

"Progressive Necrotizing Diseases; Management: Titrated Dosage Anti-Histaminics Role."

Dr. (Prof.) Anil K. Sahni M.S, F.I.C.S, Advanced D.H.A
Professor Surgery, NDMC Med. Coll. & Hindu Rao Hospital Delhi-7
A-1/ F-1 Block – A Dilshad Garden Delhi-110095 India Mobile-09873083100

ABSTRACT: Introduction- Amongst Various Skin & Soft Tissue Infection (SSTI), Progressive Necrotizing Disease (PND): Infections, Cellulitis, Fasciitis (NF) & Sometimes Myositis, Is Not Uncommon Clinical Entity, Characterized By Extensive, Rapidly Progressive Soft-Tissue Necrosis That Usually Involves The Subcutaneous Tissue And Muscular Fascia, But Can Also Affect The Skin And Muscle, Has Vivid Spectrum Of Epidemiological, Aetio-Pathogenesis, Clinical Manifestations Of Varying Severity & Differing Treatment Seeking Time Period. NF Poses A Serious Surgical Challenge Not Only Because Of Its Rapid And Progressive Nature, But Also Because Of Its Attributing High Morbidity And Mortality.

Materials & Method- Present Multi-Centric Study, Comprising About 500 Patients Presenting With Variable Stages Of Disease, Performed At, Plains Of North India Haryana, Adjoining Rajasthan & Hilly Region Of Uttarakhand, During Last More Than A Decade Duration.

Management Methodology- Management Of Pre-Existing & Or Precipitating Co-Morbidities - Diabetes, HTN, Deranged Renal Function, C-Reactive Proteins, HB%, Serum Proteins, Electrolytes, Minerals, Blood Gas Chemistry Alterations, Hepatic Dysfunction, Different Stages Of Progressive Toxicemia, Septicemia Due To Ensuing 'PF Shock Syndrome', Time Of Seeking Medical Attention & Standard Of Appropriate Care, Are Important Determinants For Overall Result Out Come, In Regards To Mortality & Morbidity Aspects.

Results- Early (Immediately/Within 2-4 Days Of Disease Onset), Medical Opinion Seeking Patients, Managed With, Described Medical Therapy Including Anti-Allergic Drug, For 3-5-7 Days, With Careful Monitoring & As Needed Immediate Follow Up Advise, Were Completely Relieved, In About 75% Of The Cases, (i.e Disease Process Cessation Control, In Clinical Stage -1(Early) & Upto Initial Clinical Stage- 2, In Early Reporting Category Patients).

Remaining About 25% Patients, Not Completely Controlled By Described Treatment Protocol & Late Presentations Of 1-2 Weeks Disease Onset With Progressive Disease Of Varying Manifestations, Responded To Management-Medical, Surgical, Supportive & Could Be Discharged Within 1-2 Weeks Of Hospital Stay.

About 5-10% Patients-Late Presentations Of Severe Progressive Disease, Without Previous Treatment & Or Not Proper Inadequate Management, In Different Stages Of Septicemia, Deteriorating G.C, Co-Existing Controlled/ Uncontrolled Co-Morbidities, Were Advised Referral To Higher Specialized Centres For Immunotherapy, Hyper-Baric Oxygen Therapy Etc. After Preliminary Management & Supportive Measures With Ensured Safe, Secure Transport, Otherwise, Continued Management At The Same Centre, With Prognosis Explained Treatment Risk Consent, Recorded Increased Morbidity And About 1-2% Mortality.

Conclusion- In Clinical Stage -1(Early) & Upto Initial Clinical Stage-2(Intermediate), Reporting Early To Clinician Category Patients, (i.e Clinically Evident Acute Inflammatory Response Phase – Usually, Initial 3-5-7 Days After Disease Process Onset, In All Types), Inclusion Of Meticulously Titrated Dosage Duration Schedule Of Anti-Allergic Medication Therapy, In Administered Medications, With Properly Instituted Supportive Measures, Controlled Necrotizing Soft-Tissue Disease Process Severity, Conversion To Next Clinico-Pathological Strata Of Disease (Infections-Cellulitis-Fasciitis-Myositis & Others.), Associated Complications, Sequelae Of The Disease Process, Safely, With Better Result Out Comes Of Subsequent Management & Decreased Morbidity, Mortality.

