achieved, all likely that doctor may have to face litigations.

Ignorance of Law is No Excuse

As soon as any Act, any law, any ordinance passes in Government Gazette, it is presumed by law that each citizen of India knows law and that is why ignorance of law is never an excuse. Doctor may have to face litigations either under CPA 1986 (Amendment 2003 or under CIVIL suit or Criminal case and sometimes simultaneously under both CPA and Criminal or Civil and Criminal. One may has to face litigation before medical council, Human Rights Commission, Government and departmental enquiry in case of Government doctors.

Reason for Litigation

- Dissatisfaction on part of patient and relatives
- Forgetting to screen for High Risk Factor
- Neglecting a symptom/positive report of screening.

Conclusion

Here are some tips to follow to avoid litigation:

- Meticulous record/proper documentation
- Expert opinion whenever needed
- Take medicolegal advice from point one.
- Emergency box should be available round the clock, Please check expiry date and update it accordingly.
- Proper history taking and examination of patient
- Proper counseling
- proper in time investigations
- Take valid consent
- Please do not issue any certificate in absence of patient or in back date
- If you are not sure of cause of death, please do not issue death certificate. Advise postmortem examination
- Take tender care with compassion of Your Patient during treatment/surgery
- Please follow standard protocols in treatment
- Cmply with the provisions of law

- Formation of local level rush team/medicolegal cell to have surgical assistance in emergency and assistance in event of sudden death on table, mob violence and other odd situations and medicolegal consequences.
- Proper communication with relative about mishap (special attention).
- Inform Police, if required
- If you are providing MTP and ultrasound services, please get your center registered under MTP and PCPNDT Act and strict compliance with the provisions of the Act.
- Security Alert every time
- CCTV coverage at every strategic point
- Avoid prescription to relatives of patient of emergency drugs at the time of emergency. You manage it by your own staff.
- Collective responsibility in unusual circumstances when treating persons are more than one. Do not blame each other.
- Adequate indemnity cover
- Identify yourself well in the court.

Have a litigation free practice

Discogenic Pain

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Introduction:

Persistent lower back pain represents a significant and far-reaching medical and societal concern, often a leading cause of disability. Statistics indicate that approximately 80% of individuals across all populations are likely to experience lower back pain at some point in their lives. Notably, research by DePalma et al[1] identified that the prevalence rates for zygapophyseal joints, sacroiliac joints, and lumbar discs were 31%, 18%, and 42%, respectively. Their findings confirmed that lumbar discs are the most frequent cause of chronic lower back pain in adults. The concept of internal disc disruption (IDD), introduced by Crock[2], refers to a discogenic pain syndrome resulting from disc degeneration and non-nerve root referred pain. IDD, responsible for discogenic lower back pain, accounts for a significant portion, ranging from 26% to 42%, of patients with chronic lower back pain[1,3,4]. It is recognized as a distinct clinical entity, distinguished from other forms of disc-related low back pain, such as lumbar disc herniation, degenerative disc disease (DDD), and lumbar segment instability[8]. Radiographic assessments of IDD patients' lumbar X-rays typically do not reveal characteristic changes seen in degenerative disc diseases, such as intervertebral space narrowing, osteophyte formation, endplate sclerosis, or gas formation within the disc space[5].

Pathophysiology:

The intervertebral disc serves as the primary joint connecting two adjacent vertebrae within the spinal column. Each disc comprises three distinct components: an inner gel-like nucleus pulposus, an outer annulus fibrosus enveloping the nucleus pulposus, and two cartilage endplates covering the upper and lower surfaces of the vertebral bodies.(**(Fig 1).**

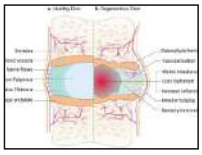


Figure 1.

In a youthful, healthy disc, it functions akin to a water bed due to the high water content within the nucleus and inner annulus, allowing the tissue to behave like a fluid. The outermost annulus functions as a resilient "skin" that contains the nucleus.

When disc degeneration occurs, it typically results from abnormalities in the matrix. At the molecular level, degeneration is marked by the production of atypical matrix components, an increase in mediators like IL-1 and TNF-α responsible for matrix degradation, heightened levels of matrix metalloproteinases (MMPs), and a decrease in tissue inhibitors of metalloproteinases (TIMPs). Numerous factors contribute to disc degeneration, including genetic predisposition, mechanical loading, and nutritional factors, all recognized as significant contributors to this degenerative process[6].

As disc degeneration advances, there is a loss of proteoglycans and water from the nucleus, leading to inadequate hydrodynamic distribution of axial stresses to the outer annulus fibrosus. This degeneration may result from an imbalance between the processes responsible for building and breaking down tissue or the loss of the stable metabolic statetypically maintained in a healthy disc. Changes in both anabolic (building) and catabolic (breaking down) processes are believed to play crucial roles in the initiation and progression of disc degeneration.

Clinical Presentation :

The clinical diagnosis of lumbar discogenic disease is characterized by specific features, including axial midline low back pain(Fig 3) , discomfort when sitting, pain experienced during flexion, positive reactions to sustained hip flexion, the absence of motor, sensory, or reflex changes, and a positive response to discography (Fig 6,7) [7,8,9,10,11,12,13,14].

