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EDITORIAL...

IMA TAMILNADU the most active branch in the country caring for the medical fraternity is now venturing on the Academic side with this journal COMPREHENSIVE MEDICAL JOURNAL OF INDIA (CMJI).

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COMPREHENSIVE MEDICAL JOURNAL OF INDIA (CMJI)
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COMPREHENSIVE MEDICAL
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Approach to "Asymptomatic" Hyponatremia

Dr. Hariharan Sathananthan, M.B.B.S., M.R.C.P
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Hyponatraemia, defined as a serum sodium concentration is lesser than135 mmol/L, is the most common disorder of body fluid and electrolyte balance encountered in clinical practice. Hyponatraemia is present in 15–20 % of emergency admissions to hospital and occurs in up to 20 % of critically ill patients. Symptoms may vary from subtle to severe or even life threatening. Despite this, the management of patients remains problematic.

It has been common practice not to evaluate or treat so called 'asymptomatic hyponatremia.' However it is being increasingly recognized that even mild degrees of hyponatremia are not benign. Mild hyponatremia is associated with subtle changes in gait and associated with increased risk of fractures. Even mild asymptomatic hyponatremia requires evaluation and treatment.

Classification of hyponatramia

Definition of hyponatraemia based on biochemical severity

'mild' hyponatraemia as a biochemical fininding of a serum sodium concentration between 130 and 135 mmol/l as measured by ion specific electrode.

'moderate' hyponatraemia as a biochemical fininding of a serum sodium concentration between 125 and 129 mmol/l as measured by ion specific electrode.

'profund' hyponatraemia as a biochemical finding of a serum sodium concentration <125 mmol/l as measured by ion specific electrode.

Definition of hyponatraemia based on time of development

'acute' hyponatraemia as hyponatraemia that is documented to exist <48h.

'chronic' hyponatraemia as hypnonatraemia that is documented to exist for at least 48h. If the hyponatraemia cannot be classified, we consider it being chronic, unless there is clinical or anamnestic evidence of the contrary.

Definition of hyponatraemia based on symptoms

Based on the symptoms hyponatraemia are diveded into 'moderately severe' and 'severe'. The distinction is based on selected observations in acute hyponatraemia; those who subsequently die more often experience what we define as severe symptoms than those who live. Moderately severe symptoms caused by brain oedema are less frequently associated with death. Nevertheless, they may rapidly progress to more severe symptoms associated with an adverse outcome.

Patients are probably never truly 'asymptomatic' in the strictest sense of the word. Very limited and subclinical signs such as mild concentration deficits are seen even with mild hyponatraemia .

Table 1: classification of symptoms of hyponatraemia

Severity	Symptom
Mild	Fatigue, gait abnormality, mild concentration deficit
Moderately severe	Nausea without vomiting Confusion Head ache
Severe	Vomiting Cardio-respiratory distress Abnormal and deep somnolence Seizures Coma (Glasgow Coma Scale ≤ 8)

Clinical and anamnestic data should be taken into account when assessing the casual relation between the hyponatraemia and a certain symptom (i.e. to assess whether the symptom has been caused by the hyponatramia or the hyponatraemia by the underlying condition/symptom). The less pronounced (e.g. mild) the biochemical degree of hyponatraemia, the more caution should be taken when considering that the hyponatraemia is the cause of the symptoms. This list is not exhaustive, and all symptoms that can be signs of cerebral oedema should be considered as severe or moderate symptoms that can be caused by hyponatraemia

Table 2: drugs and conditions associated with acute hyponatraemia (<48h)

Postoperative phase
Post – resection of the prostate, post –resection of endoscopic ulterine surgery
Polydipsia
Exercise
Recent Thiazides prescription
3.4- Methyleendioxymethamfetamine (MDMA, XTC)
Colonoscopy preparation
Cyclophosphamide (intravenous)
Oxytocin
Recently started desmopression therapy
Recently started terlipression, vasopressin

Published research suggests using a threshold of 48 h to distinguish “acute” from “chronic” hyponatraemia. Brain oedema seems to occure more frequently when hyponatraema develops in <48 h . Experimental studies also suggest that the brain needs approximately 48 h to adapt to a hypotonic environment, achieved mainly by extruding sodium, potassium, chloride and organic osmoles from its cells . Before adaptation, there is a risk of brain oedema , because the lower extracellular osmolality promotes a shift of water into the cells. However, once adaptation is completed, brain cells can again sustain damage if the serum sodium concentration increases too rapidly. Breakdown of the myelin sheath insulating individual neurons can result in what is called the osmotic demyelination syndrome . Consequently. It is important to distinguish between acute and chronic hyponatraemia to assess whether someone is at greater risk of immediate brain oedema than of osmotic demyelination . Unfortunately, in clinical practice, is often unclear, particularly for

patients presenting to the emergency room. It is often unknown when the serum sodium concentration has started decreasing. If classifying hyponataremia as acute or chronic is ot possible, we have decided to consider the hyponatraemia as being chronic, unless there are reasons to assume it is acute . There is a good reasons for this approach. Chronic hyponatraemia is much more common than acute hyponatraemia and should be managed accordingly to avoid osmotic demyelination .

Classification based on serum osmolality

A measured serum osmolality <275 mOsm/kg always indicates hypotonic hyponatraemia, as effective osmolality can never be higher than total or measured osmolality. In contrast, if calculated osmolality <275mOsm/kg, the hyponatraemia can be hypotonic, isotonic or hypertonic, depending on which osmotially active agents are present and whether or not they are incorporated in the formula.

Classification based on volume status

Patients with hyponatraemia may be hypovolaemic, euvolaemic,or hypervolaemic. Many traditional

diagnostic algorithms start with a clinical assessment ofvolume status. However, it is often not clear if volumestatus in this context refers to the extracellular fluid volume, to the effective circulating volume or to the total body water

Confirming hypotonic and excluding nonhypotonic hyponatraemia

Hyponatraemia with a measured osmolality less than 275 mOsm/kg always reflects hypotonic hyponatraemia.

Estimates of the serum sodium concentration corrected for the presence of hyperglycaemia can be obtained by adding 2.4 mmol/L to the measured serum sodium concentration for every 5.5 mmol/L (100 mg/dL) incremental rise in serum sodium concentration above a standard serum sodium concentration of 5.5 mmol/L (100 mg/dL).

Table 3 causes of non-hypotonic hyponatraemia

Setting	Serum osmolality	Examples
Presence of "effective" Osmoles that rise serum osmolality And can cause hyponatraemia	Isotonic or hypertonic	Glucose Mannitol Glycine Histidine-trytophane Ketoglutarate Maltose
Presence of "ineffective" osmoles that Raise serum osmolality but do not cause hyponatraemia	Isotonic or hyperosmolar	Urea Alcohols Ethylene-glycol
Presence of endogenous solutes that Cause pseudohypontraemia (laboratory artefact)	Isotonic	Cholesterol Protein intravenous immunoglobulins Monoclonal Gammapathies

parameters to use for differentiating causes of hypotonic hyponatraemia

interpreting urine osmolality of a spot urine sample as a first step.

If urine osmolality below100 mOsm/kg, it is recommened that accepting relative excess water intake as a cause of the hypotonic hyponatraemia.

If urine osmolality 100 mOsm/kg, it is recommened that interpreting the urine sodium concentration on a spot urine sample taken simultaneously with a blood sample.

If urine sodium concentration below30 mmol/L, it is recommened that accepting low effective arterial volume as a cause of the hypotonic hyponatraemia.

If urine sodium concentration[30 mmol/L, it is recommened that assessing extracellular fluid status and use of diuretics to further differentiate likely causes of the hyponatraemia.

- Correct interpretation of laboratory measurements requires contemporaneous collection of blood and urine specimens.
- For practical reasons, urine osmolality and sodium concentration are best determined in the same urine sample.
- If clinical assessment indicates the volume of extracellular fluid is not overtly increased and the urine sodium concentration [30 mmol/L, exclude other causes of hypotonic hyponatraemia before implicating SIAD. Consider using the diagnostic criteria listed in Tables 4, 5 and looking for known cause of SIAD
- Consider primary or secondary adrenal insufficiency as an underlying cause of the hypotonic hyponatraemia.
- Kidney disease complicates differential diagnosis of hyponatraemia. Besides possibly contributing to the hyponatraemia, the ability of the kidneys to regulate urine osmolality and urine sodium is often diminished, much like with the use of diuretics. As urine osmolality and sodium may no longer reflect the effects of the regular hormonal axes regulating water and sodium homeostasis, any diagnostic algorithm for hyponatraemia must be used with caution in patients with kidney disease.

