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Diabetic Foot Infection: Understanding the Disease its Diagnosis and Prevention

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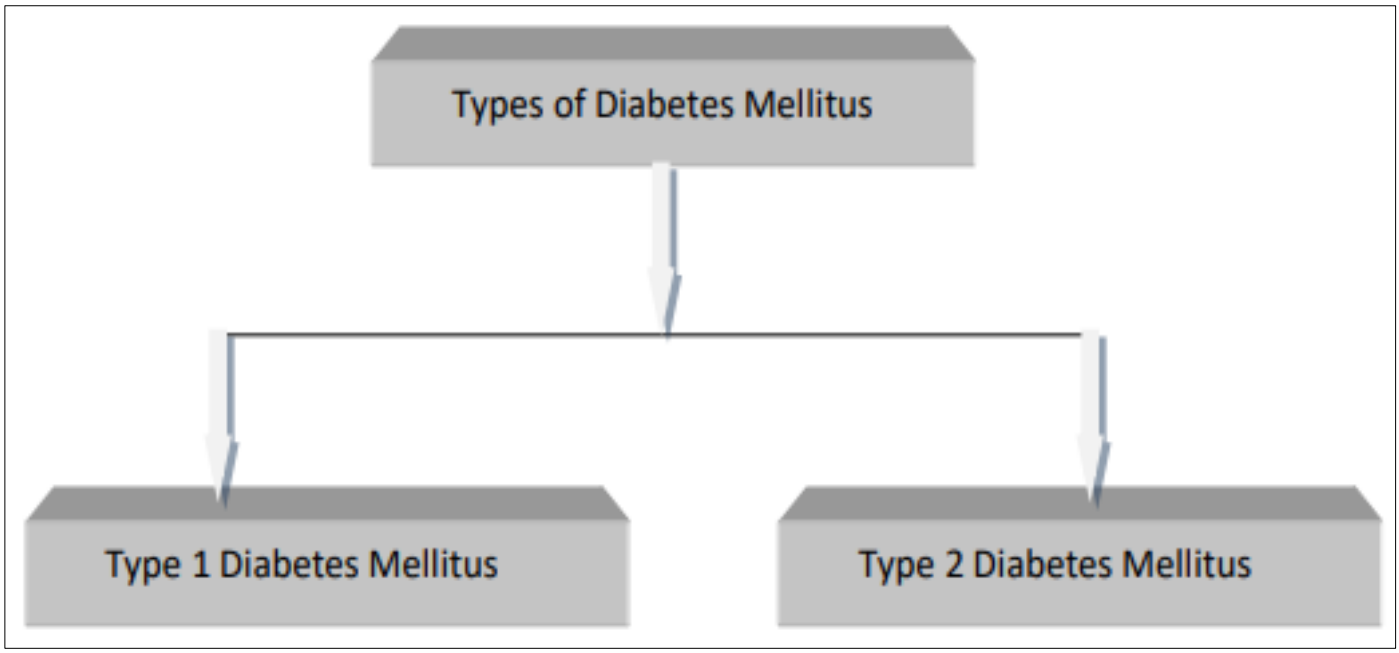
Abstract:- The purpose of this manuscript is to provide a thorough overview of current concepts regarding the evaluation and management of diabetic foot infections. One of the most serious and frequent complications that patients with diabetes mellitus come across is diabetic foot infections. A comprehensive investigation of the literature on diabetic foot infections has been conducted, with a focus on the Diabetes mellitus, pathophysiology, identifiable risk factors, evaluation (including physical examination and laboratory values), treatment approaches, and determining the infection's severity. Preventing severe limb loss and maintaining function are the goals of treating diabetic foot infections. Most diabetic foot infections are caused by polymicrobial organisms. It is probable that a variety of gram-positive and gram-negative aerobes are present at the infection site. Controlling the infection and avoiding more morbidity requires early lesion detection, timely initiation of the proper antibiotic therapy, and surgical excision of necrotic tissue and bone.

Keywords:- *Diabetes Mellitus, Pathophysiology, Risk Factors, Diagnosis, Treatment, Prevention.*

I. INTRODUCTION

A blood sugar (glucose) level that is too high results in diabetes. It develops when your pancreas produces insufficient or no insulin, or when your body isn't responding to the effects of insulin as it deserves.[1] A common characteristic of diabetes mellitus, a metabolic disease, is elevated blood glucose levels that require regular monitoring and appropriate management. Beta cells (β-cells) in the pancreas secrete the hormone insulin, which aids in the uptake of glucose into the cells for energy production and serves multiple different functions as well.[2] Insulin sensitivity or inadequate production is the causes of diabetes mellitus. Type 1 and type 2 diabetes mellitus are the most common forms. Type 1 diabetes (T1DM) is typically associated with a decrease in insulin production due to the destructive impact T-cell-mediated autoimmunity on pancreatic β-cells. Type 2 diabetes mellitus (T2DM) is characterized by insulin resistance and decreased insulin production.[3]

- *Types of Diabetes*



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- *Type 1 Diabetes* –

This type of autoimmune disease happens when your immune system, for unknown reasons, attacks and destroys the cells in your pancreas that produce insulin.[4] T1DM, or insulin-dependent diabetes, occurs when pancreatic β-cells are gradually destroyed by an autoimmune response, resulting in insulin deficiency.[5] Histologic research of the pancreas in a patient with T1DM revealed infiltration of various immune cells including T and B lymphocytes, macrophages, dendritic cells, natural killer cells, as well as islet-reactive auto antibodies and islet-reactive T-cells in the islets of Langerhans. [FIG:-1][6]