Key Words:

1. Progressive Necrotizing Disease (PND): Infections, Cellulitis, Fasciitis (PF), Myositis
2. Aetio-Pathogenesis; Allergic Origin- Insect Bite/Crawl, Exposure To Herbs, Chemicals
3. Co-Morbidities Control- Relevant Needed Investigations
4. Management Modality-Anti-Histaminics In Titrated Dosage With Other Medications
5. Surgical Procedures-Fasciotomies, Debridement & Subsequent Wound Healing By Secondary Intention
6. Comparative Better Result Outcome: Different Stages Of Disease

Date of Submission: 15-11-2017

Date of acceptance: 30-11-2017

I. INTRODUCTION

Comparatively Not Uncommon Variety Of 'Skin & Soft Tissue Infections', Progressive Necrotizing (PND): Infections, Cellulitis, Fasciitis & Sometimes Myositis, Have Vivid Spectrum Of Epidemiological, Aetio-Pathogenesis, Clinical Manifestations Of Variable Severity, Manifestations Onset & Treatment Seeking Time Period.

Available Awareness For Proper Intensive Medical / Supportive Therapy, Needed Timely Surgical Intervention, Subsequent Supervised Healing, With Pre-Existing & Or Resultant Co-Morbidities Control, Supported By Relevant Needed Investigatory Support, Especially For Intensive Care Circumstances, Determine The Over All Variable Morbidity & Mortality Of Disease.

II. NECROTIZING SOFT-TISSUE INFECTIONS, AN OVERVIEW.

Necrotizing Soft-Tissue Infections, Comprise A Spectrum Of Disease Entities That Are Characterized By Extensive, Rapidly Progressive Soft-Tissue Necrosis That Usually Involves The Muscular Fascia And Subcutaneous Tissue, But Can Also Affect The Skin And Muscle.

Necrotizing Soft-Tissue Disease, Can Be Categorized As- Infections, Cellulitis, Fasciitis, Or Myositis, Based On The Principal Soft-Tissue Layer Involved With Necrosis.

These Infections Can Have Either An Indolent Or Fulminant Presentation, Or Unpredictable Clinical Course.

The Disease Characterized By Extensive Necrosis, Primarily Involving The Superficial Fascia & Deep Fascia Onwards Subsequently, With Concomitant Thrombosis Of The Cutaneous Microcirculation, Perhaps The Most Severe Form Of Soft Tissue Infection, PND Has Bewildered Physicians For Centuries, Described Various As Haemolytic Gangrene, Acute Streptococcal Gangrene, Gangrenous Erysipelas, Necrotising Erysipelas, Suppurative Fasciitis, And Hospital Gangrene, Among Other Names.

Hippocrates (Fifth Century BC) Gave The First Description Of This Dreaded Disease.

The First Report Of This Disease Was By A Confederate Army Surgeon,

Joseph Jones (1871) U.S.A. Named This Entity '**Hospital Gangrene**'.

Meleney (1924) Beijing, Reported An Outbreak Of Hospital Gangrene And

Coined The Term **Hemolytic Streptococcal Gangrene**.

Wilson (1952), First Introduced The Term **Necrotizing Fasciitis**, & Is The Preferred Term Today Describing The Most Consistent And Key Feature Of This Disease, Rapidly Progressive Inflammation And Necrosis Of Subcutaneous Tissues And The Deep Layer Of Superficial Fascia With Sparing Of The Deep Fascia And Muscle.

However, The Term Necrotising Fasciitis Is Now Used In A Generic Sense To Include All Diffuse Necrotising Soft Tissue Infections Except Gas Gangrene (Clostridial Myonecrosis).

Diffuse Necrotising Soft Tissue Infections Include Classic Gas Gangrene, Meleney's Haemolytic Streptococcal Gangrene, Necrotising Fasciitis As Described By Wilson, And The **Gram-Negative Synergistic Necrotising Cellulitis Of Stone**.

Generally, **One Condition Cannot Be Distinguished From Another At The Time Of Diagnosis**.

The Oro-Facial Form Of NF Is Called **Cancrum Oris (NOMA)**, A Rapidly Progressive, Polymicrobial, Often Gangrenous Infection Of The **Mouth Or Genitals**.

The Mucous Membranes Of The Mouth Develop Ulcers, And Rapid, Painless Tissue Degeneration Ensues, Which Can Degrade Tissues Of The Bones In The Face.