3 Treatment of hypotonic hyponatraemia

Fig. 1 algorithm
Diagnosis of hy

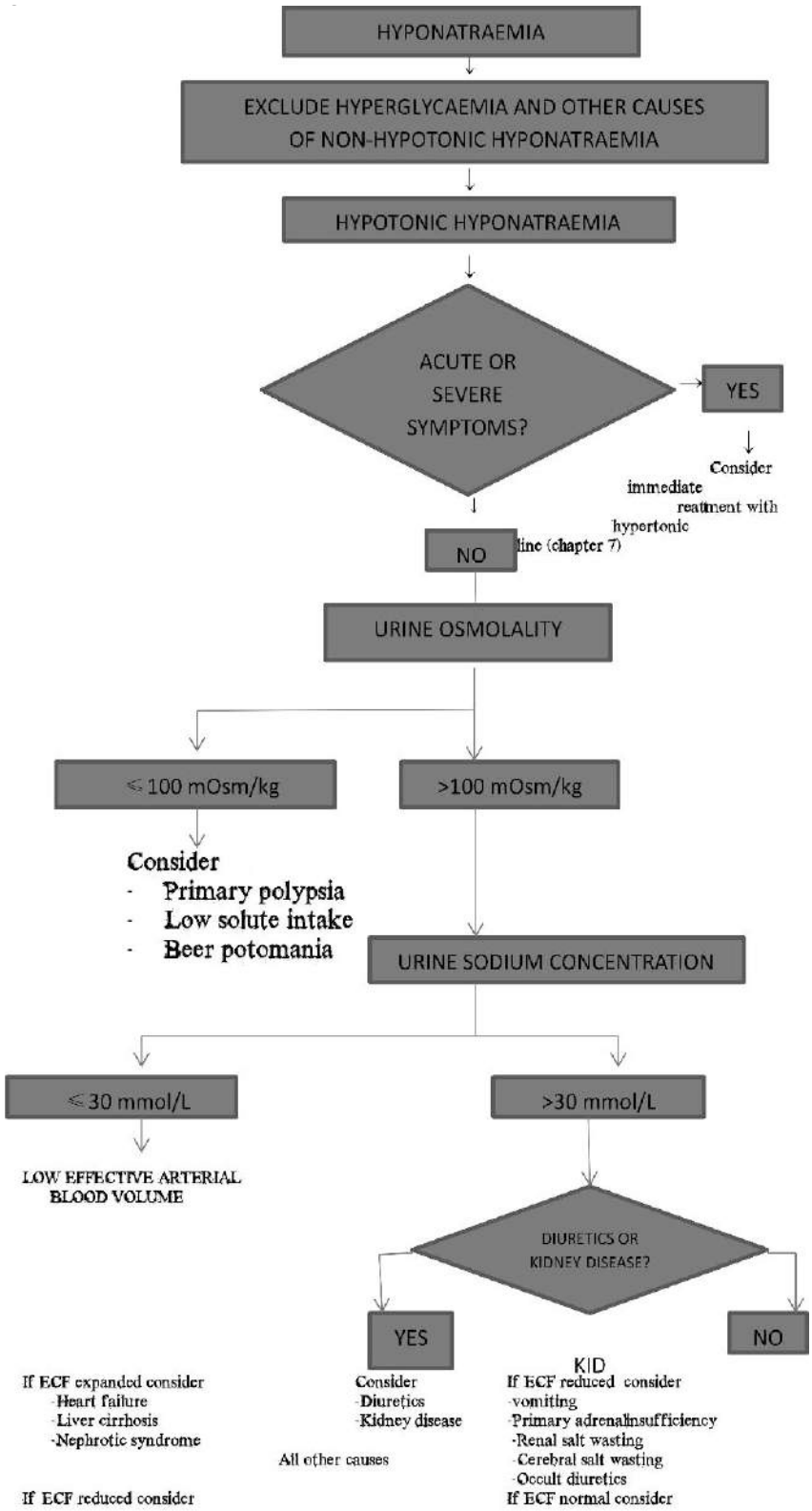

Table 4 diagnostic criteria for the syndrome of inappropriate antidiuresis

Essential criteria
Effective serum osmolality <275 mOsm/kg
Urine osmolality >100 mOsm/kg at some level of decreased effective osmolality
Clinical euvolaemia
Urine sodium concentration >30mmol/L with normal dietary salt and water intake
Absence of adrenal, thyroid, pituitary or renal insufficience
No recent use of diuretic agents
Supplemental criteria
Serum uric acid <0.24 mmol/L (<4 mg/dL)
Serum urea <3.6 mmol/L (<21.6 mg/dL)
Failure to correct hyponatraemia after 0.9% saline infusion
Fractional sodium excretion >0.5%
Fractional urea excretion >55%
Fractional uric acid excretion >12%
Correction of hyponatraemia through fluid restriction

Adapted from Schwartz et al. and Janicie et al.

Table 5 differences between SIADH and cerebral salt wasting

	SIADH	Cerebral salt wasting
Serum urea concentration	Normal- low	Normal – high
Serum uric acid concentration	Low	Low
Urine volume	Normal –low	High
Urine sodium concentration	>30 mmol/L	>>30 mmol/L
Blood pressure	Normal	Normal-orthostatic hypotension
Central venous pressure	Normal	Low

Adapted from Sherloek et al and Brimioulle et al.

Should always be interpreted in the clinical context of the individual patient.

Acute “asymptomatic” hyponatraemia (without severe or moderately severe symptoms)

Make sure that the serum sodium concentration has been measured using the same technique as used for the previous measurement and that no administrative errors in sample handling have occurred.

If possible, stop fluids, medications and other factors that can contribute to or provoke the hyponatraemia.

Start prompt diagnostic assessment cause-specific treatment is recommened.

If the acute decrease in serum sodium concentration exceeds 10 mmol/L. intravenous infusion of 3% hypertonic saline or equivalent is recommended.

We suggest checking the serum sodium concentration after 4 h, using the same technique as used for the previous measurement.

Chronic asymptomatic hyponatraemia (withouT severe or moderately severe symptoms)

General management

Stop non-essential fluids, medications and other factors that can contribute to or provoke the hyponatraemia. (not graded)

cause-specific treatment.

In mild hyponatraemia, treatment with the sole aim of increasing the serum sodium concentration is not recommended

In case of unresolved hyponatraemia, reconsider the diagnostic algorithm and ask for expert advice.

Patients with expanded extracellular fluid

treatment with the sole aim of increasing the serum sodium concentration is not recommended in mild or moderate hyponatraemia.

fluid restriction to prevent further fluid overload.

Do not use against vasopressin receptor antagonists.

We recommend against demeclocycline.

Patients with syndrome of inappropriate antidiuresis

In moderate or profound hyponatramia, we suggest restricting fluid intake as first – line treatment.

In moderate or profound hyponatraemia, we suggest the following can be considered equal second line treatment: increasing solute intake with 0.25-0.50 g/kg/day of urea or a combination of low dose loop diuretics and oral sodium chloride

In moderate or profound hyponatraemia, we recommend against lithium or demeclocycline. (1D)

In moderate hyponatraemia we do not recommend vasopressin receptor antagonists.

In profound hyponatraemia, we recommend against vasopressin receptor antagonists.

Patients with contracted circulating volume

restoring extracellular volume with intravenous infusion of 0.9% saline or a balanced crystalloid solution at 0.5-1.0 mL/kg/h.

Manage patients with haemodynamic instability in an environment where close biochemical and clinical monitoring can be provided.

In case of haemodynamic instability, the need for rapid fluid resucitataion overrides the risk of an overly rapid increase in serum sodium concentration

Advice for clinical practice

• A sudden increase in urine output to >100 mL/h signals increased risk of overly rapid rise in serum sodium concentration. If vasopressin activity is suddenly suppressed, as happens when intravascular volume is restored in hypovolaemia, free water clearance can dramatically increase, resulting in serum sodium concentrations rising more rapidly than expected. If urine output suddenly increase, we would advise measuring the serum sodium concentration every 2 h until it has stablished under stable treatement. The implicit advice to monitor urine output does not imply we advice a bladder catheter solely for this purpose. Most patients will be able to void spontaneously and collect urine for output monitoring.

• As a means of increasing solute intake, we suggest daily intake of 0.25-0.50 g/kg urea can be used. The bitter taste can be reduced by combining it with sweet tasting substances. The pharmacist may be asked to prepate the following as sachets: urea 10 g + $NaHCO_3$ 2g + citric acid 1.5 g + sucrose 200mg, to be dissolved in 50 – 100Ml water. This will result in a more palatable, slightly sparkling solution.

COMPREHENSIVE MEDICAL
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"DON'T LET DIABETES PULL DOWN OUR FOOT"

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ABSTRACT

In the present age of Global village, non-communicable environmental diseases become a major public health concern. Urbanization, life style changes, socio cultural changes, pollution, increase in stress and tensions among people in the society are the contributory factors for the increase in the prevalence of non-communicable diseases. Worldwide, above 75% of all deaths happens due to non-communicable diseases (NCDs). This is particularly more widespread in underdeveloped and developing countries, due to deprived health system. Diabetes mellitus is one among the most common non communicable diseases.

Globally Diabetes affects 6% of total population. In India, more than 62 million people which is nearly 7.1% of adult population of India have diabetes. It is found to be more common among elderly than the younger group. The combination of genetic susceptibility plus use of high calories diet and decreased activity life style pave way for the higher incidence among the middle class Indian population. Type I and Type II are the two major categories of Diabetes mellitus. Diabetes increases the risk of numerous serious health problems including Cardio vascular disorders, Kidney disorders, Neurological disorders, Foot disorders and so on.

Globally, 70 % of people lose their leg because of diabetes. People with known Diabetes mellitus have 15 times greater prevalence of losing their leg than non-diabetic persons. Peripheral neuropathy, foot deformities, minor foot trauma, infection and peripheral vascular disease are the major contributory factors for the development of diabetic foot Ulcer. Recent technological advanced combined with better understanding of the wound healing process have resulted in a myriad of advanced wound healing modalities in the treatment of diabetic foot ulcers. However, it is imperative to remember the fundamental basics in the healing of diabetic foot ulcers: adequate perfusion, debridement, infection control, and pressure mitigation. Early recognition of the etiological factors along with prompt management of diabetic foot ulcers is essential for successful outcome.

Keywords: diabetes, ulcer, prevention, infection, amputation

INTRODUCTION

All over the world for every 30 seconds one foot is amputated due to diabetes and 85% of this can be prevented if early detection and adequate care is provided. 25% of all diabetics patient will develop foot ulcerations in their life period. One third of diabetic patient will develop significant peripheral neuropathy and /or peripheral vascular done. One third of diabetic patient seek hospital

admissions due to diabetic foot ulcer. The patients face an enormous cost burden for treating diabetes in developing countries like India. The onset of diabetic foot ulcer is not spontaneous and many warning signs precedes. Diabetic gangrene is not heaven sent but is born said Dr. Elliot about 75 years ago. Predicting the factors leading to diabetic foot ulcer and its complication can inform health care professionals to selectively concentrate to prevent amputations. Understanding the predictive factors leading to leg ulcer complication including amputation is paramount for prevention.