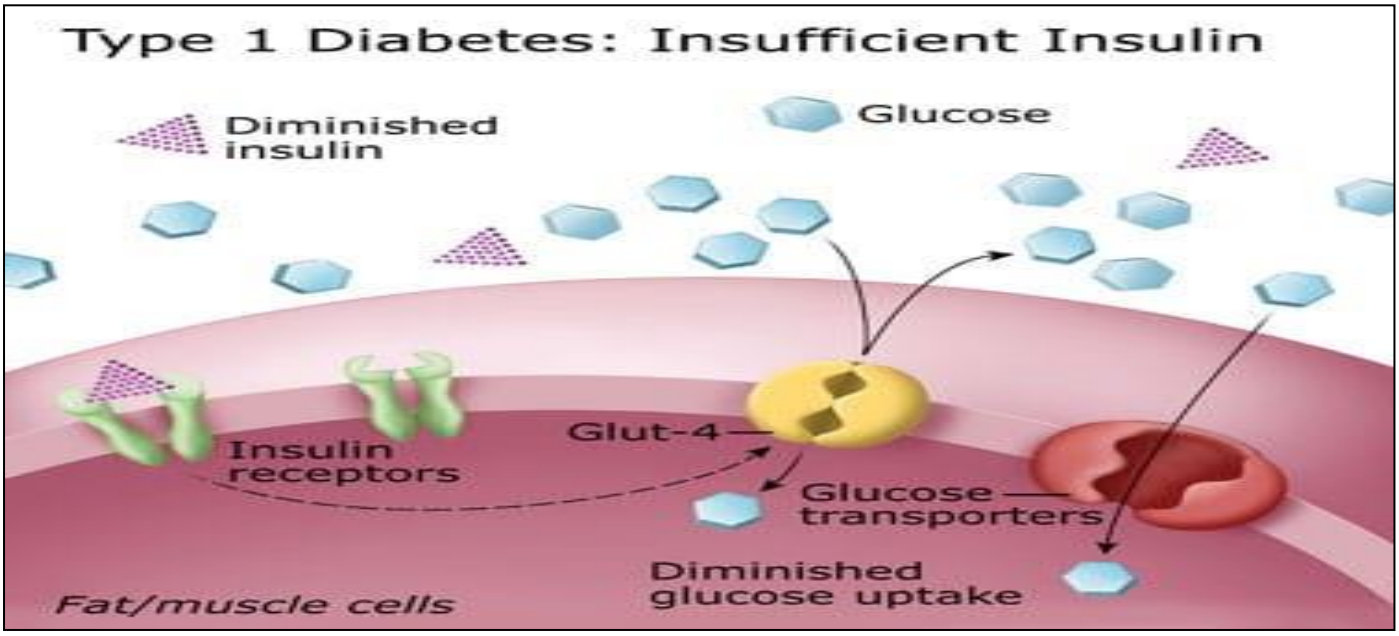

Fig 1 Type 1 Diabetes

- *Type 2 Diabetes* –

This type happens when the body either doesn't produce enough insulin or your body's cells react improperly to the insulin that is produced. It is primarily distinguished by insulin resistance and relative insulin deficiency.[7] T2DM is typically diagnosed in adults, but it is becoming more common in children and adolescents, owing to rising obesity rates. Lifestyle changes, oral medications, and, on occasion, insulin may be used to manage the condition.[FIG-2][8]

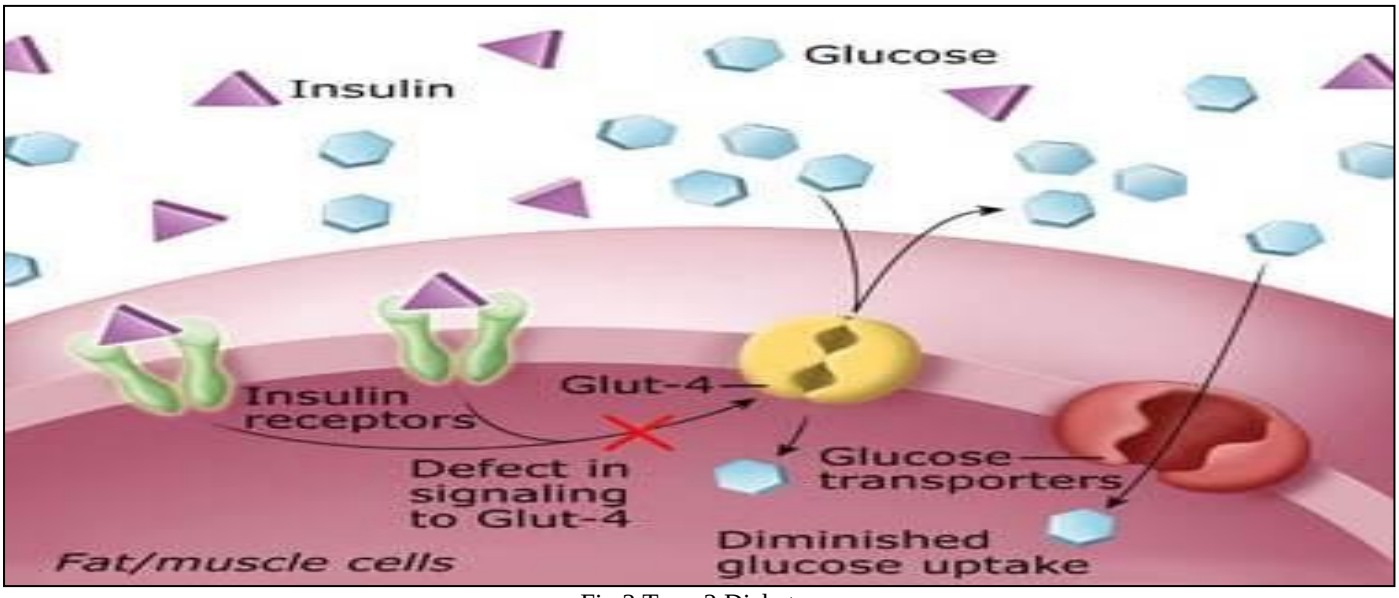

Fig 2 Type 2 Diabetes

Diabetic foot infection and diabetic foot ulcer are significant complications in individuals with diabetes mellitus.[9] These conditions arise due to combinations of factors, including poor blood circulation, neuropathy and an impaired immune response, which are prevalent in diabetic patients. Diabetic foot ulcers are chronic, non-healing wounds that can become infected, leading to severe complications if not managed properly.[10]