Noma Pudendi - Noma Can Also Cause Tissue Damage To The Genitals. The Perineal Form Is Called **Fournier's Gangrene**. **Idiopathic Scrotal Gangrene**, However, Is Different In Aetiology, Extent, And Clinical Presentation From Fournier's Gangrene.

NF Poses A Serious Surgical Challenge Not Only Because Of Its Rapid And Progressive Nature, But Also Because Of Its Attending High Morbidity And Mortality.

MICROBIOLOGY- Classification Of NSTIs Based On Microbiology Is As Follows:

Type I – Poly-Microbial Etiology (Most Common)

Type II – Mono-Microbial Etiology (Streptococcus, Staphylococcus, Clostridium); Staphylococcal NSTI Is Increasing In Prevalence Owing To More Cases Of MRSA Infection And Association With Intravenous Drug Use, Particularly The Use Of Black Tar Heroin.

Type III - Vulnificus (Associated With Sea-water Exposure, In Individuals With Underlying Cirrhosis).

CAUSATIVE ORGANISMS-

Mono-Microbial Pathogens: Streptococci (Especially Strepto-Coccus Pyogenes – Group A)

Clostridium Perfringens (Gas Gangrene) And Other

Clostridial Species Staph Aureus Vibrio Vulnicans Aer Onomonas Hydrophila

Polymicrobial Pathogens: Mixed Aerobe-Anaerobe Bacterial Flora (E.Coli, Bacterioides Fragillis , Strep And Staph) Fourniers Gangrene Is Typically Polymicrobial Infection With Aerobes And Anaerobes, Such As Coliforms, Klebsiella, Streptococci, Staphylococci, Clostridia, Bacteroids, And Corynebacteria
Mixed Aerobe: Anaerobe Bacterial Flora (E.Coli , Bacterioides Fragillis)

PATHOPHYSIOLOGY- The Primary Site Of Pathology Is In The Superficial Fascia.

Bacteria Proliferate Within The Superficial Fascia And **Elaborate Enzymes And Toxins** Enabling The Organisms To Spread Through The Fascia. The Precise Mechanism Of Spread Has Not Been Fully Elucidated But Some Investigators Have Attributed It To Expression Of Bacterial Enzymes Such As **Hyaluronidase**, Which Degrades The Fascia.

Resultant Key Pathological Process, From This Uncontrolled Proliferation Of Bacteria Is **Angio-Thrombotic Microbial Invasion And Liquefactive Necrosis** Of The Superficial Fascia. Histologically, Necrosis Of The Superficial Fascia, Polymorphonuclear Leukocyte Infiltration Of The Deep Dermis And Fascia, Thrombosis And Suppuration Of The Veins And Arteries Coursing Through The Fascia, And Microorganism Proliferation Within The Destroyed Fascia Are Seen. As This Process Progresses, Occlusion Of Perforating Nutrient Vessels To The Skin Causes Progressive Skin Ischemia.

Organisms Spread From The Subcutaneous Tissue Along The Superficial & Deep Fascial Planes, Presumably Facilitated By Bacterial Enzymes And Toxins. This Deep Infection Causes Vascular Occlusion, Ischemia, And Tissue Necrosis. Superficial Nerves Are Damaged, Producing The Characteristic Localized Anesthesia. Septicemia Ensues With Systemic Toxicity.

The Underlying Event That Is Responsible For The Cutaneous Manifestations Of NF - As The Disease Evolves. Initially A Horizontal Phase Predominates With Rapid Spread Through The Fascia & Extensive Undermining Of The Apparently Normal Looking Skin. As The Condition Progresses, Ischemic Necrosis Of The Skin Ensues With Gangrene Of The Subcutaneous Fat, Dermis And Epidermis, Manifesting Progressively As Bullae Formation, Ulceration And Skin Necrosis.

"Flesh-Eating Bacteria."-Important Bacterial Factors Include-

Surface Protein Expression And Toxin Production.

M-1 And M-3 Surface Proteins, Which Increase The Adherence Of The Streptococci To The Tissues, Also Protect The Bacteria Against Phagocytosis By Neutrophils.

Directly Toxic Streptococcal Pyrogenic Exotoxins (Spe) A, B, And C Tend To Be Produced By Strains Causing Necrotizing Fasciitis.