Limb loss results due to delay in wound healing, insufficient treatment of foot infection and bacterial resistance..

Numerous trial blessing innovative and explorative researchers are being carried out on diabetic foot ulcers and the methods of curing them from time immemorial. Foot ulcer management requires multidisciplinary approach, by health care specialists. Debridement, offloading, and infection control plays a major part in management of Diabetic foot ulcer. Management of underlying systemic illness, such as hypertension, hyperlipidemia, atherosclerotic heart disease, obesity, or renal disease, is essential. It is also necessary to treat the arterial insufficiency, infection with suitable antibiotics, offloading the area of the ulcer, and wound care. Though there have been many treatment options for diabetic ulcer, optimal results are yet to be obtained.

There are various strategies in wound dressing to facilitate wound healing. If the standard treatment fails to heal the Diabetic foot ulcer, supplementary and advanced treatment modalities would be required. They are comparatively effective and have minimal side effects. They are collagen products (COL), biological skin equivalents (BSE), biological dressings (BD), silver products, intermittent pneumatic compression therapy (IPC), negative pressure wound therapy (NPWT), electromagnetic therapy (EMT), keratinocytes, platelet-derived growth factor (PDGF), platelet-rich plasma (PRP), hyperbaric oxygen (HBOT), topical oxygen, Honey dressing and ozone oxygen etc..

PATHOPHYSIOLOGY

Atherosclerosis and peripheral neuropathy occur with increased frequency in persons with diabetes mellitus (DM).

Diabetes-related atherosclerosis

Overall, people with diabetes mellitus (DM) have a higher incidence of atherosclerosis, thickening of capillary basement membranes, arteriolar hyalinosis, and endothelial proliferation. Calcification and thickening of the arterial media (Mönckeberg sclerosis) are also noted with higher frequency in the diabetic population, although whether these factors have any impact on the circulatory status is unclear.

Diabetic persons, like people who are not diabetic, may develop atherosclerotic disease of large-sized and medium-sized arteries, such as aortoiliac and femoropopliteal atherosclerosis. However, significant atherosclerotic disease of the infrapopliteal segments is particularly common in the diabetic population. Underlying digital artery disease, when compounded by an infected ulcer in close proximity, may result in complete loss of digital collaterals and precipitate gangrene.

The reason for the prevalence of this form of arterial disease in diabetic persons is thought to result from a number of metabolic abnormalities, including high low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL) levels, elevated plasma von Willebrand factor, inhibition of prostacyclin synthesis, elevated plasma fibrinogen levels, and increased platelet adhesiveness.Fig 1(a) & 1(b) shows the arteriolar Hyalinosis and Medial Calcification.

Fig 1(a) :Arteriolar Hyalinosis

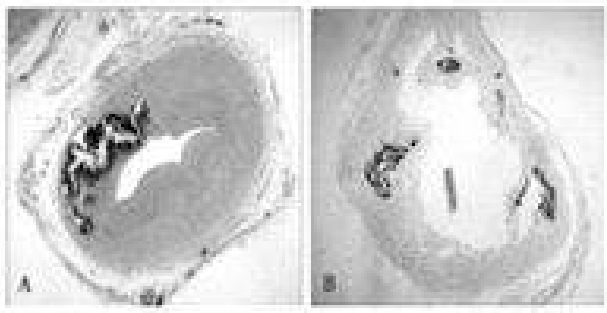


Fig1(b) : Medical Calafeofal

Diabetic peripheral neuropathy:

The pathophysiology of diabetic peripheral neuropathy is multifactorial and is thought to result from

- Vascular disease occluding the vasa nervorum
- Endothelial dysfunction
- Deficiency of myoinositol-altering myelin synthesis
- Diminishing sodium-potassium adenine triphosphatase (ATPase) activity
- Chronic hyperosmolarity, causing edema of nerve trunks
- Effects of increased sorbitol and fructose.

The result of loss of sensation in the foot is repetitive stress; unnoticed injuries and fractures; structural foot deformity, such as hammertoes, bunions, metatarsal deformities, or Charcot foot, further stress; and eventual tissue breakdown. Unnoticed excessive heat or cold, pressure from a poorly fitting shoe, or damage from a blunt or sharp object inadvertently left in the shoe may cause blistering and ulceration. These factors, combined with poor arterial inflow, confer a high risk of limb loss on the patient with diabetes.

Diabetic Peripheral neuropathy will have three component and all these three namely sensory. motor and autonomy neuropathy constitute for the formation of ulcer. The algorithm of Diabetic neuropathy causing foot ulcer are shown in fig 2(a),(b),©.

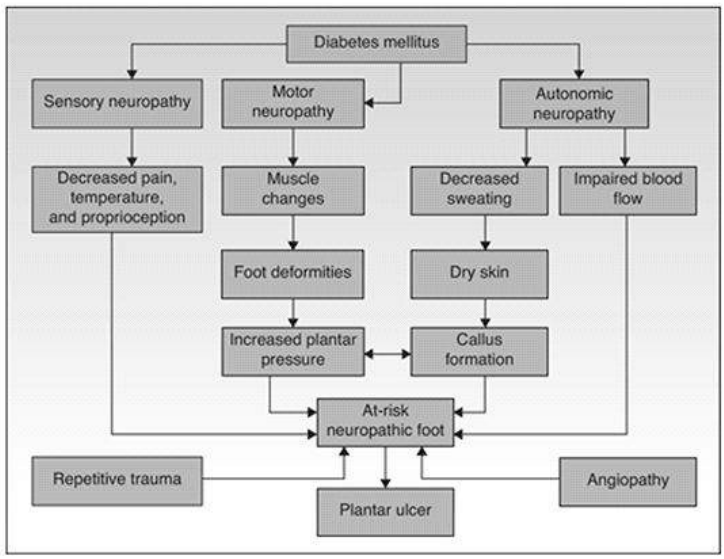
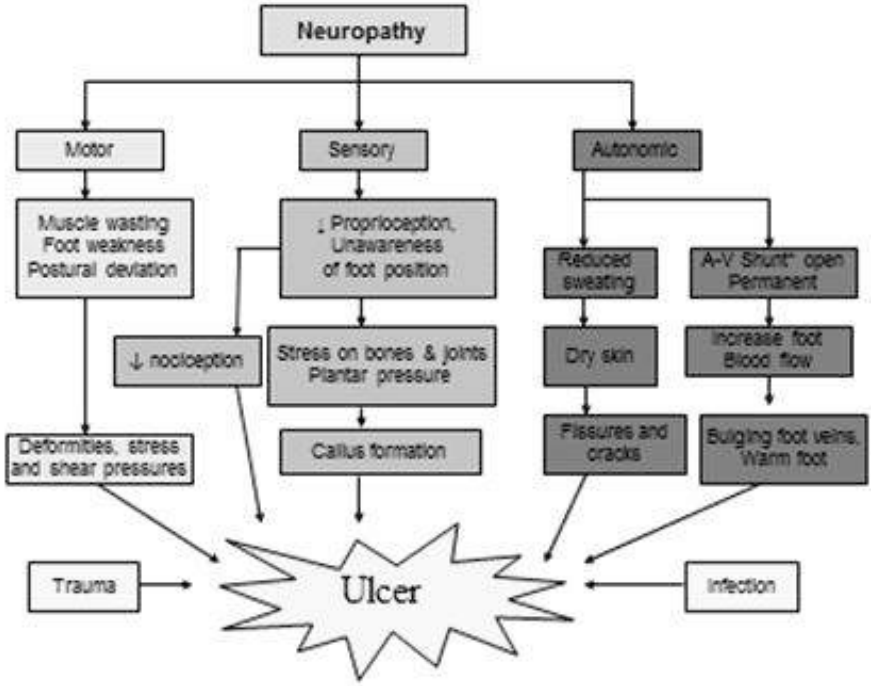
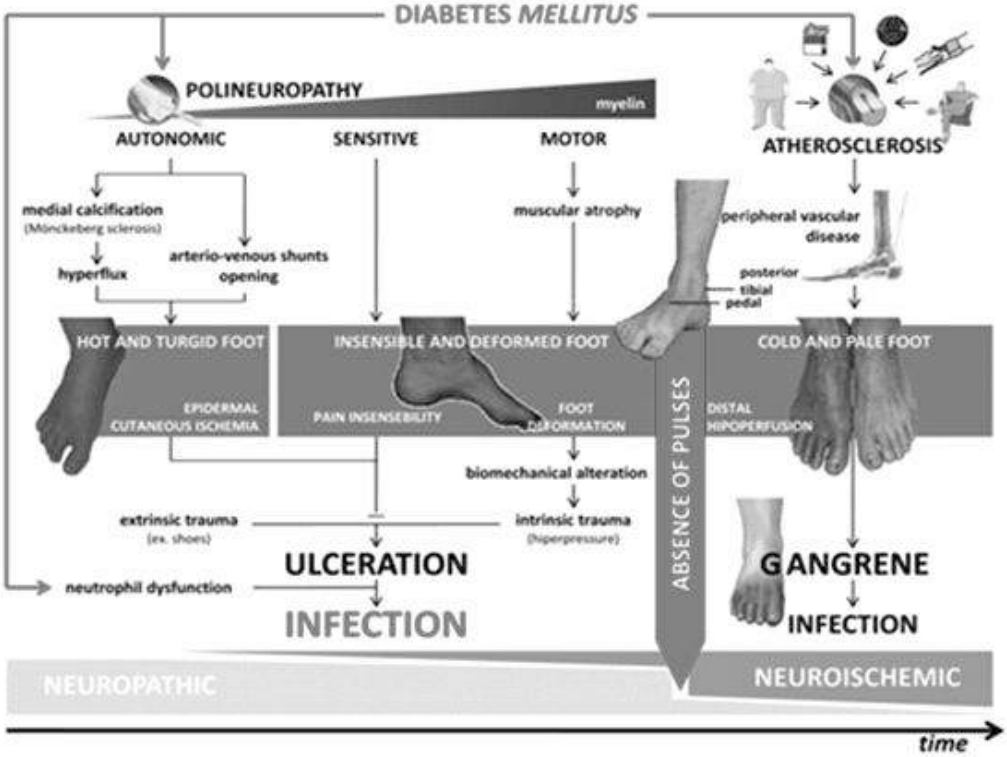


Figure: 2(a),(b),© - Diabetic Peripheral neuropathy causing Foot ulcer.