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Among diabetic patients, these infections and their after effects are accountable for the majority of hospital admissions and the most typical reason behind disability.[11] Patients from low - and middle - income nations currently represent generally 81% of the prevalence. By 2045, about 665.5 million citizens of these countries will be at risk for diabetes.[12]

In clinical practice, foot infections constitute the many common bacterial infections those patients with diabetic complications knowledge. DFU refers to a wound continuum that begins with a very superficial ulcer and progressing to a deep-tissue infection and finally osteomyelitis. Peripheral neuropathy linked to diabetes damages the distal nerves of the limbs, which reduces pain perception and causes numbness to develop over time.[13]

According to estimates from the International Diabetes Federation, 10 percent of the world's population, or 537 million people, had diabetes in 2021. Over the previous five years, this number has increased by 1.2%. By 2045, this number is predicted to rise to 783 million.[14]

II. MICROBIOLOGY OF DFU

According to recent research, bacterial infection is a major factor in DFU's chronicity. Skin surface bacteria typically infect DFU, which then goes on to form colonies with intricate bacterial polycultures.[15] Even though skin surfaces are frequently the site of bacterial introduction in DFU, eventually obligatory non-native bacteria find a home in the environment that early invaders created. Sometimes, bacteria that inhibit DFU release toxins. Toxins released by bacteria exacerbate wounds and impede the healing process.[16]

Another obstacle to DFU treatments is the isolation of pathogenic bacteria or bacterial strains. Determining the contribution of a single bacterium or a mix of various bacterial species to DFU infections is challenging. Even benign bacteria can serve as a breeding ground for more dangerous bacteria. The maintenance of chronic DFU is primarily carried out by a synchronized combination of cooperating bacteria that form functional pathogenic groups.[17]

The co-aggregated bacteria form a symbiotic association that works in concert to form the biofilm. These bacterial infections interfere with the host's immune system, prolong DFU, are resistant to antimicrobial therapies, and slow the healing process. [18] It is discovered that bacterial infections are multidrug resistant in over 70% of DFU cases.

Therefore, when treating chronic DFU, it's important to determine bacterial diversity, the presence of biofilms, and multidrug resistance.[19]

➢ *Causes[Fig-3][20]*

- **Neuropathy**- Diabetic neuropathy leads to loss of sensation in the feet, which means that minor injuries and pressure sores can go untreated, developing into ulcers.[20]
- **Peripheral Arterial Disease**- Reduced blood flow caused on by PAD raises the risk of infection and impairs wound healing.[20]
- **Hyperglycemia**- Because high blood sugar diminishes the immune system, infections are more common and more difficult to treat.
- **Foot deformities**- Structural changes in the feet, such as Charcot foot, can lead to abnormal pressure points, causing skin breakdown and ulceration.[21]

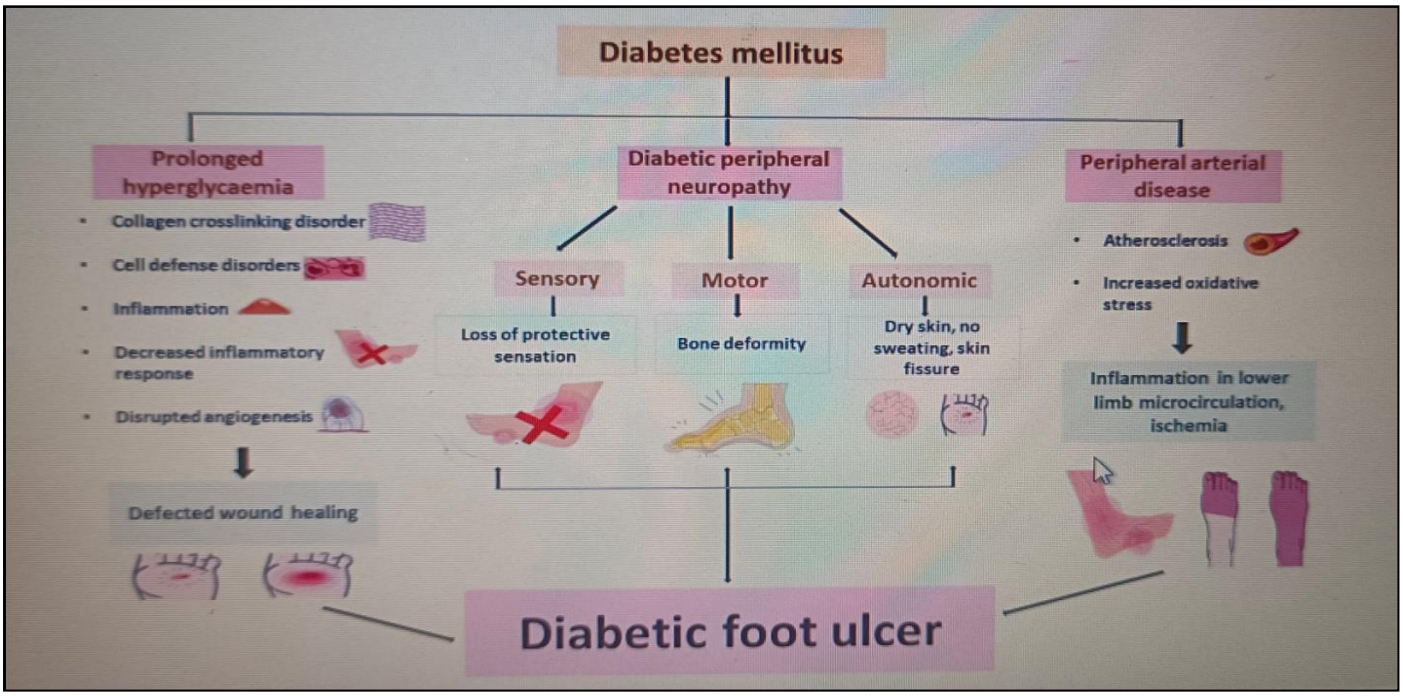

Fig 3 Causes of Diabetic Foot Infection

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➢ *Risk Factors for Diabetic Foot Conditions*