These **Pyrogenic Exotoxins, Together With Streptococcal Super-antigen (SSA)**, Lead To The Release Of Cytokines And Produce **Clinical Signs Such As Hypotension.**

The Etiological Agent May Also Be A *Staphylococcus Aureus* Isolately Harboring The Enterotoxin Gene Cluster SEG, SEI, SEM, SEN & SEO, But Lacking All Common Toxin Genes, Including Pantan-Valentine Leukocidin.

The Poor Prognosis Associated With Necrotizing Fasciitis Has Been Linked To Infection With Certain Streptococcal Strains. Community-Acquired Methicillin-Resistant *S Aureus* (MRSA) Has Also Been Associated With Necrotizing Fasciitis.

Single-Nucleotide Changes Are The Most Common Cause Of Natural Genetic Variation Among Members Of The Same Species.

INVESTIGATIONS-

Laboratory- Leukocytosis With Neutrophilia, Acidosis, Altered Coagulation Profile, Impaired Renal Function, Raised Creatinine Kinase Levels, And Raised Inflammatory Markers, Such As C-Reactive Protein Levels, Coagulation Profile (DIC) Etc.

Plain X-Ray Films Can Demonstrate **Subcutaneous Gas**, But This Is A Specific Not A Sensitive Finding (Positive In Fewer Than 25% Of Cases) And Absence Of Gas Does Not Exclude NF.

Computed Tomography And Magnetic Resonance Imaging (MRI) Might Be Useful In Cases Where Signs Are **Equivocal Or Diagnosis Is In Doubt.**

Asymmetrical Fascial Thickening, Fat Stranding, And Gas Tracking Along Fascial Planes Are Important Imaging Findings.

C. T Scans Are Estimated To Have A **Sensitivity Of 80%** For Detecting Necrotizing Soft Tissue Infections.

MRI- In Cases Of Cellulitis, Will Demonstrate **Subcutaneous Thickening With Fluid Collection.** However, When There Is **Deep Fascia Involvement With Fluid Collection**, Thickening, And Enhancement After Contrast Administration, Necrotizing Infections Must Be Considered.

The Reported **Disputed Sensitivity Of MRI -100% With A Specificity Of 86%**, While Other Argue That Only Early Cases Of NF, MRI Might Not Show Fascial Involvement.

If **Clinical Suspicion Is High**, Surgeons Can Opt To **Explore & Perform Tissue Biopsies (Frozen Section Biopsies)** Rather Than Delay Treatment For Imaging Studies.

Additional Tests Include **Needle Aspiration & Incision Biopsy** From The **Effected Lesions & Or Draining Lymph Nodes**. Negative Results, However, Cannot Exclude NF. **Surgical Exploration** Is Preferable. **Macroscopic Findings** During Surgical Exploration Include **Gray Necrotic Tissue, Lack Of Bleeding, Thrombosed Vessels, "Dishwater" Pus, Noncontracting Muscle, And A Positive "Finger Test" Result**, Which Is Characterized By Lack Of Resistance To Finger Dissection In Normally Adherent Tissues.

Various Other Modality And Techniques Have Been Proposed To Aid In The Early Diagnosis & Management Of Necrotizing Fasciitis.

THE LRINEC (Laboratory Risk Indicator For Necrotizing Fasciitis) SCORE: A Tool For **Distinguishing Necrotizing Fasciitis From Other Soft Tissue Infections, Demographic And Clinical Variables And Outcome Of Patients** Used In The Developmental Cohort-Mean Age Gender Male Female Co-Morbidities, Diabetes Mellitus, Peripheral Vascular Disease, No Comorbidities Variables, Hypotension, Multiple-Organ Failure At Admission.

Laboratory Risk Indicator For Necrotizing Fasciitis (Lrinec) Score-

C-Reactive Protein, Total White Cell Count, Hemoglobin, Sodium, Creatinine, Glucose,

The LRINEC Score Is Capable Of Detecting Early Cases Of Necrotizing Fasciitis Among Patients With Severe Soft Tissue Infections. **A Lrinec Score Of 6** Should Raise The Suspicion Of Necrotizing Fasciitis, And **A Score Of 8** Is Strongly Predictive Of This Disease.