Neuropathy
Motor
Sensory
Autonomic
Muscle wasting
Foot weakness
Postural deviation
↓ Proprioception,
Unawareness
of foot position
Reduced
sweating
A-V Shunt* open
Permanent
↓ nocioception
Stress on bones & joints
Plantar pressure
Dry skin
Increase foot
Blood flow
Deformities, stress
and shear pressures
Callus formation
Fissures and
cracks
Bulging foot veins,
Warm foot
Trauma
Ulcer
Infection


DIABETES MELLITUS
POLINEUROPATHY
myelin
AUTONOMIC
SENSITIVE
MOTOR
ATHEROSCLEROSIS
medial calcification
(Mönckeberg sclerosis)
hyperflux
arterio-venous shunts
opening
muscular atrophy
peripheral vascular
disease
posterior
tibial
pedal
HOT AND TURGID FOOT
EPIDERMAL
CUTANEOUS ISCHEMIA
INSENSIBLE AND DEFORMED FOOT
PAIN INSENSIBILITY
FOOT
DEFORMATION
COLD AND PALE FOOT
DISTAL
HIPOPERFUSION
ABSENCE OF PULSES
biomechanical alteration
extrinsic trauma
(ex. shoes)
intrinsic trauma
(hiperpressure)
ULCERATION
neutrophil dysfunction
INFECTION
GANGRENE
INFECTION
NEUROPATHIC
NEUROISCHEMIC
time

Staging and Classification of Diabetic Foot Ulcer:

Staging and classification are important for

1. Assume the etiology
2. Predict this prognosis
3. Choose appropriate treatment
4. Monitor program
5. Serve as a form of communication

There are numerous classifications of Diabetic foot ulcer. Based on the etiology the diabetic foot ulcers are classified as

1. Neuropathic ulcer
2. Neuro Ischemic ulcer

The differentiating features of each ulcers are shown in figure 3

CHARACTERISTIC	NEUROPATHIC ULCERS	ISCHEMIC ULCERS
PATIENTS AGE	Younger(fifth to sixth decades)	Older (seventh to eighth decades)
PEDAL DEFORMITIES	Hammer or claw toes, Charcot deformity	None
SKIN	Warm with good colour	Cool, often red
ULCER FEATURES	Wet with drainage, often located on pressure points of planter surface	Dry black eschar, often located on dorsal surface of feet and toes
PULSES	Intact	Diminished to absent
PAIN	Often absent	Often painful

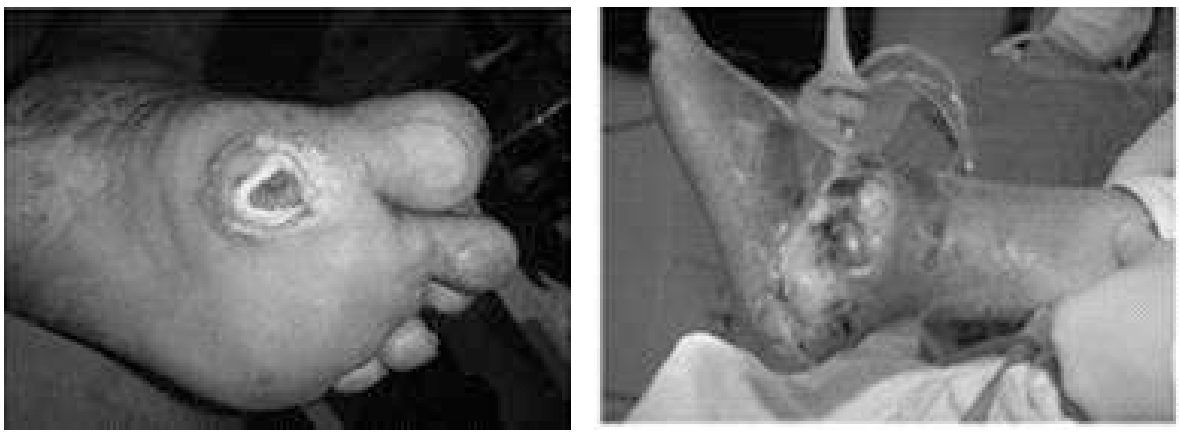
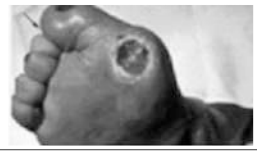


Fig 3: Neuropathy vs Ischemic ulcer

Wagner Classification of diabetic foot ulcers

Wagner classification is based on depth and infection in to 6 grades. However this is not inter connected and hence ulcer with infection in the superficial ulcer will not be included in the classification as shown in fig.4.

Grade 0	Grade 1	Grade 2
No ulcer in a high risk foot 	Superficial ulcer involving the full skin thickness but not underlying tissues 	Deep ulcer penetrating down to ligaments and muscle but no bone involvement or abscess formation 

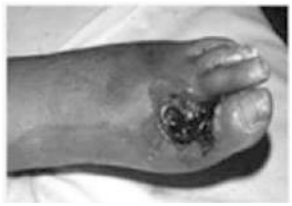
Grade 3	Grade 4	Grade 5
Deep Ulcer with cellulitis or abscess formation often Osteomyelitis 	Localized gangrene 	Extensive gangrene involving the whole foot 

Fig.4-Wagner classification

University of Texas Diabetic Wound Classification System

University of Texas classified foot ulcers based on depth as grade and presence or absence of infection /or ischemia as stages. The classification is shown in Table 1

Stage	Grade			
	0	I	II	III
A (no infection or ischemia)	Pre- or Post-Ulcerative lesion completely epithelialized	Superficial wound not involving tendon, capsule, or bone.	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint.
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	Infection and ischemia	Infection and ischemia	Infection and ischemia	Infection and ischemia

Table 1. University of Texas classification

PEDIS classification is based on the expansion of each letter

- P-Perfusion
- E-Extent
- D-Depth
- I-Infection
- S-Sensation

The system of PEDIS is shown in Table 2.

Grade	Perfusion	Extent	Depth	Infection	Sensation	Score
1	No PAD	Skin Intact	Skin Intact	None	No Loss	0
2	PAD NO CLI	$<1cm^2$	Superficial	Surface	Loss	1
3	CLI	$1-3cm^2$	Fascia, Muscle, tendon	Abscess, Fascitis, Arthritis		2
4		$>3cm^2$	Bone or joint	SIRS		3

Table 2.PEDIS classification

FACTORS CONTRIBUTING FOOT ULCERATION

Multiple intrinsic and extrinsic factors contribute the formation of diabetic foot ulcer as shown in Table 3

Intrinsic factors	Extrinsic factors
• Bony prominences • Limited joint mobility • Deformities • Callus formation • Previous foot ulcer • Neuroarthopathy (charcot)	• Walking barefoot • Inappropriate footwear • Falls and accidents • Objects inside shoes • Thermal trauma • Activity level

Table 3. Factors contributing diabetic foot ulcer

With these intrinsic and extrinsic factor with neuropathy, vasculopathy and immune dysfunction results in diabetic foot ulcer. Fig. 5.

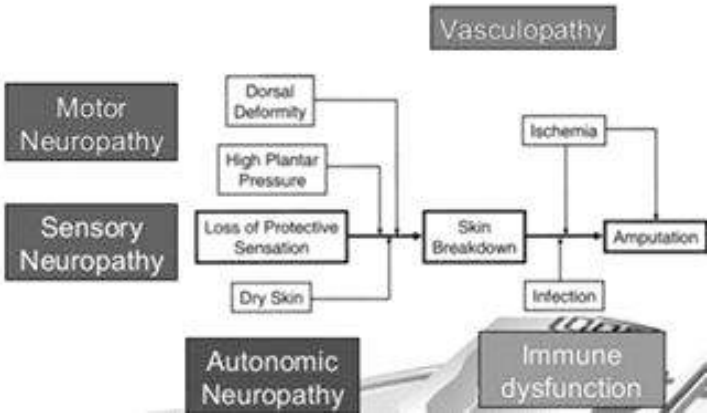


Fig 5. Interplay of various factor in diabetic foot ulcer

Various treatment modalities:

1. Wound debridement :

Never be kind with diabetic foot. All devitalized tissues must be removed till fresh bleeding come. Debridement can be done by

- Surgical (Surgical debridement shown in fig 6 a.)
- Larva (Larva debridement shown in fig 6.b.)
- Hydro surgery
- Autolytic
- Ultrasonic