Discogenic pain is often described as a dull, aching, and gnawing sensation [9]. Although it primarily occurs in the lower back, it can also be accompanied by referred pain to the lower extremities, typically above the knees and without following a dermatomal pattern. In cases of referred leg pain, patients report a deep tissue discomfort, often described as a sensation of expanding pressure [9]. This type of discogenic pain involving the legs is always somatic in nature and has a sclerotomal distribution, expanding into broader areas that can be challenging to pinpoint (as illustrated in Figure 2). Although the precise boundaries of the pain can be elusive, patients can usually identify its central or core location. Since somatic referred pain doesn't result from nerve root compression, it doesn't exhibit neurological radicular signs.

Diagnosis :

Disc degeneration often becomes apparent in magnetic resonance imaging (MRI)

T2-weighted (Fig 4) images as a reduction in signal intensity, often referred to as a "black" disc. While MRI can reveal the presence of a degenerated disc and an annular tear, it may not provide a clear distinction between a disc that is pathologically causing pain and one that is simply undergoing normal age-related changes[15].

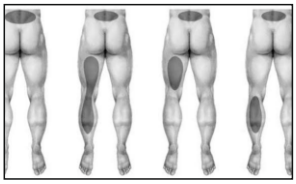


Figure 2.

Figure 3.

The diagnostic criteria for Internal Disc Disruption (IDD) established by the International Association for the Study of Pain (IASP) include the following criteria: the emergence of a concordant pain response during discography (Fig 6,7), the presence of internal annular disruption as revealed by CT after discography (CTD)[Fig8], and the involvement of at least one adjacent disc without a concordant pain response[15].

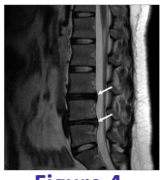


Figure 4.

Furthermore, the "Modified Dallas Discogram Description" method[16,17] (Fig 5)classifies the degrees of annular disruption into four grades as follows:

- Grade 0: Contrast medium remains within the normal nucleus pulposus.
- Grade 1: Contrast medium flows into the inner third of the annulus through an annular fissure.
- Grade 2: Contrast medium flows into the middle third of the annulus.
- Grade 3: Contrast medium flows into the outer third of the annulus, extending circumferentially less than a 30° arc at the disk center.
- Grade 4: Contrast medium flows into the outer third of the annulus, extending circumferentially more than a 30° arc at the disk center.
- Grade 5: Contrast medium leakage into the outer space.

Grades 0, 1, and 2 are considered normal findings, while Grades 3 and above indicate annular disruption.The history of intradiscal procedures traces back to the aftermath of World War II. In 1941, Jansen and Balls derived the chymopapain enzyme from papaya fruit. In 1956, Thomas elucidated the chemical structure of this material. The first injection of chymopapain inside the disc occurred in 1964 by Smith. In 1994, Menno Sluijter described intradiscal RF thermocoagulation, a significant milestone. Ozone chemonucleolysis was introduced by Verga in 1983.

The Saal brothers played a pivotal role in popularising intradiscal electrothermal therapy in 1998. In 2004, Finch introduced the discTRODE procedure, and more recently, biacuplasty has gained recognition as a treatment method for disc-related issues. This historical timeline underscores the ongoing evolution and development of intradiscal procedures for addressing various spinal conditions.

Treatment :

Indeed, the management of chronic low back pain offers a variety of treatment options, often with differing opinions among clinicians regarding the most effective approach.

Pharmacological treatments typically encompass the

use of analgesics, nonsteroidal anti-inflammatory drugs, muscle relaxants, anticonvulsants, and tricyclic antidepressants, often complemented by physical therapy.

In addition to these conventional treatments, there are several alternative options for managing discogenic pain, including:

1. Intradiscal Injections:
- Chemonucleolysis.
- Ozone injection.

2. Annuloplasty:
- Intradiscal electrothermal therapy (IDET).
- Radiofrequency (RF) posterior annuloplasty (RFA).
- Biacuplasty.

3. Percutaneous Disc Decompression:
- Laser discectomy.
- RF coblation (plasma discectomy).
- Mechanical disc decompression .
- Manual percutaneous lumbar discectomy (PLD).

4. Endoscopic Percutaneous Discectomy.

These alternative treatments provide a range of options for patients and their healthcare providers to consider when addressing chronic low back pain, particularly when discogenic pain is a concern. The choice of treatment often depends on the individual patient's condition and the recommendations of their healthcare team.

Ozone chemonucleolysis is a medical procedure that entails the introduction of ozone gas (a mixture of oxygen and ozone, O2-O3) at a concentration of 20/30 µg/ml in volumes ranging from 40 to 60 ml. This process is typically repeated 8 to 14 times. The injection is typically administered into the paravertebral musculature and within the area affected by the hernia. In most cases, the injection procedure is relatively painless and well-tolerated by patients, with only a brief, localised sensation of pain.

Biacuplasty is a novel annuloplasty procedure that involves the use of a bipolar system. This technique is performed using two cooled radiofrequency (RF) electrodes, which are strategically placed on the posterolateral sides of the intervertebral annulus fibrosus. The placement of these electrodes is guided by fluoroscopy. Initially, two 17-gauge transdiscal introducers are positioned within the posterior annulus, employing a posterolateral oblique approach. Subsequently, RF probes are introduced through each of these introducers bilaterally, creating a bipolar configuration. The temperature of the electrodes gradually increases to 55°C over a span of 11 minutes during the procedure. Biacuplasty is designed to address specific issues related to the intervertebral discs in cases of chronic low back pain. (Fig 9,10,11).

Nucleoplasty (Fig 12,13) is a medical procedure that was first performed in 2000. It involves the use of radiofrequency (RF) coblation to achieve decompression of a contained herniated disc through a combination of disc removal and thermal coagulation. The indications for this procedure include patients experiencing low back pain, with or without radiculopathy, and those with confirmed herniated discs via MRI, who have not responded to conservative therapy. However, patients with spinal stenosis, a significant loss of disc height (50% or more), severe disc degeneration, spinal fractures, or tumors should be excluded from this procedure.