Although the literature currently in publication emphasizing risk factors for diabetic foot ulcers, the body's demonstrated resilience to random variables linked to infections related to diabetes is not as strong.[22]

- Fringe neuropathy
- Vascular (blood vessel) insufficiency
- Irregular life systems and biomechanics
- Hyperglycemia and other metabolic derangements
- Impeded neutrophil work
- Disabled wound mending and overabundance collagen cross-linking
- Infection related disabilities
- Maladaptive conduct from the quiet side
- Insufficient healthcare arrangement

➢ *Types of Diabetic Foot Complications*

The degree of tissue loss, size, perfusion, infection site, depth, area, and sensation can all be used to categorize diabetic foot ulcers.[3] The variations in these parameters, which ultimately classify diabetic foot ulcers into different grades, are dependent on an individual's age, sex, medical conditions, and current multiple conditions, including a loss of peripheral sensation (LOPS) and peripheral arterial diseases (PAD).[23]

According to Meggitt and Wagner, diabetic foot ulcers can be classified into 6 grades:-[FIG-4][24][25]

- Grade zero: Pain, possible hyperkeratotic lesion or bone deformity, absence of skin lesion.
- Grade One: Possible bone deformity, subcutaneous tissue loss, superficial viable or necrotic ulcers, and lack of penetration into the skin's deeper layers
- Grade Two: More profound penetrations, involving deep fascia, tendons, ligaments, and bones; noticeable bone deformity in certain areas; absence of osteomyelitis or an abscess.
- Grade Three: Severe infection symptoms (such as redness, heat, and swelling); the presence of osteomyelitis, a deep abscess, or tendinitis.
- Grade Four: Gangrene (dry, wet, infected, or non-infected) in the forefoot or toe; below-knee amputations require surgical foot ablation with minimal blood supply.
- Grade Five: Amputation below the knee is highly advised when gangrene spreads to the entire foot and shows no signs of healing.

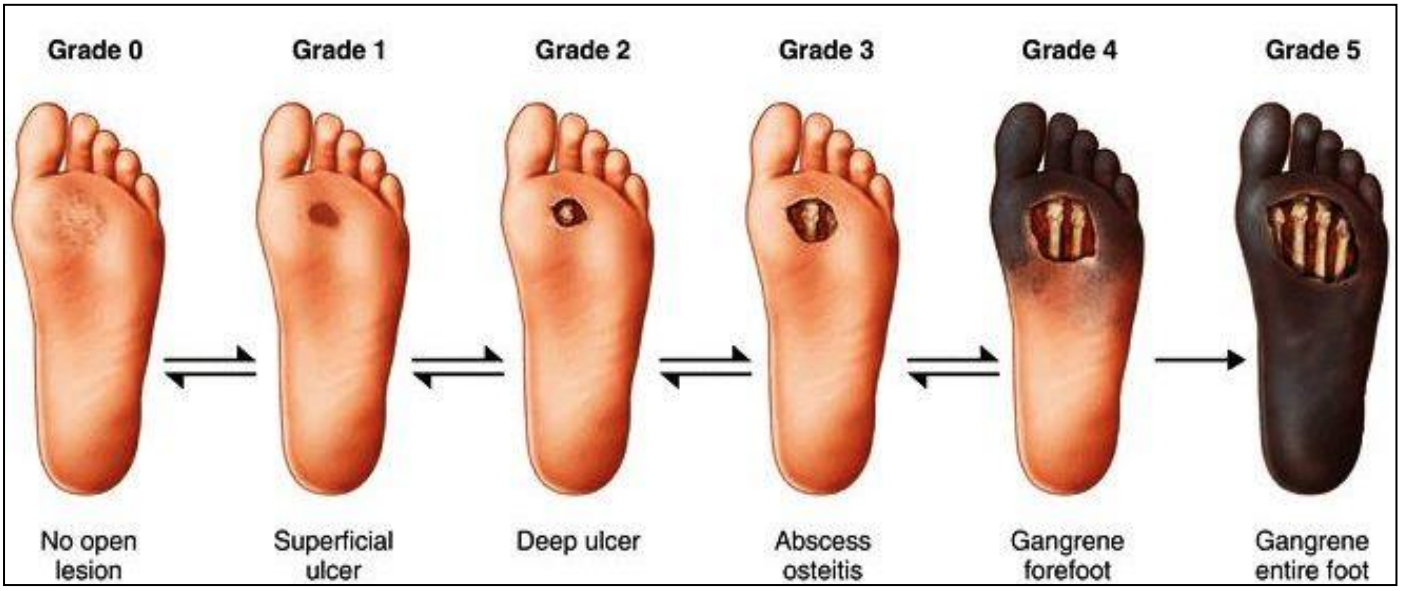

Fig 4 Grades of Diabetic Foot Ulcer

III. PATHOPHYSIOLOGY

DFU is a full-thickness dermal wound that is situated in the exposed or weight-bearing region beneath the ankle. There is a trio that is used to explain the pathologic mechanisms of DFU.[11] This group of three conditions consists of neuropathy, vascular insufficiency, and secondary infection brought on by foot trauma.[26]

Diabetic foot ulcers (DFUs) have a complex etiology. There isn't just one risk factor that causes foot ulcers. It takes a combination of factors to have a significant enough effect to cause ulceration.[FIG-5][27]