RISK FACTORS For Necrotizing Fasciitis- Diabetes, Chronic Disease, Immunosuppressive Drugs (e.g. Prednisolone), Malnutrition, Age > 60 Years, Intravenous Drug Misuse, Peripheral Vascular Disease, Renal Failure, Underlying Malignancy, Obesity, Odontogenic Infection, Malignancy, Chicken Pox, Local Penetrating Trauma Or Animal Bite Recent Surgery (e.g. Abdominal Or Peritoneal Surgery)

However, Group A Strep -Has Particularly Anity For Young Healthy Patients ?

No Previous Exposure Makes It Highly Virulent.

Non-Steroidal Anti-Inflammatory Drug Use Has Been Implicated In Severe Necrotizing Streptococcal Infections. It Is Postulated That Nonsteroidal Anti-Inflammatory Drugs Impair Lymphocyte Function & Suppression Of Symptoms And Signs Of Inflammation Leads To Later Diagnosis, Especially In Patients Presenting Early With Nonspecific Symptoms.

Risk Factors For NF In The Pediatric Population- Include Malnutrition And Skin Infections e.g. Varicella.

DIFFERENTIAL DIAGNOSIS- Cellulitis, Phlegmasia Cerulea Dolens, Compartment Syndrome

COMPLICATIONS- Common Complications That May Be Encountered Include:

1. Compartment Syndrome, Leading To Volkmann's Ischaemia, Volkmann's Ischaemic Contracture, Or Gangrene;
2. Septic Arthritis Or Osteomyelitis;
3. Septicaemia & Multiple Organ Failure Syndrome
4. Herniation Of Intraabdominal Organs;
5. Joint Stiffness;
6. Contractures And Trismus

PROGNOSIS- Considerable Mortality Due To OverAll NF, Is Comparatively Higher In Patients With **Streptococcal Toxic Shock Syndrome**.

Diabetic Patients, Especially Those With Diabetic Ketoacidosis Or Hyperosmolar Hyperglycemic Nonketotic Acidosis Have **Increased Morbidity & Higher Mortality**.

Delay In Needed Surgical Intervention, For >24 Hours Is An **Independent Risk Factor For Mortality**.

Extensive Debridement Resulting In Muscle Loss, Sometimes Resultant Considerable Postoperative Morbidity, As A Period Of Rehabilitation To Regain Function Of The Affected Areas. **Scarring And Disfigurement** Can Also Be Substantial.

PROPHYLAXIS- Potentially Lethal Disease, NF, Needs **Complete Awareness Regarding** Chemoprophylaxis, Medical Therapy, Need For Surgical Interventions, Subsequent Physiotherapy, Strategy For Impending Shock Management, To The **Primary Care Health Providers**.

Education To Household Contacts, For Disease Control & Prevention Recommendations For **The Community** With Invasive Group A Streptococcal Disease & Other Causative Factors, Advising Them To Seek Medical Attention Immediately on Symptoms Developments.

PREVENTION- Necrotising Fasciitis Is Largely A **Preventable Disease**, But Prevention Will Involve A Multidisciplinary Commitment And Action By Individuals, Health Personnel, And Policy Makers.

Preventive Measures Involve: Good Oral And General Body Hygiene;

- Prevention And Control Of Malnutrition;
- Prevention Of All Childhood Immunisable Diseases, Such As Measles, Through National Mass Immunisation Programmes; And
- Education On Early Recognition And Treatment Of NF.

III. MATERIALS & METHOD

Discussed Multi-Centric Study, Comprising About 500 Patients Presenting With Variable Stages Of Disease, Performed At, Plains Of Northern India Haryana, Adjoining Rajasthan & Hilly Region Of Uttarakhand, During Last More Than A Decade Duration.

CLINICAL ASSESSMENT, Primarily Based Upon-

CLINICAL HISTORY- Age (Paediatric, Adult, Geriatric Group), **Sex** (Both Male & Female), **Nutritional Status**-Usually Satisfactory, Sometimes Malnourished, **Occupation**- Commonly Agricultural Workers With Increased Susceptibility For **Exposure To Chemicals- Fertilizers, Insecticides & Agricultural Produce** Especially - ***'Gyar'***, **Co-Existent Herbs & Weeds** Etc.(Utilized For Guar Gum & Other Uses.),Exposure. To **Unknown Toxic Herbs, Weeds**-Commonly Known As **'Vishkanta'**- Poisonous Thorn Injury, **Insect Bite / Sting / Crawl** On Affected Extremity. History Suggestive Of – **Direct/ Indirect Trauma, Therapeutic Injections, IV Canulations, Drug Abuse, Surgical Interventions, Procedures, Catheterizations, Surgeries Of Different Anatomical Regions, Parietal & Or Visceral Infections**, Various Body Organ System- **Exogenous/Endogenous Infections**.