Here's an overview of the Nucleoplasty procedure:

1. A 17-gauge obturator stylet is inserted into the disc. Sometimes, a discogram is performed at this stage to confirm the location and provoke a positive response.

2. With caution to avoid the anterior annulus, RF waves are used to ablate the nucleus pulposus as the wand is advanced. This process converts tissue into gas, which is subsequently removed through the needle.

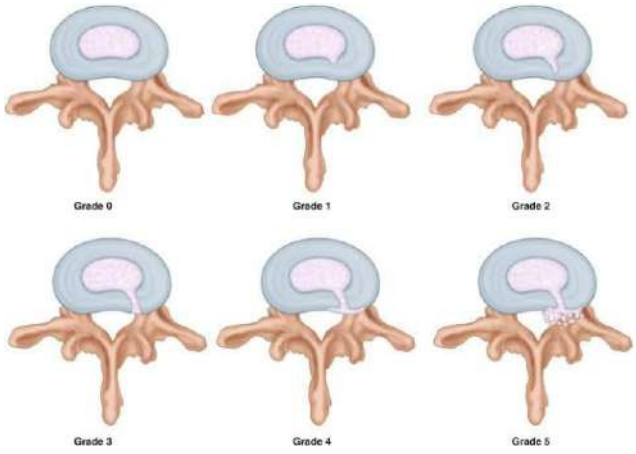
3. As the wand is withdrawn, thermal coagulation occurs, treating the channel and denaturing nerve fibers adjacent to the channel within the nucleus pulposus.

4. This process can be repeated up to six times within a single disc, with the tip of the catheter positioned at different clock positions (2, 4, 6, 8, 10, and 12 o'clock) during each cycle.

In recent times, there has been a growing interest in

developing strategies to biologically repair or regenerate degenerated discs. These approaches primarily aim at achieving two main objectives: restoring the structure of the disc and alleviating pain[19]. While biologically based treatments have shown promise in restoring disc structure, their ability to provide pain relief is still uncertain. However, recent findings from animal studies have indicated changes in cytokine expression after the injection of growth factors, suggesting a potential mechanism for pain relief. Moreover, the first human clinical trial for growth factor injection therapy is currently underway, offering hope for insights into clinical outcomes.

Mesenchymal stem cells (MSCs) also show promise in reducing pain through their ability to decrease inflammation. A recent study suggests that MSCs can stimulate the production of anti-inflammatory cytokines[20]. Nonetheless, further research is necessary to fully understand the mechanisms underlying pain relief in these biological treatments. These developments represent exciting avenues in the quest to address both the structural and pain-related aspects of degenerated discs.



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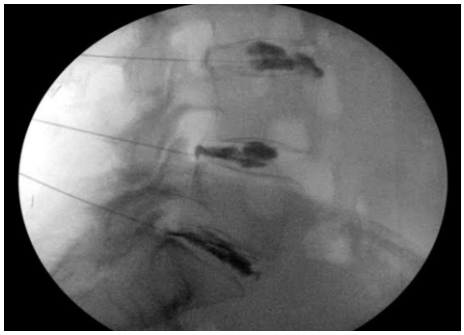

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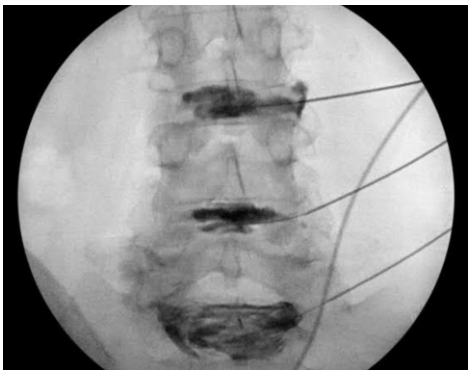

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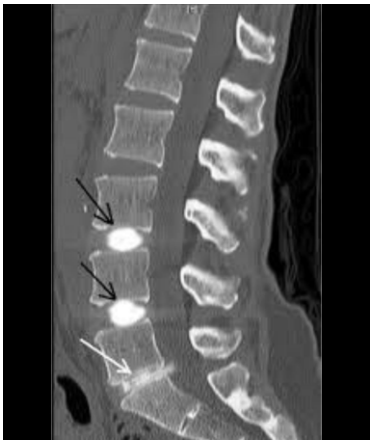

Figure 8.

Figure 9.

From Fragile to Resilient: Confronting Osteoporosis Head-On

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Introduction

The term ''osteoporosis'' is derived from two Greek words : ''Osteo'', which mkeans ''bone'' and ''porosis'' , which means ''porous'' or ''spongy''. Therefore , the word ''osteoporosis'' essentially means ''porous bone''. This name accurately reflects the characteristic feature of the condition,where the bones become less dense and more fragile, resulting in an increased risk of fractures.

Osteoporosis is a silent and often underestimated disease, poses a significant public health challenge worldwide. It is a systemic skeletal disorder characterized by low bone mass, microarchitectural deterioration of bone tissue, and an increased susceptibility to fractures. This article aims to provide a comprehensive overview of osteoporosis, including its pathophysiology, risk factors, diagnosis, management, and prevention strategies.

Definition

The World Health Organization has defined osteoporosis as bone mineral density (BMD) of 2.5 standard deviations below the peak mean bone mass of young , healthy adults.

Epidemiology

In India, about 20% of women and 10-15% of men could be osteoporotic. By absolute numbers, about 26 million in India suffer from osteoporosis.