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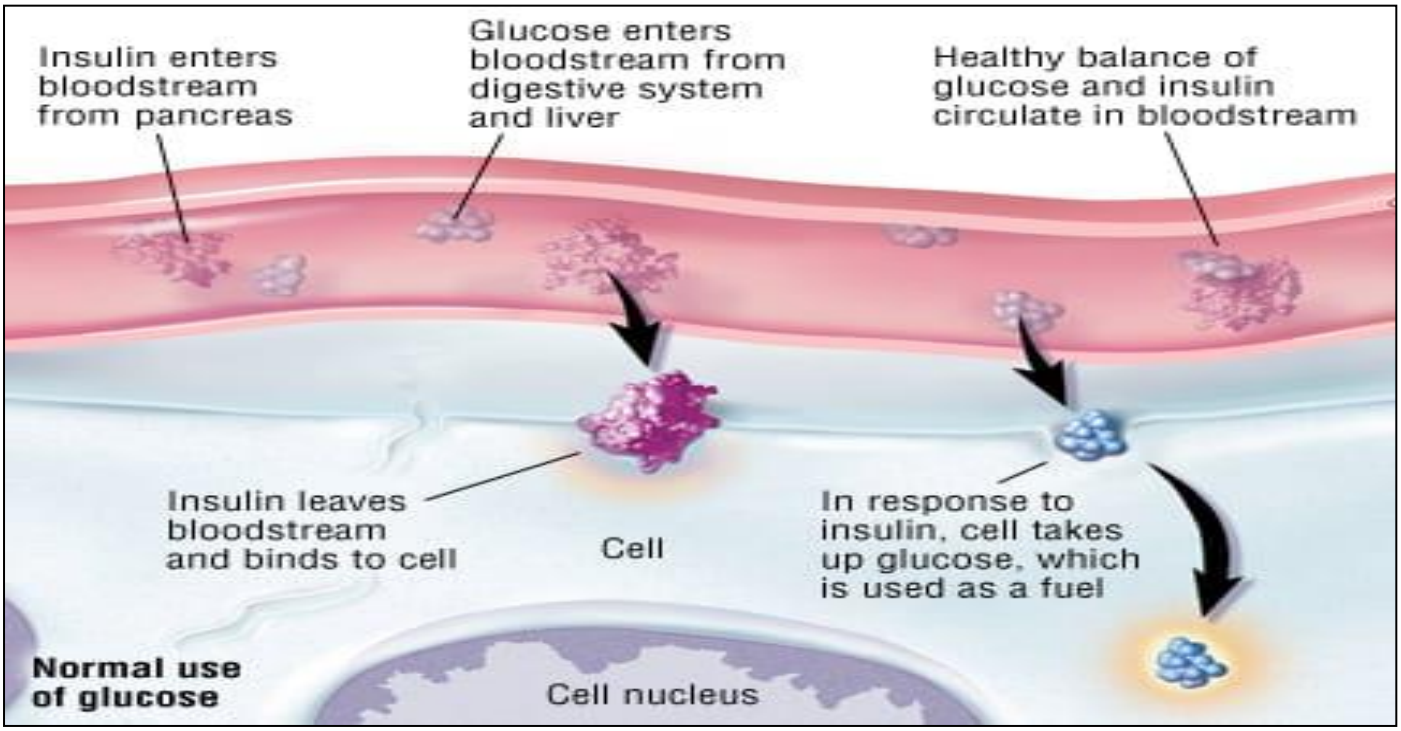

Fig 5 Pathophysiology of Diabetes

➢ *Neuropathy*

Peripheral neuropathy, or loss of sensation, is a common occurrence, accounting for 20% of cases at diagnosis and occurring 8–12 years after type 2 diabetes developments. It is also a contributing factor to the development of ulcers.[28] Diabetic peripheral neuropathy affects autonomic, motor, and sensory nerve functions as well as the regular activities of the nerves throughout the body. Peripheral neuropathy has been reported to occur in 16% to 66% of cases due to diabetes.[29] Peripheral nerves receive insufficient blood supply, blood flow is easily disrupted, and automatic blood flow regulation is compromised. We can comprehend why peripheral nerve neuropathy is different from other complications thanks to these anatomical features. Peripheral nerves are susceptible to ischemia due to these characteristics. Damage to nerve endings in diabetic neuropathy causes action potentials to be interrupted, which causes hyper excitability and pain perception.[30] Advanced glycation end-products (AGEs) must be taken into consideration from a pathological standpoint. Hyperglycemia-induced up regulation of aldose reductase and sorbitol dehydrogenase results in to an increase in fructose and sorbitol production. These glucose products accumulate and cause osmotic stress, which lowers nerve cell myoinositol synthesis and nerve conduction.[31] The patient is highly susceptible to a slight injury without realizing it until it develops into an ulcer when the nerve becomes inflamed. The possibility of patients' foot ulcers getting worse. Compared to non-neuropathic diabetic patients, the sensory loss might raise up to seven times.[32] Diabetes can cause neuronal autonomic dysfunction in addition to sensory neuropathy, which impairs sweat production and leaves the foot vulnerable to dryness, skin cracking, and fissuring.[33]

➢ *Vascular Insufficiency*

In peripheral arteries, hyperglycemia results in abnormalities of smooth cells and malfunction of endothelial cells. Because of changes in endothelial cell proliferation, thickening of the basement membrane, decreased nitric oxide synthesis, increased blood viscosity, changes in microvascular tone, and decreased blood flow, endothelial dysfunction is the most serious impairment affecting microcirculation.[34] Peripheral nerve injury is caused by hyperglycemia by a number of events, including oxidative stress, protein kinase C (PKC) activity, the polyol pathway, adenosine triphosphate (ATP) deficit, and proinflammatory reactions. A lack of ATP causes axonal transport to be hindered, especially in axons rich in mitochondria that produce nerve energy. This can lead to axonal damage and diabetic neuropathy.[35] Along with the trio, poor wound healing has been identified as a major factor in the advancement of DFU. Significantly, molecular alterations at the DFU site occur before the tissue anomalies that are visible to the naked eye. Wounds typically go through numerous phases of healing, including homeostasis, inflammation, proliferation, and remodeling. While chronic non-healing DFUs stall in several phases, acute wounds move through these processes in a linear fashion.[36][Flow Chart- 1]

➢ *Secondary Infection*

In diabetics, the interaction of immunopathy, diabetic neuropathy, diabetic vasculopathy, and metabolic variables facilitates the onset and advancement of infections, ischemic ulcers, and gangrene, which may result in amputation.[37] Neutrophils typically produce granular molecules during the initial stages of wound healing, a process known as neutrophil extracellular traps (NETosis), which kills invading pathogens.[38] On the other hand, NETosis