**The Guar Or Cluster Bean, Also Known As Gavar, Guwar, Or Guvar Bean, A Legume.*

*Botanical Name- Cyamopsis Tetragonoloba, Is An Annual Legume And The Source Of Guar Gum. The African Species Cyamopsis Senegalensis. Further Domesticated In India And Pakistan, For Many Centuries(About 80% Of World Production), Grows Well In Semiarid Areas, But Frequent Rainfall Is Necessary.Very Valuable Plant Within A Crop Rotation Cycle, As It Lives In Symbiosis With Nitrogen-Fixing Bacteria. A Source To Replenish The Soil With Essential Fertilizers And Nitrogen Fixation, Before The Next Crop With Multitude Of Different Functions For Human And Animal Nutrition But Its Gelling-Agent-Containing Seeds (Guar Gum) Are Today The Most Important Use. Demand Is Rising Rapidly Due To Industrial Use Of Guar Gum In Hydraulic Fracturing (Oil Shale Gas), Beside Various Other Uses. Guar Gum Extraction Technology Commercialization-USA(1953) & A Decade After In India. Alternative - Alfne Gums.**

Depending Upon The Total Duration Of Illness At The Time Of Seeking Medical Attention, Varying From – Days To Week,

CLINICAL SYMPTOMS-

Of **Spreading Swelling Erythema**, Severe Constant **Pain** Out Of Proportion To Clinically Evident **Local Inflammation & Infection Of Involved Limb** With Erythematous, Tender, Smooth, Shiny & Tensely Swollen Skin, Colour Changes, Dark Patches, Blisters & Bullae, Subsequent Pre-Gangrenous Changes, Wooden Hard Consistency Of The Sub Cutaneous Oedema, Hypo-Aesthesia,

Features Of Toxicæmia, Septicæmia- Sepsis Fever, Chills, Myalgias, **Haemodynamic Unstability, Developing Organ Failure** Evidence.

Pre-Existing Medical Illnesses – Diabetes, Hyper-Tension, Tuberculosis Etc.

SITE-

Most Common Affections Are- Distal Parts Of Superior & Inferior Extremities-

(Hands, Fore-Arms, Feet, Lower Legs), With Upward Progression,

However, Occasional Involvement Of Face, Nape Of The Neck, Chest, Breast, Abdominal Wall, Back, External Genitalia, Perineum Etc., Is Not Very Uncommon.

CLINICAL STAGES-

Clinical Features Of Necrotizing Fasciitis As The Disease Progress,

Stage 1 (Early)- Tenderness To Palpation (Extending Beyond The Apparent Area Of Skin Involvement) Erythema Swelling Warm To Palpation.

Stage 2 (Intermediate)- Blister Or Bullae Formation (Serous fluid), Skin fluctuance, Skin Induration

Stage 3 (Late)- Hemorrhagic Bullae Skin Anesthesia Crepitus Skin Necrosis With Dusky Discoloration Progressing To Frank Gangrene.

MANAGEMENT-

Depending Upon **Progressive Necrotizing Disease- Infectious Stage**

In Consideration Of Overall General Condition, Nutritional Status, Vital Parametres,

Following **"Treatment Protocol"** Is Initiated:

(A.) MEDICATIONS

1. Appropriate Broad Spectrum Anti-Biotics;
Essentially Combined With An-aerob Antimicrobial, In Adequate Dosage.
2. Anti-Inflammatory, Analgesic, Anti-Pyretic
With Trypsin-Chymotrypsin/ Serratio-Peptidase Preparations,
In Adequate Dosage Duration Schedule For Round The Clock Coverage.
3. Medications For Proper Ant-Acid Cover
4. B-Complex, Anti-Oxidants & Other Nutrient Supplements
&

ANTI-HISTAMINIC- Cetrizine- Adults—5 To 10 Milligrams (Mg) OD/

Levo-Cetrizine / Chlorpheniramine - 4 Milligrams (Mg) TDS-QID As Needed Has Been Commonly Used.