Pathophysiology

Bone is a dynamic tissue that undergoes a continuous process of remodeling, involving bone resorption by osteoclasts and bone formation by osteoblasts. Osteoporosis occurs whenthis balance is disrupted, leading to increased bone resorption and decreased bone formation. The result is weakened bone with reduced density and altered architecture.

Risk Factors

Several factors contribute to the development of osteoporosis, including:

1. Age: The risk of osteoporosis increases with age, especially in postmenopausal women due to

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hormonal changes.

2. Gender: Women are at a higher risk than men because of lower peak bone mass and hormonal changes.

3. Family history: A family history of fractures or osteoporosis can increase an individual's risk.

4. Hormonal changes: Reduced estrogen levels in menopause and low testosterone levels in men can accelerate bone loss.

5. Lifestyle factors: Smoking, excessive alcohol consumption, a sedentary lifestyle, and poor nutrition can weaken bones.

6. Medications: Certain medications, such as glucocorticoids, anticonvulsants, and some cancer treatments, can contribute to bone loss.

7. Medical conditions: Conditions like rheumatoid arthritis, hyperthyroidism, and celiac disease can affect bone health.

Diagnosis

The diagnosis of osteoporosis typically involves a combination of clinical assessments and specialized tests to evaluate bone density and assess fracture risk. Here are the primary investigations and tests used for diagnosing osteoporosis:

1. Medical History and Physical Examination:

The initial step in the diagnosis of osteoporosis involves a thorough medical history and physical examination. This helps the healthcare provider identify risk factors, assess symptoms, and look for signs of underlying medical conditions that may contribute to bone loss.

2. Fracture Risk Assessment:

Clinicians often use tools like the FRAX® (Fracture Risk Assessment) tool to estimate a patient's risk of experiencing a major osteoporotic fracture (hip, spine, forearm) over the next 10 years. FRAX considers clinical risk factors such as age,sex, family history, prior fractures, smoking status, alcohol consumption, and certain medical conditions.

3. Dual-Energy X-ray Absorptiometry (DXA) Scan:

DXA is the gold standard for measuring bone mineral density (BMD). It is a painless and non-invasive test that involves a low-level X- ray to assess BMD at specific sites, typically the hip and spine. Results are reported as T-scores, which compare an individual's BMD to that of a healthy young adult (peak bone mass) and Z- scores, which compare BMD to age-matched peers. A T-score of -2.5 or lower indicates osteoporosis.

4. Laboratory Tests: Blood tests may be conducted to rule out underlying medical conditions that can affect bone health. These tests may include:

- **Serum Calcium:** To check for abnormal calcium levels.
- **Serum Phosphorus:** To assess phosphorus levels, which are related to bone health.
- **Vitamin D Levels:** To determine if a deficiency is present, as vitamin D is essential for calcium absorption.
- **Thyroid Function Tests:** To assess thyroid hormones, as thyroid disorders can impact bone health.
- **Role of X-ray:** X-rays can reveal the presence of fractures, including vertebral (spinal) fractures that are common in osteoporosis. These fractures may occur with minimal trauma and can go unnoticed by the patient. By identifying vertebral fractures, healthcare providers can diagnose osteoporosis and determine the extent of bone fragility.

Screening

Osteoporosis is a silent disease until fracture happens. Therefore , screening becomes so much important to prevent low-trauma fractures .

Screening guidelines differ in different regions of the world where different cost– benefit models are employed. In the United States and Canada, routine BMD screening with DXA is recommended in all women aged 65 years and older. This recommendation is endorsed by the US Preventative Services Task Force (USPSTF, grade B recommendation). There is less consensus about the utility of routine osteoporosis screening in older men. The USPSTF did not find sufficient evidence to recommend routine screening

for men.

The optimal BMD screening interval remains unclear for individuals who do not meet initial intervention thresholds. Several screening guidelines suggest a minimum of 2 years between repeated BMD tests based on limitations in the precision of DXA testing.

Treatment

The treatment of osteoporosis aims to reduce the risk of fractures, improve bone density, and manage any underlying causes or contributing factors. Treatment plans are tailored to each individual based on their specific needs, risk factors, and the severity of the condition. Here are the primary components of osteoporosis treatment:

NOF Guidelines for treatment initiation in post-menopausal women

1. Previous vertebral hip fracture
2. T-score below -2 by hip DXA
3. T-score below -1.5 by hip DXA and 1 or more of the risk factors
4. Personal history of fracture as an adult
5. History of fragility fracture in first degree relative
6. Low body weight (less than 127 lb)
7. Current smoking
8. Oral corticosteroid (more than three months)

A) Pharmacological Management:

A number of agents exist to treat osteoporosis but due to possible side effects, they should be carefully considered depending on the clinical comorbidities of each patient. To assist the clinician with initiating osteoporosis medications based on risk and benefits to an individual

patient, the National Osteoporosis Foundation (NOF) has created guidelines for initiating pharmaceutical agents in postmenopausal women. The qualifying group should have one of the criteria listed below:

Several classes of medications can be prescribed to manage osteoporosis,

including:

1. Bisphosphonate: These drugs inhibit bone resorption and include medications like alendronate, risedronate, ibandronate, and zoledronic acid.