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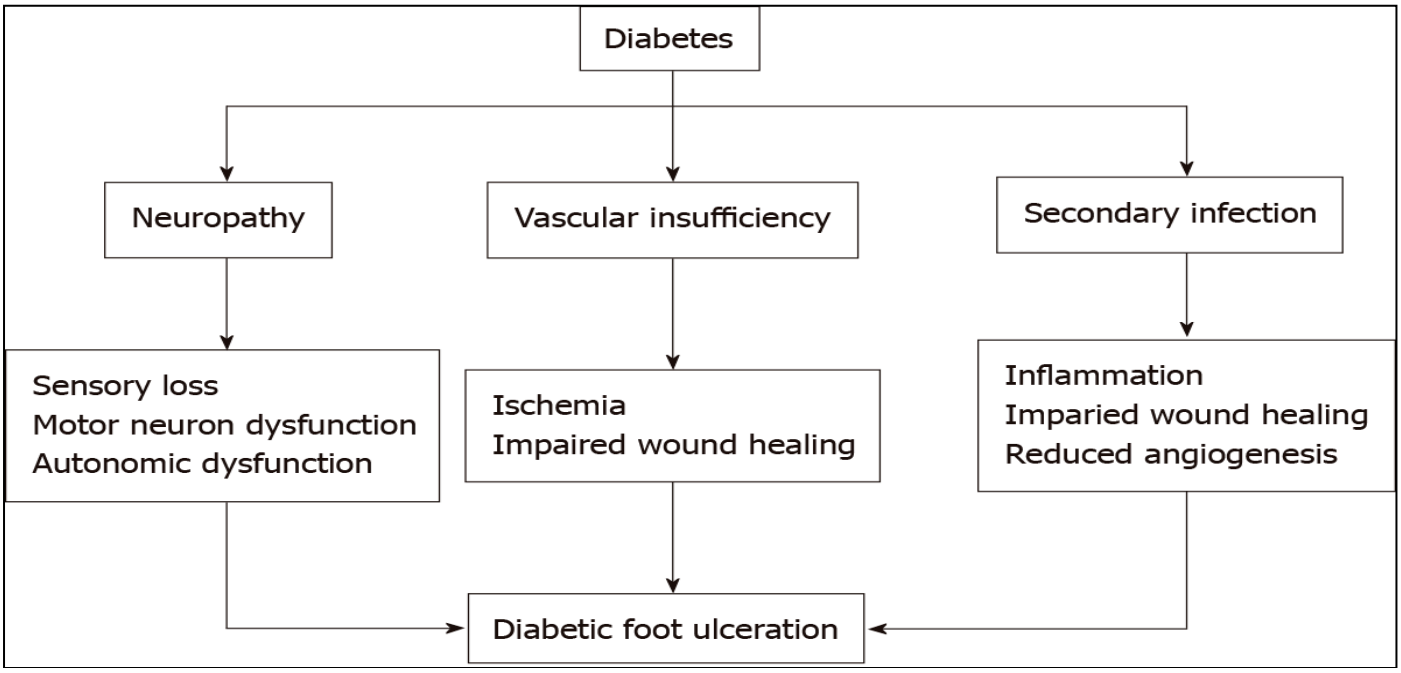
becomes dysregulated in a diabetic milieu, leading to an overproduction of cytokines and superoxide as well as a proinflammatory cascade that delays wound healing.[39] Reduced leukocyte activity, an improper inflammatory response, and disruption of cellular immunity (inhibition of fibroblast proliferation, impairment of the basal layer of keratinocytes, reduction of epidermal cell migration) all damage the immune system. Patients with inadequately managed diabetes showed a considerable reduction in leukocyte phagocytosis, and correction of hyperglycemia was strongly connected with an improvement in microbiocidal rates.[40] DFUs are caused by a variety of bacteria, the most common of which being Staphylococcus aureus. MRSA infections are linked to higher hospitalization rates and a higher risk of limb amputation, even if they don't seem to have an impact on death.[41][FIG-6]