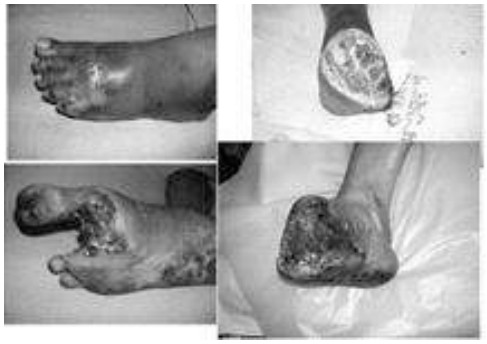


Fig 6.a: Surgical Debridement

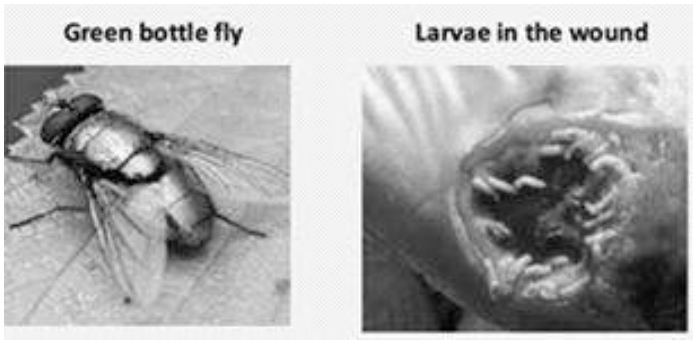
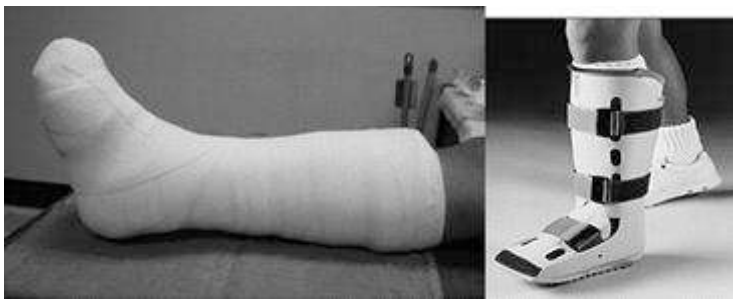


Fig 6.b: Larva for wound debridement

2. Pressure off loading:

Pressure mitigation is the most important treatment in neuropathy ulcer. Either total contact cast or diabetic air walker is used. Using Barometric study on dynamic and static method the potential neuropathic ulcer development can be prevented by giving different pressure soles.(Fig 7 a and b.)



Total Contact Cast **Diabetic Air Walker**

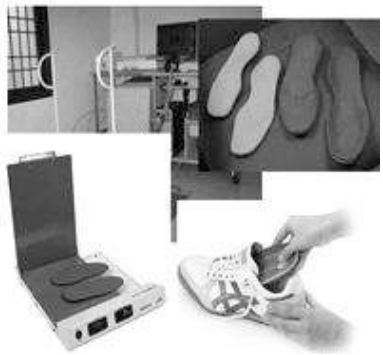


Fig : 7 (a) (b) Pressure Offload methods .

3. Platelet Rich Plasma:

Autologous plasma concentrate delivers high concentration of growth factors in the wound. Growth factors in the PRP initiate chemotaxis, promotes proliferation and angiogenesis can be used as gel, powder preparation or local injections in and around the wound. The details of plasma preparation and results of PRP use are shown in fig 8(a) & (b)

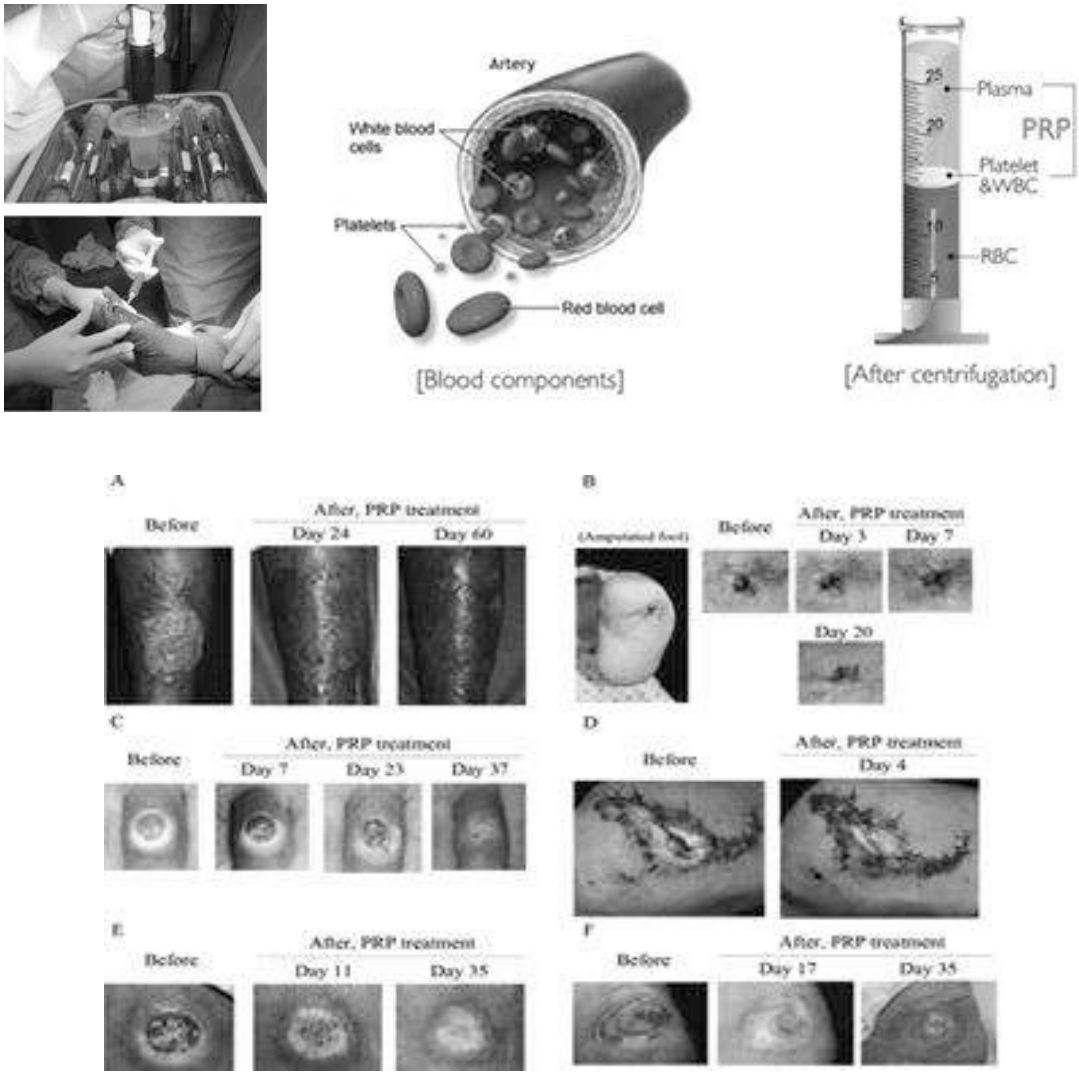


Fig :8 (a) and (b) Platelet rich Plasma

4: Epidermal growth factor:

Recombinant Human epidermal growth factors initiates and promote healing in neuropathic foot ulcer when applied locally.

5. Bone Marrow Aspirate:

Bone marrow stem cells and progenitor cells are potential new therapeutic option to induce angiogenesis. Bone marrow aspirate concentrate is made from fluid taken from bone marrow. The bone marrow aspirate contains stem cells that can help the healing of some bone and joint conditions. Bone marrow aspirate concentrate is obtained with a minimally invasive procedure that avoids the risks of an open bone graft procedure. The instruments and method shown in fig (9).

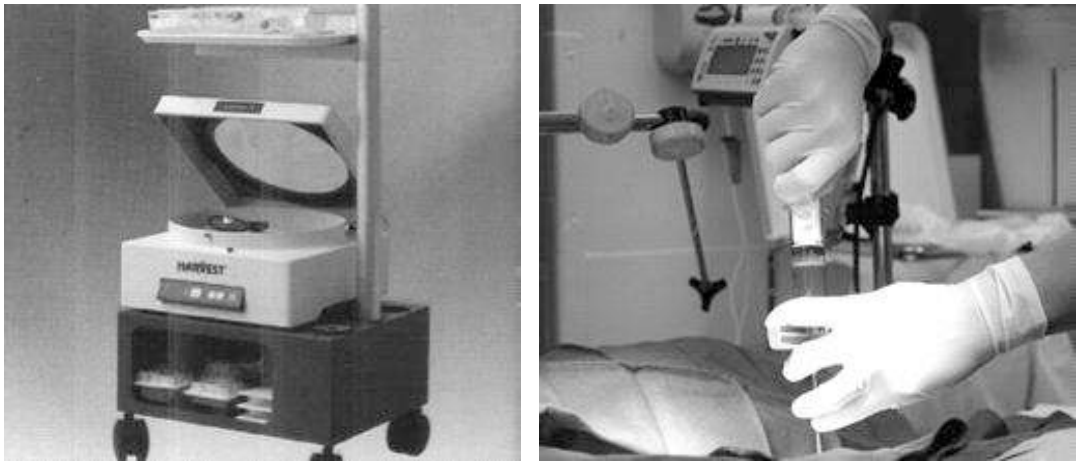


Fig: 9 -Bone marrow aspiration

6. Hyperbaric oxygen therapy:

Increasing the partial pressure of oxygen is the main therapeutic value of hyperbaric oxygen therapy. When a patient breathes pure oxygen at 3 times atmospheric pressure, arterial oxygen pressures in excess of 2000 mmHb are possible. This is around 20 times higher than normal. This is bactericidal to clostridium perfringens, stops toxin production in gas gangrene, and more rapidly displaces carbon monoxide from possible neurological damage. Fig 10.