Three bisphosphonates—alendronate (Fosamax), risedronate (Actonel), and zoledronic acid (Reclast)—have been found to improve bone mineral density (BMD), reduce the risk of hip and other nonvertebral fractures, and prevent vertebral fractures. Both alendronate and risedronate are recommended if osteoporosis is caused by overuse of steroid medications, but risedronate also prevents steroid-induced osteoporosis. Because both medications reduce the occurrence of vertebral and nonvertebral fractures by about 50

%, they are currently termed “agents of choice.” Another bisphosphonate, ibandronate, reduces the incidence of vertebral fractures by approximately 50 % over three years. Whereas these drugs can be taken orally, zoledronic acid (ZA) is administered intravenously which may help to increase adherence to therapy. Zoledronic acid is the most potent of the bisphosphonates and has demonstrated significantly better reduction in bone turnover markers relative to alendronate

2. Teriparatide and Abaloparatide: These medications, approved by FDA are synthetic forms of parathyroid hormone (PTH) and has been demonstrated to increase BMD as well

as reduce the likelihood of vertebral and nonvertebral fractures in women. Unlike other treatments, it is an anabolic agent that stimulates bone formation. Reported side effects include hypercalciuria, causing acute gout, leg cramps, or dizziness with orthostatic hypotension

Head-to-head trials of bisphosphonates have produced insufficient evidence to prove or disprove any single agent's superiority in preventing fractures; similarly head-to-head trials of bisphosphonates compared to teriparatide or raloxifene have produced insufficient evidence to prove or disprove relative superiority

3. Monoclonal Antibodies: Denosumab is a medication that blocks the action of osteoclasts, thereby reducing bone resorption. Compliance is also favourable with this agent, given its twice annual administration in a doctor's office. It is approved by FDA for use in the treatment of fracture and to increase bone mass in men with osteoporosis at high risk of fracture. Denosumab is administered as a single subcutaneous injection of 60 mg every 6 months. Unlike zoledronic acid, denosumab is not cleared

renally and therefore can be safely administered to those with renal insufficiency

4. Calcitonin: It is secreted by thyroid parafollicular cells, acts to suppress osteoclastic activity that leads to small increases in bone mass and reduction in vertebral, but not hip or distal extremity,
fracture risk. Approved for women who are at least five years postmenopausal, it is administered intranasally, with potential adverse effects of congestion or epistaxis. Given its limited effect, calcitonin is not considered a first-line treatment.

5. Selective Estrogen Receptor Modulators (SERMs): Medications like raloxifene can help maintain bone density and reduce fracture risk in postmenopausal women.

6. Treatment of osteoporosis in men includes the usual supplementation with calcium (1200-1500 mg/day) and vitamin D (800-1000 IU/day).

The choice of medication depends on factors like the patient's risk profile, preference, and potential side effects. A healthcare provider will determine the most appropriate treatment.

B) NON-PHARMACOLOGICAL MANAGEMENT:

1. EXERCISE

- With a known history of osteoporosis, exercises making use of either passive or active spine flexion are to be avoided
- Abdominal and back musculature should be strengthened in neutral spinal positioning, with progression toward spine extension as tolerated.
- Weight-bearing exercise is paramount because it helps to stimulate osteoblasts to form bone. Selecting the proper physical exercise can increase muscle strength and

BMD thereby decreasing the risk of appendicular fractures and related mortalities in the elderly

2. Fall Prevention

Reducing the risk of falls is crucial, as falls can lead to fractures in individuals with osteoporosis. This involves:

- Home modifications to remove tripping hazards and improve safety.
- Use hand rails as you go up and down steps and on escalators.
- Keep floors free of clutter. Be sure all carpets and area rugs have skid-proof backing or are tacked to the floor.
- Install grab bars on the bathroom walls beside the tub, shower and toilet. Use a non- skid rubber mat in the shower or tub.
- Vision correction, if needed.
- Balance and strength training exercises.
- Assistive devices like canes or walkers for those at higher risk of falls.

3. Management of Underlying Conditions: If osteoporosis is secondary to an underlying medical condition or medication, managing that condition or adjusting medications may be necessary.

4. Nutritional Supplements: In addition to dietary changes, calcium and vitamin D supplements may be prescribed if dietary intake is insufficient.

5. Smoking Cessation and Alcohol Moderation: Encourage patients to quit smoking, as smoking can weaken bones. Limiting alcohol consumption is also advisable, as excessive alcohol intake can negatively impact bone health.

Conclusion

Osteoporosis is a common and potentially debilitating condition that affects millions of individuals worldwide. Early diagnosis, risk factor assessment, and appropriate management are essential to mitigate the impact of this disease. By emphasizing prevention and adopting a comprehensive approach to bone health, healthcare providers can contribute significantly to reducing the burden of osteoporosis and improving the quality of life for affected individuals.

Outline of Food Care & Screening and Management of Neuropathy in Diabetes

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Food Care

Diabetic foot ulcers are the leading cause of nontraumatic lower limb limb amputations. A complex background of pathophysiologic events has been identified with advances in molecular biology such that newer interventions are possible. Bioengineered skin substitutes currently play an important role in the diabetic fool ulcer, particularly in the "stalled" wounds that fail to show with more focused targeting of these pathophysiologic events. The major background impairment is that of vasculogenesis brought about as a direct result of hyperglycemia, advanced glycation end product/methylglyoxal generation, and it's direct effect on endothelial progenitor cell production and attraction to the wound site. At the same time , glycation end products affect immunity and inflammation, increasing the susceptibility to infection. Thus, reactive oxygen species scavengers, methylglyoxal scavengers, vascular endothelial growth factor stimulators as well as various lipids and neuropeptides are all potential agents that could be incorporated into such templates. The logical sequence of events and possible interventions is detailed. In this manner, intrinsic healing is encouraged in preference to our current attempts at adding unknown quantities of specialized cellular and growth factor components which seem to provide very temporary advantages.