Flow Chart:-1 Pathophysiology of Diabetic Foot Ulcer

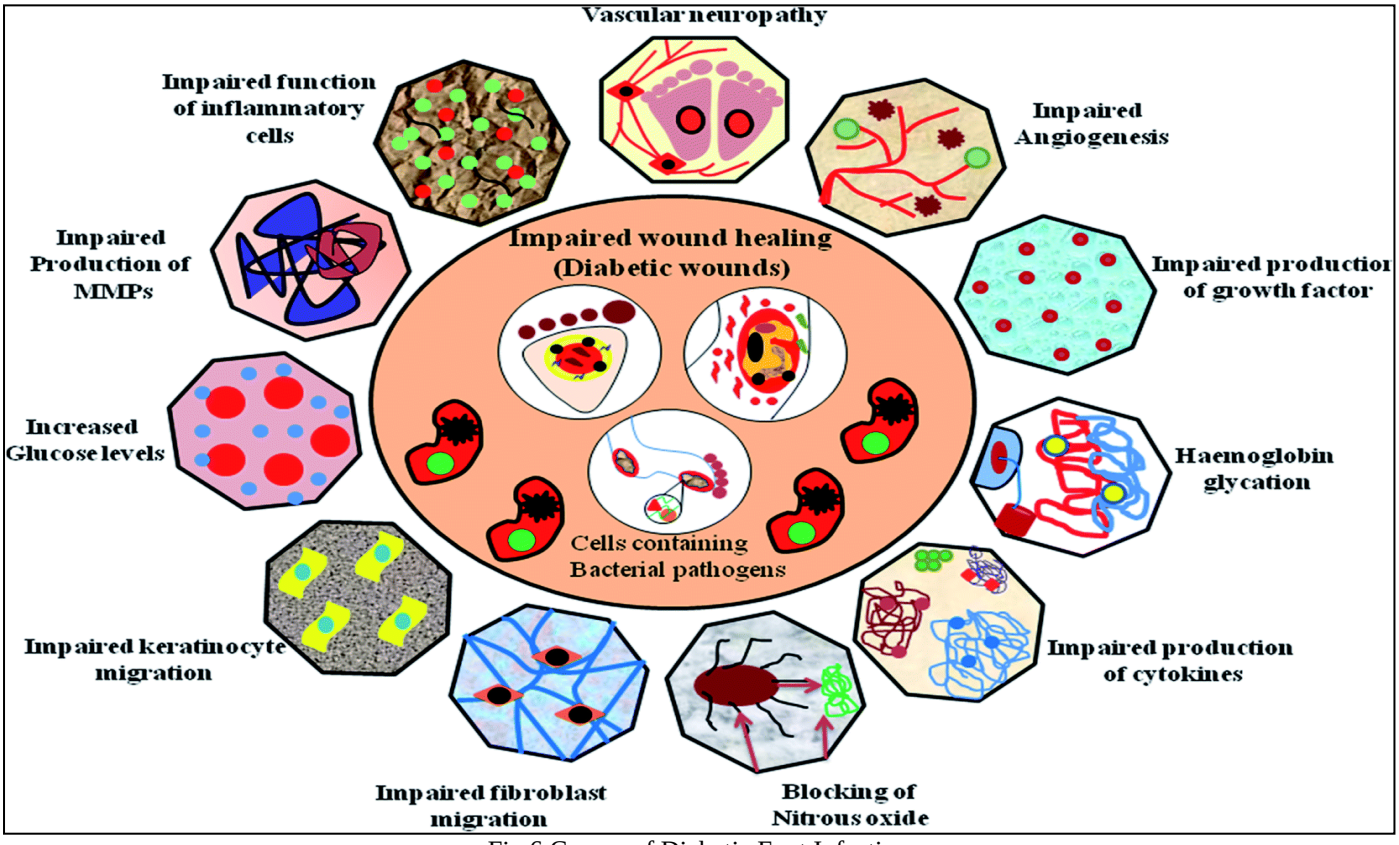


Fig 6 Causes of Diabetic Foot Infection

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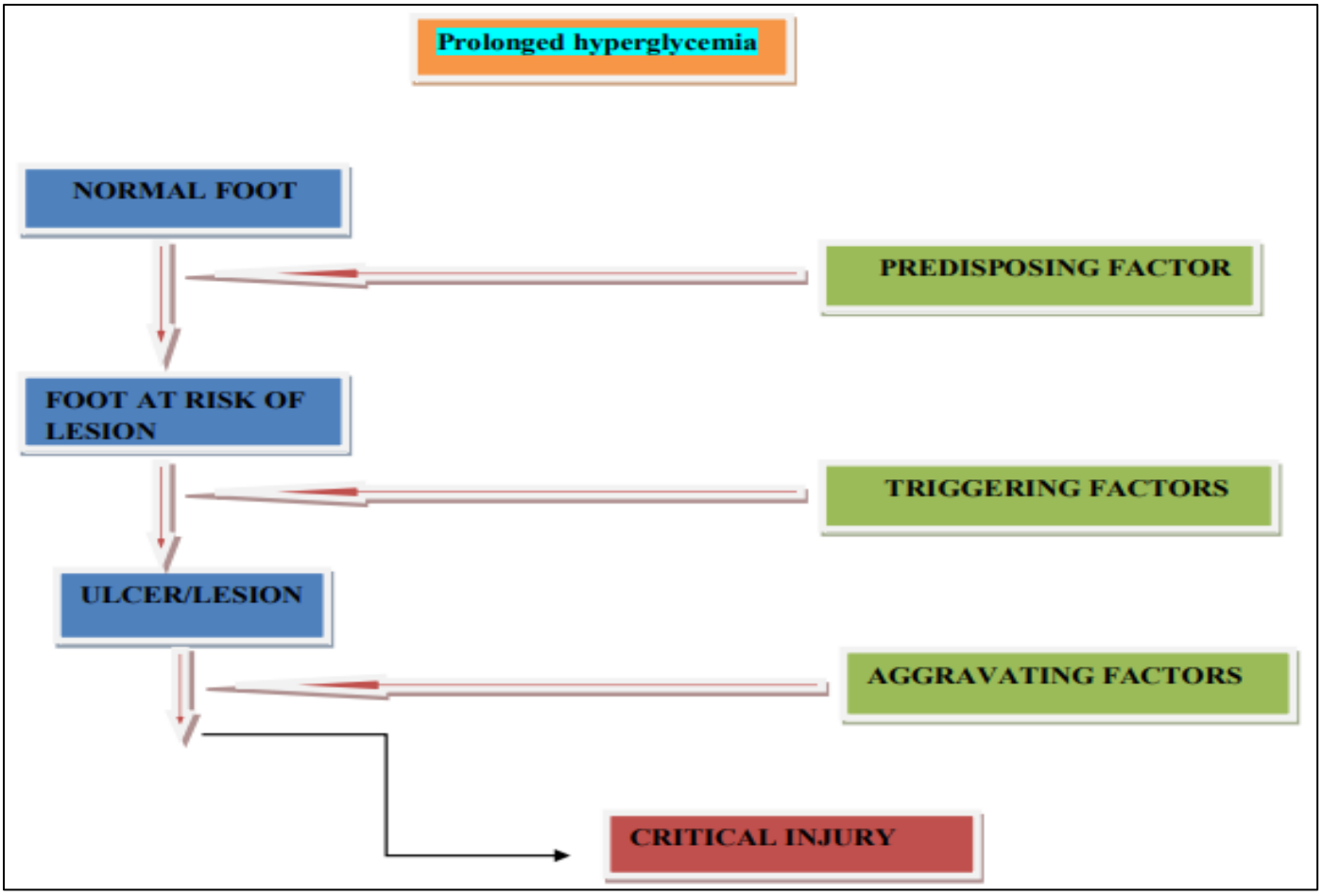
➢ *Flow Chart Depicting Pathophysiology of DFI*



Flow Chart:-2 Pathophysiology Diabetic Foot Infection

➢ *Symptoms [42]*

- **Ulceration -** Open sores or wounds on the feet, often painless due to neuropathy.
- **Infection Signs -** Redness, warmth, swelling, and pus discharge from the ulcer site.
- **Systemic Symptoms -** Fever, chills, and general malaise if the infection becomes severe.