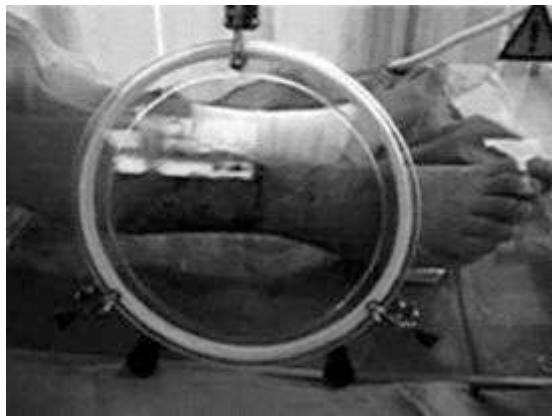
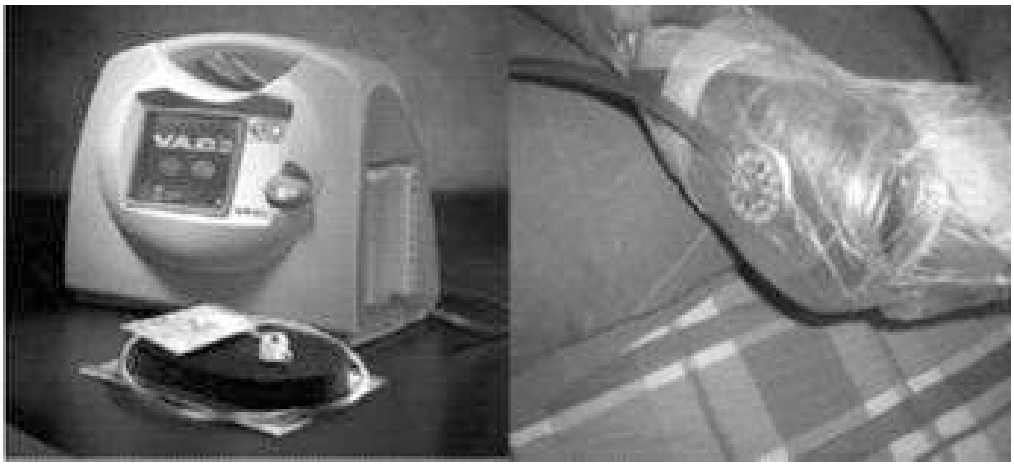


Fig 10: Hyperbaric oxygen therapy unit

Primary effects of HBOT:

- Vascoconstriction
- Angiogenesis
- Leucocyte oxidative killing
- Antibiotic properties
- Toxin inhibition

7. Negative pressure wound therapy/VAC:



VAC therapy applies sub atmospheric pressure to the wound to create an environment to promote wound healing by granulation tissue formation

VAC has 3 components

1. VAC therapy unit: provides intermittent and continuous negative pressure with safety features
2. Sensa T.R.A.C. Technology: regulates pressure
3. Granufoam / White foam
   - Removes infectious materials
   - Provides protected wound healing environment
   - Removes exudate
   - Reduces edema provides moist healing environment
   - Promotes perfusion
   - Facilitates cell migration and proliferation

8. Direct Arterial surgical intervention:

Endovascular: Balloon angioplasty +/- stent

Surgery: Bypass

- Anatomical
  - Aorto-bifemoral
  - Ileo-femoral
  - Femoro-popliteal
- Extra-anatomical
  - Axillo-bifemoral
  - Femoro-femoral

9. Tenotomy

Using open method or needle the flexor contracture is released by cutting the flexor tendon capsule. This procedure may be used in the treatment of flexible digital contractures, and is especially useful in distal digital hyper keratotic lesions and/or distal digital ulcerations or in the neuropathic or diabetic patient with preulcerative or ulcerative distal lesions. This minimally invasive technique has been expanded to include flexor dominant hammer toe deformities, hallux malleus deformities, and floating digital deformities.

The key to choosing this procedure is that the digital deformity must be flexible or semi-rigid at the inter phalangeal joint level and no contracture or a reducible deformity at the metatarsophalangeal joint level (Figure 11),

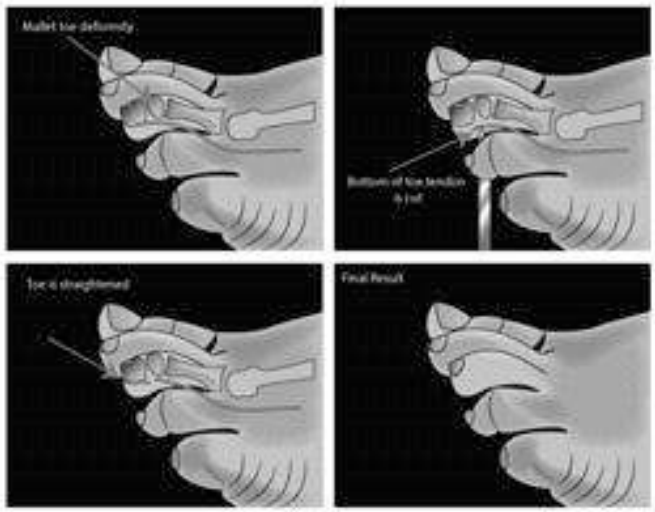


Figure 11 : Tenotomy

10. TA Lengthening:

In severe contracture of the foot ,the tendoachilic tendon is partially cut and patient is put on complete cast for 6 weeks. Achilles tendon is one of the main biomechanical stresses that led to the ulceration.

TA lengthening surgery is perfomed through a longitudinal incision at least 7cm in length. The subcutaneous tissue must be carefully dissected to reduce risk of injury to the sural nerve. The deep fascia and paratenon must be carefully separated and then anatomically re-approximated. This procedure helps in healing of long standing forefoot ulcers in diabetic foot patients & also helps in preventing recurrence of ulcers at forefoot.

Conclusion:

Prevention is better than cure and most often the Diabetic foot ulcer can be prevented when the affected patient follow adequate foot care as follows

Educate patients on proper foot care-the DO's

- Check your feet every day for cuts, cracks, bruises, blisters, sores, infections, unusual markings
- Use mirror to see the bottom of your feet if you can not lift them up
- Check the color of your legs & feet-seek help if there is swelling, warmth or redness
- Wash and dry your feet every day, especially between the toes
- Apply a good skin lotion every day on your heels and soles. Wipe off excess.
- Change your socks every day.
- Trim your nails straight across.
- Clean cut or stratch with mild soap and water and cover with dry dressing.
- Wear good supportive shoes or professionally fitted shoes with low heels(under 5cm)
- Buy shoes in the late afternoon since your feet swell by then.
- Avoid extreme cold and heat(including the sun)
- See a foot care specialist if you need advice or treatment.

Patient shall be advised not to do

- Cutting down corns or callouses
- Treating in-growing toenails or blisters with a razor or scissors.
- Using over the counter medications to treat corns and warts
- Applying heat with hot water bottle or electric blanket
- Taking very hot baths
- Using lotion between your toes.
- Walking barefoot inside or outside
- Wearing tight socks, garter or elastics or knee highs
- Siting for long periods of time
- Smoking

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Taking on Breast Cancer the Non-invasive Way

CURA

While breast cancer continues to pose life-threatening risk to women globally, its diagnosis presents greater challenges for healthcare providers given the painful nature of screening methods like X-ray mammography. IR thermography, which is a non-invasive breast imaging technology used by CURA's MAMRIT System, can be the best alternative to screen women any number of times to increase their chances of survival many fold.

Calculating the Risk Breast cancer incidences in India are estimated to double by 2025. But studies have shown that unlike other cancers, early detection of breast cancer can reduce one-third of deaths. However, using X-ray mammography for screening purpose in younger women population in India poses a challenge, as those with dense breast cannot be imaged frequently.

Women who undergo X-ray mammography once avoid them in future due to painful experience. Hence there is a need for painless breast cancer screening without any radiation such as IR thermography, which can be used on same subject any number of times.

How Thermography Works?

Computerised breast thermography is the visual representation of the distribution of temperature patterns exerted on the skin surface due to the presence or absence of any underlying pathology and the regional vascularity. The thermal patterns are highly sensitive to the environmental factors in the examination area and are a major drawback of the conventional thermography devices. But despite this drawback, thermography has shown results of improving survival rate of over 80 per cent patients in developed countries.

MAMRIT

MAMRIT is a novel product designed and developed by TUSCANO, a CURA company. MAMRIT System ("Mammary Rotational Infrared Thermographic System") for breast imaging is a non-invasive, image acquisition and processing system for monitoring breast tissue changes without any radiation. It comprises a high resolution IR camera, integrated with the inbuilt temperature control and monitoring system and with the multi-axial rotating arm and positioning set up that are enclosed within a closed chamber and the special design on its top as the patient couch used for patient positioning. The configuration of the device enables an image is captured all through 360 degrees of each breast.

This "No Touch Mode" breast imaging visualises up to 24 frames of each breast on its side views and on two temperature conditions. Around 100 images are acquired and are used for analysis purpose.

Clinical Outcomes

Based on the recent clinical studies, it is found that the device can be used for:

• Screening purpose – as a primary imaging modality prior to ultrasound as additional information from thermography can be used as baseline for ultrasound.

• Therapy effectiveness monitoring – in case of chemotherapy to find whether the patient is responding to the drug and/ or its characterization.

Compatibility with other imaging modalities

The various frames captured in thermography can directly be correlated with ultrasound clock position. Also, the acquired thermal images are stored in DICOM format and there is a provision to import other modality images for comparison with the thermography images facilitating clinical analysis.

It is the “first and simple solution” providing all necessary inputs for further evaluation towards diagnosis. For younger women, it can be used along with ultrasound for periodical followups until a clinical course of action towards correction can be taken. IR based thermography can be used any number of times among elderly women for periodical monitoring. For cancer patients undergoing chemotherapy, this can be a facilitating tool to find tissue response to treatment and further on changing the course of treatment.