Food care recommendations

- For all patients with diabetes, perform an annual comprehensive foot examination to identify risk factors predictive of ulcers and amputations. The foot examination should include inspection, assessment of foot pulses, and testing for loss of protective sensation (LOPS) (10- g monofilament plus testing any one of the following : vibration using 128 – Hz tuning fork, pinprick sensation, ankle reflexes, or vibration perception threshold).

- Provide general foot self – care education to all patients with diabetes.
- A multidisciplinary approach is recommended for individuals with foot ulcers and high – risk feet, especially those with a history of prior ulcer or amputation.
- Refer patients who smoke , have Lops and structural abnormalities, or have history of prior lower – extremity complications to foot care specialists for ongoing preventive care and lifelong surveillance.
- Initial screening for peripheral arterial disease (PAD) should include a history for claudication and assessment of the pedal pulses. Consider obtaining an ankle – brachial index (ABI), as many patients with PAD are asymptomatic.
- Refer patients with significant claudication or a positive ABI for further vascular assessment and consider exercise, medications, and surgical options.

SCREENING & MANAGEMENT OF NEUROPATHY

ADA guidelines

- All patients should be screened for distal symmetric polyneuropathy (DPN), starting at diagnosis of T2DM and 5years after the diagnosis of T1DM and at least annually thereafter, using simple clinical

tests.

- Electrophysiological testing or referral to a neurologist is needed, except in situations where the clinical features are atypical.
- Screening for signs and symptoms of CAN should be instituted at diagnosis of T2DM and 5 years after the diagnosis of T1DM. Special testing is rarely needed and may not affect management or outcomes.
- Medications for the relief of symptoms related to painful DPN and autonomic are recommended because they may reduce pain and improve quality of life.

Management of diabetic neuropathy

- The primary care physician needs to be alert for the development of neuropathy – or even it's presence at the time of initial diabetes diagnosis – because failure to diagnose diabetic polyneuropathy can led to serious consequences, including disability and amputation. **Management of diabetic neuropathy should begin at the initial diagnosis of diabetes.**
- Consider any patient with clinical evidence of diabetic peripheral neuropathy to be at risk for foot ulceration, and provide education on foot care. If necessary, refer the patient to a podiatrist. Admit patients for an infected diabetic foot ulcer or gangrene.
- Patients with diabetic peripheral neuropathy require more frequent follow –up, with particular attention to foot inspection to reinforce the need for regular self – care. The provision of **regular food examinations and** reinforcement of the educational message on foot care have been shown in several studies to significations reduce of ulceration and even amputation.
- The primary care physician is responsible for educating patients about the acute and chronic complications of diabetes, including the psychological impact of sexual dysfunction in both men and women. The importance of involving a neurologist (preferably with expertise in peripheral neuropathy) in the treatment of patients with diabetic neuropathy cannot be overemphasized.

Tight Glycemic control

- Of all treatments, is probably the most important for slowing the progression of neuropathy. Because rapid swing from hypoglycemia to hyperglycemia have been suggested to induce and aggravate neuropathic pain, the stability of glycemic control may be as important as the actual level of control in relieving neuropathic pain. **Tight and stable glycemic control**
- **The Diabetes Control and complications Trial(DCCT)** demonstrated that tight **blood sugar control** in patients with type 1 diabetes **decreased the risk of neuropathy by 60% in 5 years.** The effect of tight glycemic control on polyneuropathy in patients with type 2 diabetes or those with impaired glucose tolerance/impaired fasting glucose is not as clear and requires further prospective study.
- **A 2012 Cochrane review indicates that tight glycemic control prevent** the development of clinical neuropathy and reduces nerve conduction and vibration threshold abnormalities in patients with either type 1 or type 2 diabetes .
- **Diabetic Neuropathic pain Management**
- Many medications are available for the treatment of diabetic neuropathic pain. Oral agents include antidepressants and anticonvulsant drugs. According to the 2011 guideline issued by the American Academy of Neurology (AAN), American Academy of physical Medicine and Rehabilitation (AANEM) and the American Academy of physical Medicine and Rehabilitation (AAPMR) guideline for the treatment of painful diabetic neuropathy(PDN)
- **Pregabalin** is recommended for treatment of diabetic neuropathic pain. The drug has been proven effective and can improve quality of life.
- **Gabapentin and sodium valproate** should also be considered for diabetic neuropathy pain management.
- According to a cochrane review evaluating gabapentin for chronic neuropathic pain and fibromyalgia, **gabapentin leads to significant pain relief in patients with chronic neuropathic pain when compared with a placebo.**
- According to the 2011 AAN/AANEM/AAPMR **guideline, dextromethorphan, morphine sulfate, tramadol and oxycodone** should be considered for PDN treatment.
- Topical therapy with capsaicin or transdermal lidocaine may be useful in some patients, especially those with more localized pain or those in whom interactions with existing oral medications is a

concern.