Table 1 Infectious Profile of DFI[43]

INFECTION	MICROORGANISMS
Cellulitis	*Staphylococcus aureus*
Ulcer untreated with antibiotics	*Staphylococcus aureus, Streptococci*
Ulcer treated with antibiotic or on long term therapy	*MRSA, MSSA, CONS, Streptococci, Enterococci, Enterobacteriaceae, Pseudomonas aeruginosa, Corynebacterium spp. Candida spp.*
Necrotizing fasciitis of myonecrosis (generally polymicrobial)	Anaerobic gram positive cocci *Enterobacteriaceae*, Non fermenting gram negative bacilli, anaerobes

➢ *Diagnosis*

- **Clinical Examination -** Examining the feet for abnormalities, infection symptoms, and ulcers.[44]
- **Microbiological Cultures -** To identify the pathogens causing the ulcer, tissue biopsies or swabs are taken from the ulcer site.[44]
- **Imaging -** To determine the degree of infection and rule out osteomyelitis, use X-rays, MRIs, or CT scans.[45]
- **Vascular Assessment -** To assess blood flow to the feet, Doppler studies or angiography is used.[45]

➢ *Treatment[46]*

- **Wound Care -** To encourage healing, use suitable dressings, debridement (removing dead tissue), and routine cleaning.[47]
- **Infection Management -** Customized antibiotic treatment based on the pathogens found and the intensity of the infection.
- **Offloading -** Relieving pressure on the ulcerated area by using casts, orthotics, or special footwear.[48]

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- **Blood Sugar Control -** Strict glycemic management to speed up the healing of wounds and immune system performance.
- **Surgical Intervention -** To stop the infection from spreading in severe cases, surgical debridement or amputation may be required.[49]

➢ *Prevention[50]*

- **Foot Care Education -** Instructing the patients how to take care of their feet on a daily basis, wear appropriate footwear, and treat minor injuries quickly.[50]
- **Regular Check-ups -** Frequent trips to medical professionals for diabetes management and foot exams.
- **Control of Risk Factors -** To reduce the chance of complications; maintain ideal blood pressure, cholesterol, and blood sugar levels.

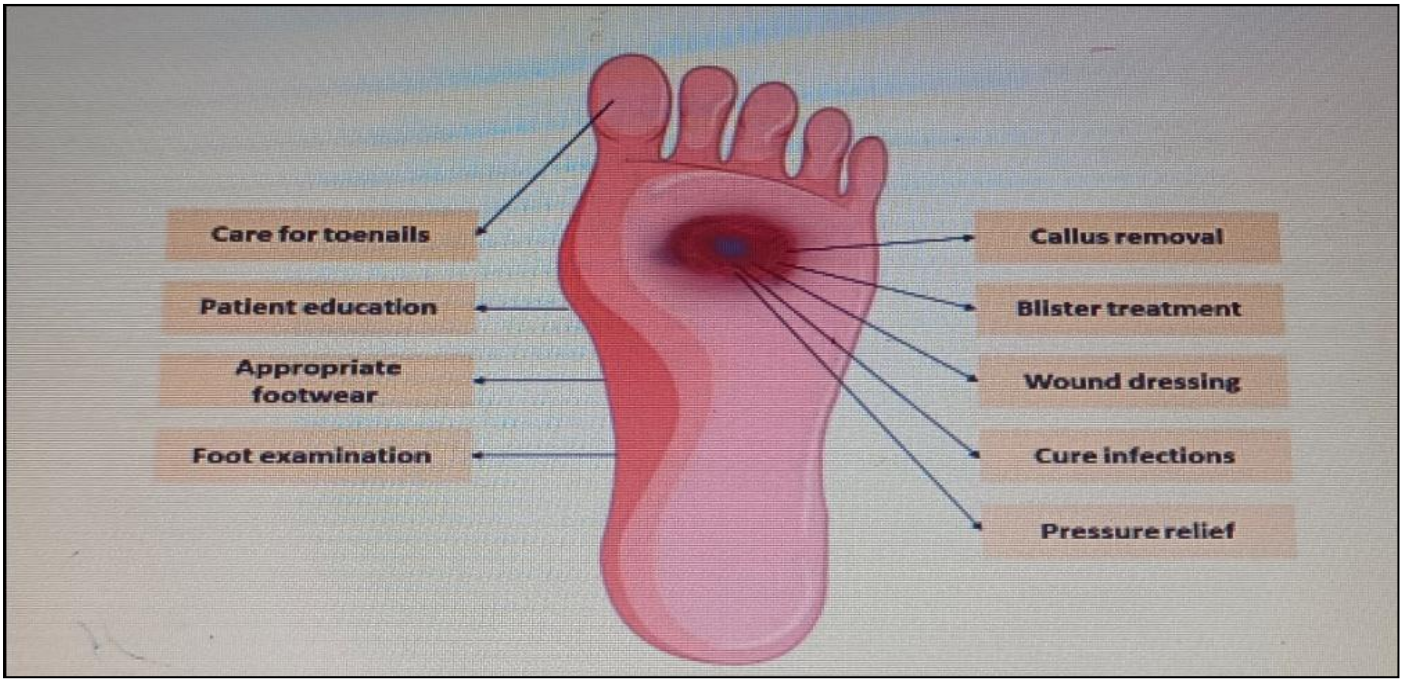

Fig 7 Prevention of Diabetic Foot Infection

IV. CONCLUSION

Diabetes-related foot infections and ulcers are dangerous side effects that need to be treated promptly and thoroughly in order to avoid serious consequences, such as amputation.[51] A multidisciplinary strategy is required for successful treatment, which includes managing wounds, preventing infections, and treating underlying conditions like elevated blood sugar and poor circulation.[52] The incidence and severity of complications related to diabetic feet can be considerably decreased with appropriate education and preventive measures.[53]

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