Patient Benefits

- No radiation exposure
- No contrast injection
- No painful breast compression
- Non-invasive
- No touch
- Good comfort through design for climbing and resting during examination
- High privacy (no exposure of breast to examiner)

Clinician Benefits

- Increased Productivity (digital information is enough for review)
- Programmable profile for a patient
- Easy data retrieval and format (compatible with other medical imaging devices)
- Storage facility for previous visits

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ECG Quiz

Dr. B A. Muruganathan., M.D., FRCP (Glasg, London, Ireland)., FACP (USA)., FPCP (Philippines)., FICP.,
Shristi A. G. Hospital, Tirupur, Tamilnadu.

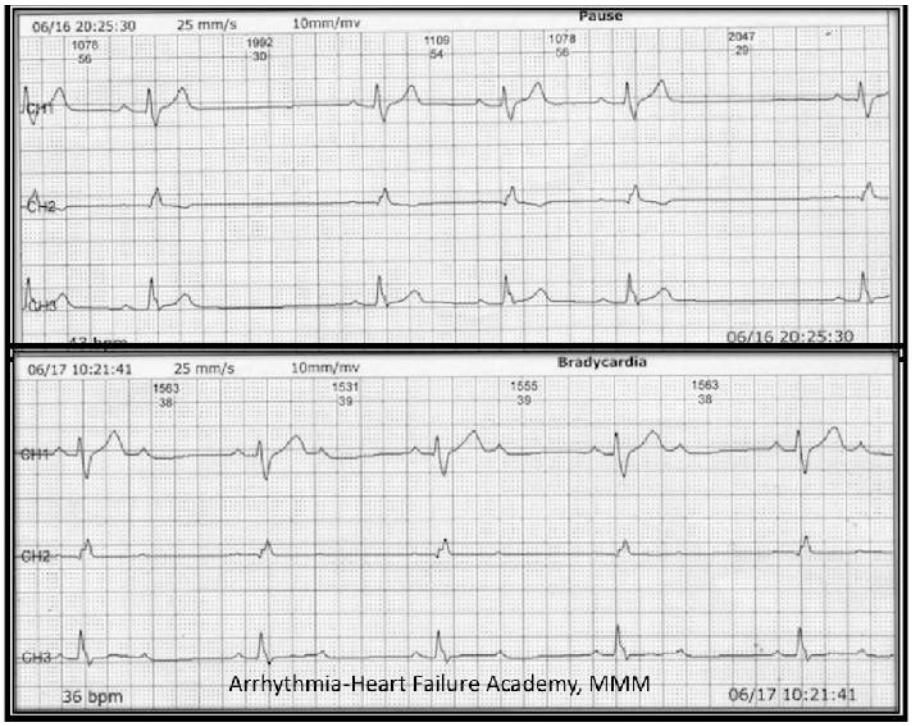
A 60 yr, M, fatigability for the last 3 months. Normal clinical parameters, blood tests and imaging studies. No H/O drugs.

What is the ECG diagnosis?

Dr Ulhas Pandurangi
Chief: Dept of Cardiac Electrophysiology and Pacing, Madras Medical Mission

Answer

The Holter strips reveal bradycardia which may explain patient's symptoms. The PR intervals (200-240ms) are consistent with first degree AV Block. The QRS is wide (140ms) suggestive of Bundle Branch Block. The upper panel shows sinus bradycardia and two episodes of sinus pauses (up to 2 sec). The lower panel shows every second P wave is blocked. The second P wave is appearing 0.8sec after the first. In normal circumstances, it should have been conducted. The above ECG findings: Sinus Bradycardia, Sinus pauses, AV Block, Bundle branch block should prompt further studies, e.g.TMT, to prove presence of chronotropic incompetence as the cause of symptoms. Electrophysiological Study may also be required. A combination of sick sinus disease and AV Block is not uncommon. A rate responsive dual chamber pacemaker should be considered in symptomatic patients.



A RARE CASE OF SCRUB TYPUS WITH PANCREATITIS

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Abstract

Scrub typhus or Bush typhus , a form of typhus, caused by Orientia (formerly Rickettsia) tsutsugamushi, an intracellular parasite, is an acute infectious disease of variable severity that is transmitted to humans by an arthropod vector of the Trombiculidae family. “Tsutsuga” means small and dangerous and “mushi” means insect or mite. It affects people of all ages including children. Humans are accidental hosts in this zoonotic disease. Clinical severity of scrub typhus ranges from mild to fatal. Fever, headache, myalgia, arthralgia and lymphadenopathy are common clinical features of Scrub typhus. Scrub typhus may be complicated by Interstitial Pneumonitis, Acute Respiratory Distress Syndrome (ARDS), Acute Renal Failure, Acute Liver Failure, Meningitis and Myocarditis. Acute Pancreatitis is a rare complication of scrub typhus. We report a case of acute pancreatitis in a 21 years old male with Scrub typhus. Patient presented with high grade fever with abdominal pain for 6 days duration, laboratory investigations revealed elevated pancreatic enzymes and abdominal Computed Tomography showed features suggestive of acute pancreatitis. He was successfully treated with parenteral azithromycin and supportive therapy followed by oral doxycycline. This case is presented for its rarity and to evaluate for Scrub typhus in cases of febrile pancreatitis in endemic area.

Keywords: Scrubtyphus , Pancreatitis, Zoonotic Disease, Rickettsial Disease, Orientia tsutsugamushi

Introduction

Scrub typhus is a widespread Asian zoonotic rickettsial disease that is caused by a parasite, Orientia tsutsugamushi, transmitted by the bite of larval trombiculid mites (chiggers). In South India it is estimated that 40 to 50% of cases of undiagnosed acute febrile illness are attributable to scrub typhus [1]. It starts to grow at the location of the bite and a characteristic skin lesion known as an ‘eschar’ is formed. The rickettsia then spreads systemically via the hematogenous and lymphogenous routes. The infected patient develops various systemic symptoms and reaction including fever, exanthematous rash, myalgia, and diffuse lymphadenopathy. Clinical severity of the scrub typhus ranges from mild to severe, sometimes fatal. Reported severe complications of scrub typhus include Pneumonitis, Acute Renal Failure (ARF), Acute Respiratory Distress Syndrome (ARDS), Myocarditis, and Septic shock. Acute pancreatitis, though common in dual infection(Scrub typhus with Leptospirosis) [2], rare in scrub typhus infection alone[3].

Case report:

21-year-old male, farmer from Nellore (Andra Pradesh), non-alcoholic, was admitted with high fever, myalgia, dyspnea and abdominal pain for 6 days. No H/O of drug intake and no significant past medical or family history. On admission, he was icteric, tachypneic, dyspneic, febrile (104 F), with pulse rate of 117beats/min, respiratory rate of 22/min and blood pressure of 110/60 mmHg. Eschar was seen over the left axilla (Fig no 1). On abdominal examination he had epigastric

tenderness with hypoactive bowel sounds and splenomegaly. Lab investigations revealed neutrophilic(91%) leucocytosis, haemoglobin of 14.2 g/dL with Hct 38.2%, platelet count of 1,66,000/mm3 and CRP 15.0 mg/dl . Serum Liver, Renal and Pancreatic profiles were as follow: total bilirubin 2.4 mg/dL; ALP 114 U/L; SGPT 163U/L ; SGOT 89 U/L; albumin 2.1 g/dL; amylase 541 U/L; lipase 645 U/L; BUN 49 mg/dl, creatinine 1.1 mg/dL, calcium 8.5, LDH 895. MPQBC, Leptospirosis(IgM), Dengue, Urine and blood c/s were negative. Serologic test (IgM-ELISA) for Orientia tsutsugamushi came as positive and Weil-Felix test was contributory (OX:K titre 1:80). Viral markers(HIV, HbsAg, HCV) were negative. In view of young age he was investigated for Anti-Nuclear Antibody and Anti ds DNA antibody and was noncontributory. Lipid profile was normal. Chest radiography showed basal alveolar infiltrates (Fig no.2) and Abdominal CT was suggestive of acute pancreatitis with mild splenomegaly. He was treated with inj. Azithromycin 500mg for 5 day with appropriate pain management and fluid management, followed by oral doxycycline. He improved symptomatically and became afebrile in 3 days with normalization of pancreatic enzymes.

Discussion

Scrub typhus is one of the most common neglected infectious disease of rural south-eastern Asia, where an estimated 1 million cases occur each year, mainly among people of engaged in logging, clearing of land, and working in rice field. More than 80% of the patients present with fever, rash, myalgia and generalized lymphadenitis and 50%-80% have an inoculation eschar. The cases are often mild, but pneumonitis, menigo-encephalitis, DIC, or renal failure is commonly observed if left untreated and death may occur as a result of these complications, usually late in the second week of the illness (4). Pancreatitis is a rare complication of scrub typhus. The mechanism responsible for the development of the acute pancreatitis may involve intrinsic inflammatory process via vasculitis, but the precise nature of this mechanism is not well known. Fatality rate of the scrub typhus ranges from 1% to 35%, depending on the infecting strain, host factors, and time of initiation of the treatment. Although scrub typhus with multiorgan failure is a life-threatening disease, a favourable outcome can be expected if appropriate antibiotics are started within 3 days of illness. Doxycycline is drug of choice for Scrub typhus, chloramphenicol being an alternative in very sick cases. Azithromycin is indicated for children, pregnant women and for doxycycline resistant cases. Rifampicin is another alternative for resistant cases.

We present this case for the rarity of Pancreatitis in Scrub typhus and the favourable outcome by timely diagnosis and instituting appropriate antibiotics.