- The 2011 AAN/AANEM/AAPMR guideline recommends that both of these agents be consider in for treatment of PDN. In clinical trials, **Capsaicin has been effective in reducing pain in PDN,** but many patient cannot tolerate the side effects, such as burning pain on contact with warm/ hot water or in hot weather.
- Patients should be assessed every 6 weeks so that adverse effects can be monitored if possible. Decrease or increase drug dose if indicated.
- In a review of 6 trials (2220 patients) on
- Duloxetine's effects on painful diabetic peripheral neuropathy (3 trials), Lunn et al concluded that 60mg of duloxetine daily can relive the pain of peripheral neuropathy in the short-
- The 2001 AAN/AANEM/AAPMR guideline recommends considering the antidepressants amitriptyline, venlafaxine and **duloxetine for the treatment of PDN,** although data are insufficient to recommend one of these agents over the others.
- There was no difference identified between gabapentin and tricyclic antidepressants in the achievement of pain relief in diabetic neuropathy.
- During pregnancy, prescribing medicine for pain control is difficult. The best hope for pain control in rare cases of young women with severe neuropathy is to control their blood glucose diligently and try to control pain with acetaminophen. At the end of the third trimester, the physician can prescribe amitriptyline, gabapentin and other medications as indicated if the benefit clearly outweighs the risk to the fetus.

Experimental Therapies

Aldose reductase inhibitors

Aldose reductase inhibitors block the rate – limiting enzyme in the polyol pathway that is active in hyperglycemic states. Numerous studies of aldose reductase inhibitors (eg , alrestatin, sorbinil, tolrestat, epralrestat) have been published in the past 30 years, but many of the earlier trials had problems related to poor study design

Epralrestat is currently marketed only in Japan and India. Epalrestat reduces intracellular sorbitol accumulation, which has been implicated in the pathogenesis of late – onset complication of diabetes mellitus.

Epalrestat 150 mg/ day for 12 weeks improved motor and sensory nerve conduction velocity and vibration threshold compared with baseline and placebo with diabetic neuropathy. Subject symptoms, including pain, numbness, hyperesthesia, coldness in the extremities, muscular weakness, dizziness, and orthostatic fainting, were also improved.

Alpha – lipoic acid

In a multicenter placebo – controlled trial of the antioxidant alpha – lipoic acid, Ziegler and colleagues reported short- term symptomatic relief of neuropathy symptoms in patients with type 2 diabetes and symptomatic neuropathy.

Actovegin

A deproteinized derivative of calf blood, actovegin contains inorganic substances (eg, electrolytes, trace elements) and organic components (eg, amino acids, oligopeptides, nucleosides, glycosphingolipids). Actovegin also contains inositol phosphooligosaccharides (IPOS), which are through to elicit central and peripheral insulin effects. Ziegler et al found that **treatment with actovegin improved neuropathic symptoms, vibration perception threshold, sensory function and quality of life** in 567 patients with type 2diabtes mellitus and diabetic polyneuropathy. In this multicenter, randomized, double – blind trial, sequential intravenous(200mg/d) and oral(18000 mg/d) actovegin treatment was given over 160 days.

Spinal cord stimulators and other therapies

Pain medicine specialists have been experimenting with spinal cord stimulator implants in severely painful cases. One such study of 10 patients showed that median background and peak pain scores at the end of the study were, respectively, 77 and 81with the stimulator off and 23 and 20 with the stimulator on. Exercise tolerance significantly improved at 3 months (n = 7, median increase 85%) and at 6 month. Further study is necessary.

Alternative and complementary therapies for pain (eg, acupuncture) are under investigation

Take home Message:

Specific treatment for the underlying nerve damage is currently is not available, other then improved glycemic control, which may modestly slow progressing in T2DM but not reverse neuronal loss.

- DPN symptoms and especially neuropathic pain, can be severe, have sudden onset, and are associated with lower quality of life, limited mobility , depression and social dysfunction.
- There is limited clinical evidence regarding the most effective treatment for individual patient needs given the wide range of available medications.
- Two drug have been approved for relief of DPN pain in the U.S (pregabalin and duloxetine) but neither of these affords complete relief, even when used in combination.
- Venlafaxine, amitriptyline, gabapentin, valproate, opioids (morphine sulfate, tramadol and oxycodone controlled – release) may also be effective and could be considered for treatment of painful DPN.
- Head – to-head treatment comparisons and studies that include quality – of life outcomes are rare, so treatment decisions must often follow a trial – and error approach.
- Given the range of partially effective treatment options, a tailored and step – wise pharmacological strategy with careful attention to relative symptom improvement, medication adherence and medication side effects is recommended to achieve pain reduction and improve quality of life.

Polycystic Ovarian Syndrome

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PCOS or polycystic ovarian syndrome is a well-known and hugely discussed subject today. But what is PCOS? Patients often asks me whether cysts have decreased in number or size after treatment of PCOS. Truly speaking the name polycystic is a misnomer. In a normal female in her reproductive age one follicle ovulates each month. In PCOS that event is deficient and that is the reason that there are many follicles seen in the ovaries. So, there is no question of decrease in number or size of the cysts. The main issue is whether she is ovulating or not.

PCOS presents in different phases of life. We often find obese adolescent girls with excessive hair growth on their face or acne or a hyperpigmented line over their nape of the neck, who are having delayed period. When we ask about the family history of these patients, we very often get a history of diabetes among her family member(s). One point to be noted is when we do ultrasound of such a patient we may or may not get the features of polycystic ovaries. But still, we diagnose her to suffer from polycystic ovarian syndrome. So, ultrasound diagnosis is not essential for PCOS.

Some patients present after their marriage with a history of failure to conceive. So, infertility is also one of the main presenting symptoms in this disease. In fact, it is an alarming issue that 70-80% of infertile women suffer from PCOS!

The question arises why this disease is becoming so common nowadays. First of all, there is a genetic aspect of PCOS, because the disease runs in the family. Now if the girl who is genetically predisposed to PCOS leads an unhealthy life style she is very likely to get the disease. Just tell me, how many of the adolescent girls regularly play outdoor games or how many of them avoid sweets, chocolates and fast food. They are either busy with their studies or mobiles. So, if they are genetically predisposed, they are very likely to develop this disease which happen in most of the cases.

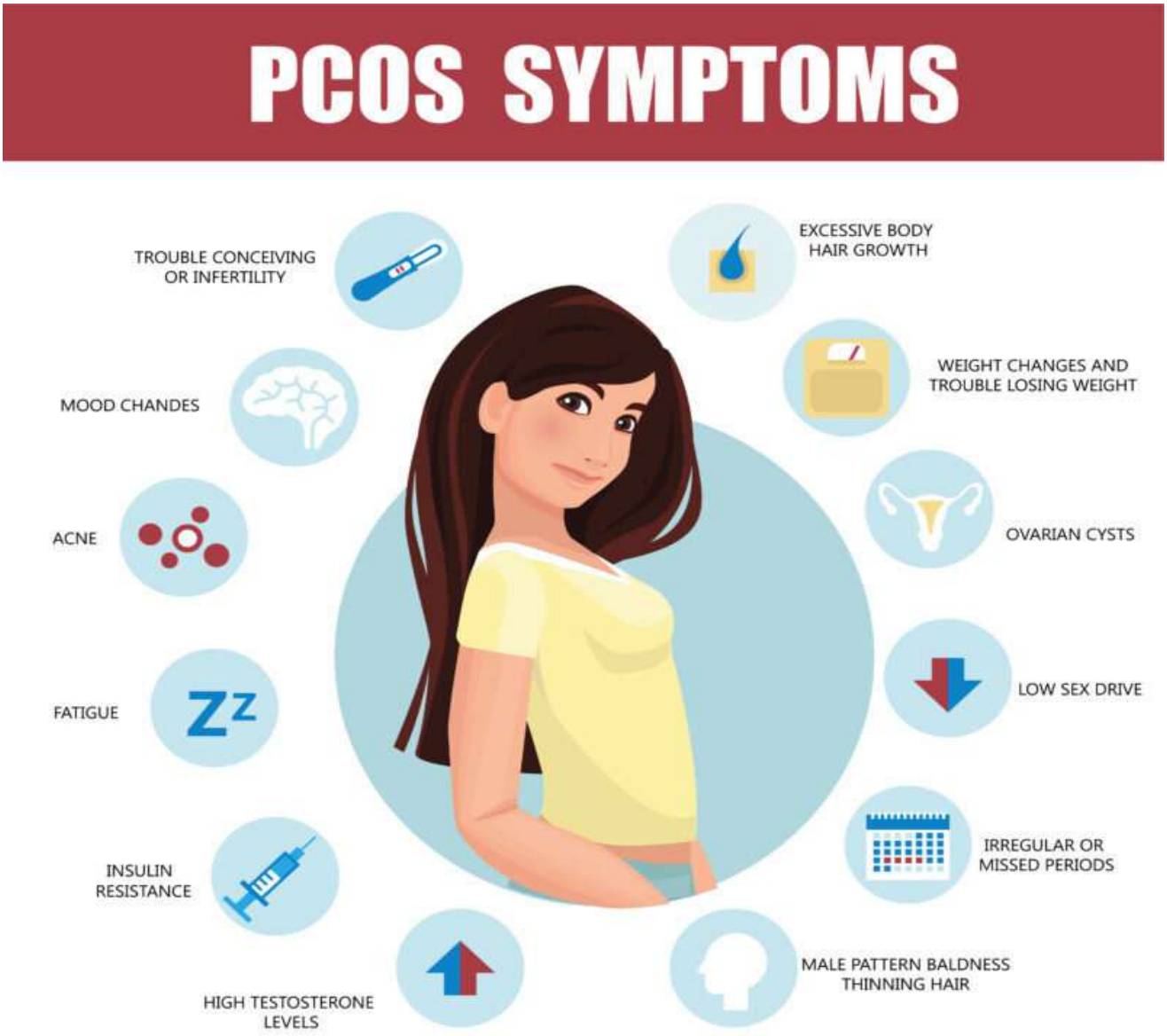
One may ask what is the problem if I have PCOS and can adjust with irregular periods. First of all, infertility problem (which I have already mentioned), obesity, diabetes (because the hormone insulin cannot function properly) and its several consequences, and a syndrome called metabolic syndrome, which encompasses a group of risk factors which make the person susceptible to cardiovascular disease. They will have abnormal lipid profile, hypertension and increased risk of heart attack and stroke! Not only that, they are more prone to develop depression and cancer of uterus too!

Now the main question - can we prevent or cure the disease? Truly speaking, there are three ways for prevention and cure - WEIGHT REDUCTION, WEIGHT REDUCTION and WEIGHT REDUCTION. You maintain an optimum BMI (18.5-24.9 kg/m^2) and have regular periods, then even if you have PCOS in your ultrasound report there is nothing to worry. Studies have shown than 5-10% of weight reduction can restore fertility in

an infertile woman. Of course, you have to consult your gynecologist for the treatment and he or she will prescribe the required medicines, but the main goal will be weight reduction.

Many a times we face a problem - a PCOS patient who bleeds regularly while on medicines again starts to have irregular periods when her treatment is stopped. Most of the time what happens is when they start to bleed regularly, they become reluctant to avoid weight gain. Now come to a practical point - you cannot take medicines throughout your reproductive life, but to avoid having PCOS and its long-term complications, you have to maintain a healthy life style. Only that can eliminate this deadly disease from our society.

One point to consider is that even though most of our PCOS patients are obese, lean PCOS may also occur. They should also do regular exercise and avoid getting obese and of course they have to take medicines from the doctors.



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