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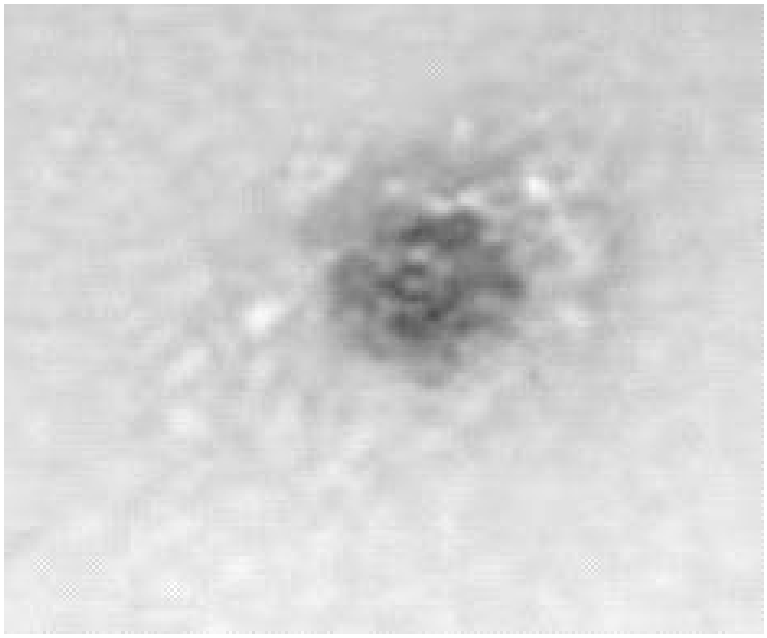

Fig 1 : Eschar

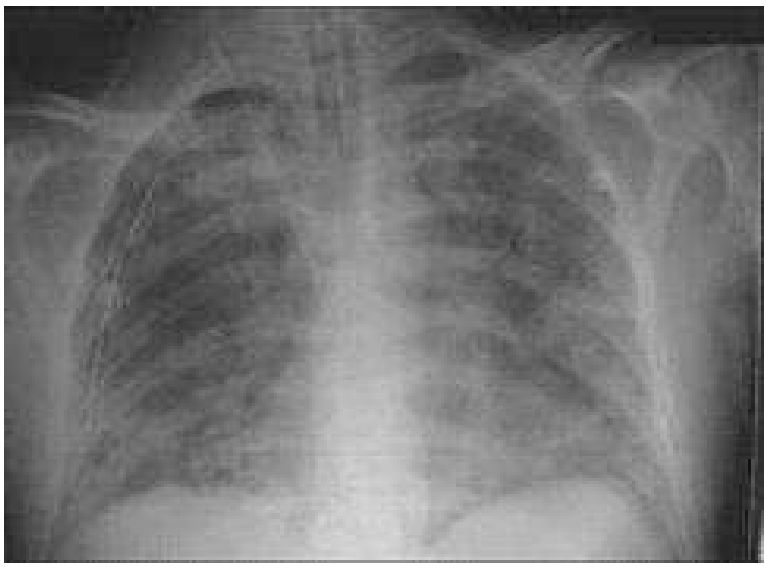

Fig : 2 X – Ray showing bilateral basal infiltrates suggestive of early ARDS.

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LATEST MANAGEMENT IN DIABETIC FOOT AN INDIAN PERSPECTIVE

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Introduction:

Worldwide diabetes is the leading cause of morbidity and mortality. Diabetic Foot (DF) is the major contributing factor for morbidity and mortality among diabetic patients. Global epidemiological studies have indicated pandemic increase in the incidence of diabetes, especially in the Asian countries (1). In developing countries like India, diabetic population increases along with this diabetic co morbidities also increases. In India, DF is the common complication and it is worsen by the socio cultural factors which include bare foot walking, socioeconomics of patients, lack of knowledge on diabetic foot (2). DF infection constitutes 10% of diabetes-related hospital admissions (3). Diabetic patients with foot related problem spend (32.3%) more of their total income towards treatment than diabetic patients without foot problem (9.3%) (4). It is estimated that the median annual direct and indirect cost associated with diabetes care to be 25,391 INR and 4970INR respectively (5).This confront imposed by foot related diabetic problem leads to emergence of preventive strategies, effective intensive management at the early stage of the disease and foot care education in preventing newer problems and surgical interventions (6).

DF Burden in India:

DF complications increase the cost towards their health care which poses a high economic burden to the diabetic patients and in turn to the nation. Among diabetic patients DF is the major cause of hospital admission and it is due to poor socio-cultural practices in India (7). Prevalence of diabetic complications such as neuropathy and peripheral vascular disease (PVD) was high among diabetic patients (8,9). DF infection is the major cause of amputation. A hospital-based multi-centric Indian study highlights that 6–11% of Diabetic patients developed foot infection, and 3% towards amputation (10). In India, it is also reported that diabetic patients with DF infection leads to major (29.1%) and minor (70.9%) amputation, majority of them had neuropathy (82%) and PVD (35%). Further study also showed that infection was a significant cause of amputation in approximately 90% of the study population (11).

Diabetic foot complication is considered as the most important public health problem for patients with diabetes, which eventually progressed to sepsis or gangrene, and extends the hospital stay and significant mortality (12). DF related problem persist worldwide, perhaps there may be variation based on region and clinical presentation. In a study conducted among three different ethnics namely Germany, Tanzania, and India, showed the different underlying risk factors and foot problem among T2DM patients. In a sample of 613 T2DM patients, with average diabetes duration of 14 and 12 years in Germany and India and 5 years in Tanzania. In all the regions neuropathy was common and PVD was high in German (48%), less in Tanzania (12%) and India (13%). In Germany, insufficient footwear is the major risk factor of foot ulcers, whereas in Tanzania and India, irregular foot care and lack of footwear were the common risk factor (13). A multicentre study,

showed prevalence of neuropathy, a major risk factor, was high among South India, than North Indian population. However PVD was common in both populations. Lack of appropriate foot care practices was the major cause of increasing prevalence of foot infection (14).

Management in Diabetic Foot:

Managing diabetes foot complication required multidimensional approach which includes mechanical control; wound control, microbiological control, vascular control, metabolic control and educational control. The intensive foot care education strategies helps in preventing the diabetic related foot problems, recurrent foot ulceration and amputation. A follow up study conducted among T2DM patients with foot ulcer highlighted that those patients who followed the foot care advices healed faster and recurrent ulceration is prevented, whereas those patients who doesn't follow the advices developed reulceration and required surgical intervention (15).

Diabetic neuropathy develops well before the development of foot deformity and gait abnormalities along with appearance of pressure area. Walk continuously without prompt attention to the foot deformity provokes the zone of inflammatory detachment beneath the zone of hyperkeratosis, which leads to mal-performance, and make a way into deeper planes and predisposes to the development of infection. The continued weight bearing is the major mechanical factor which worsens diabetic foot. Other mechanical factor such as ill fitted shoes, acquired deformities, nail disease and prolonged bed rest have to identified and treated. Hard to heal foot ulcer leads to amputation, when combined with the complications such as infection and gangrene.

Treatments such as Vacuum Assisted Closure therapy (VAC), growth factors, Hyperbaric Oxygen therapy (HBOT) are highly recommended for diabetic foot ulcers. The clinical evidences also suggested increased healing rate when compared to standard care of treatment (16).

The negative pressure wound therapy (NPWT) using vacuum dressing improves the healing rate of the chronic wound. This therapy works based on the application of well controlled sub atmospheric pressure to the wound environment, sealed with wound dressing connected with vacuum pump (17,18). VAC is effective in treating DF ulcers, it is efficient in enhancing the skin-graft and improves infection treatment. Thus DF ulcers treated using with VAC showed faster wound bed preparation, increased closure of wound with minimal graft rejection when compared to that of standard care.

Hyperbaric Oxygen Therapy (HBOT), is the better treatment option for the patients with non healing ulcers, thus it is promoted as adjuvant therapy (19). In HBOT, patient's breaths 100% oxygen, under a pressure of greater than sea level, which increases the growth of new blood vessels, reduces swelling and inflammation, further deactivates toxins and other metabolic wastes and improves the healing rate(20) .

The use of growth factors as the therapeutic aids and measuring the cytokine levels are the up-coming trends in regeneration and wound healing. The potential benefits of the growth factors have been demonstrated in both animal models and also in patients with different types of wound healing disorders, for example, GM-CSF (granulocyte-macrophage colony stimulating factor), PDGFs, FGFs (fibroblast growth factors) on the wound healing process (21). Neuropathic ulcers due to the destruction of endogenous growth factors in T2DM patients can be treated with the epidermal growth factor (EGFs) (22). Application of hEGFs (0.04% [w/w]) daily for 12 weeks heals diabetic foot ulcer with significant reduces in healing time (23). The safety and efficacy of the rhEGF (REGEN-DTM 150) in healing diabetes foot ulcer has been evaluated in a phase III study. The findings emphasized that the 69 % of foot ulcer healed in the study group, whereas in control group only 12% has healed. Further in postmarketing surveillance phase IV study of rhEGF, showed

increased wound closure and improved quality of healing of diabetic foot ulcer. Hence growth factor based therapy plays an important role in increasing the wound healing rates and also p[revents amputation (24).

Conclusion:

Preventing peripheral neuropathy by tight glycemic control would be the most effective primary prevention for diabetic foot ulcer or infection; it also prevents the progression of ulcer to gangrene and amputation. Diabetic education is major prevention tool and has to the integral part of prevention strategies. The major practice to prevent diabetic foot ulcer are prompt and regular foot examination, avoid bare foot walking, preventive footwear for patients with high risk foot. Diabetic foot ulcers tend to become chronic and non healing ulcer for various reasons; thus they have to be given an extra care than the routine treatment. The available latest techniques such as VAC, growth factors, HBOT, increase the healing rate of the ulcer. Thus multidisciplinary and holistic approach is a need of this hour to manage diabetic foot. Early diagnosis treatment for PVD and peripheral neuropathy along with regular follow up prevents foot ulcer, apart from strict glycemic control and treatment of ulcer.

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