Other Frequently Available Anti-Allergic Medications Are- Fexofenadine-Adults And Teenagers—60 Mg BD As Needed Or 180 Mg OD. / **Loratadine-**Adults And Children 6 Years Of Age And Older—Mg OD / **Clemastine-**Adults And Teenagers—1.34Mg BD Or 2.68 Mg OD-TDS, As Needed.

(B.) SUPPORTIVE MEASURES-In View Of **Non Availability Of Anti-Allergic Ointment**

Soframycin Oint. Local Application, Betadine Soln Sterile Dressing, Hygeine Maintainence.

Advice For Covering The Exposed Effected & Other Extremities, Is Of Utmost Importance.

Tetanus Immunization, If Need.

The Above Mentioned, "Treatment Protocol" Had Been Able To Resolve, The **Early Reported**

(Within 1-2 Days Of Onset), **PND-Inflammatory Phase Patients**, Within 5-7-10 Days, **In About 75% Cases.**

The Remainder (About 75%) – Partial / No-Responder Cases, Presenting With Persistent Swelling & Other Features Suggestive Of Disease Progression To 'Cellulitis', 'Fascitis', **& Or First Time Presenters**, Were **Advised Admission-**

1. All Medications Of The Above Mentioned 'Tt. Protocol', Started **Using Parentral Route.**

2. Resuscitation- Correction Of Depletion- Importantly Correction Of Any Existing **Physiological Derangements, Such As Fluid And Electrolyte Imbalance.**

3. Nutritional Support Is Required From The First Day Of The Patient's Admission To Hospital (Preferably In Intesive Care), To Replace Lost Proteins And Fluid From Large Wounds And/OR The Resultant Toxic Shock. **Metabolic Demands** Are Similar To Those Of Other Major Trauma Or Burns, Which Means That The Patient **Needs Twice The Basic Caloric Requirements.**

Blood Transfusion, May Be Multiple B.T, Blood Substitute, Plasma Expanders, Necessary To Correct Anaemia, Improve Nutritional Status & Hence 'Wound Healing'.

Until **Aspirated Fluid/Blood Culture** Results Are Available,**Wide Spectrum Coverage With Intravenous Antibiotics (With An Awareness Of Resistance In The Patient Population Being Treated)** Is Started.

These Antibiotics Cover *S Pyogenes*, *S Aureus* (Including Community-Acquired MRSA If Indicated, According To Local Resistance Patterns), And **Gram-Negative Aerobes And Anaerobes** As Clinically Indicated. In Particular, **Gram-Negative Organisms Would Be Suspected In-**Perineal And Abdominal Wall Wounds, Necrotic Diabetic Foot Ulcers,

And In Heavily Contaminated Wounds Associated With Devitalizing Major Trauma.

(C.)SURGICAL PROCEDURES-

Depending Upon The Clinical Stage Of The Disease,Varying From Site & Severity Of Cellulitis, Fascitis, Accompanying Features Of Toxicaemia- Septicaemia & Pre-Existing Medical Illnesses e.g Diabetes & Others, **Following Surgical Procedures Are Planned-**

FASCIOTOMIES- Usually In **Emergency O.T-** Based Upon The **Principle Of Abdominal Compartment Syndrome** Management, In The Involved Part Of Effected Extremity (Usually Lower Legs, Hands, Fore-Arm, Feet), **2-3 Release Incisions** Are Given, Along Underlying Anatomical Structures, (**Anter-Lateral, Antero-Medial, Posterior-** In Lower Legs, Fore Arm, Feet, Dorsum / Palmar **Anatomical Creases-** In Hands, **Skin, Sub-Cutaneous Tissue Essentially Upto Fascia Deep**(Confirmed By Pouting Muscles), Sometimes Depending Upon Underlying Muscles Oedema Tension Status- Release By Splitting Muscles At 2-3 Places Is Helpful.

Oozing Oedema Fluid Is Sent For- Culture & Sensitivity, Cytology, Bio-Chemistry, Simultaneously **Some Debriebed Tissue** Can Be Sent For **Histo-Pathology Confirmation.**

Milking Of Oedema Fluid Can Be Beneficial For Faster Results.

Needed Haemostasis Followed By **Wound Wash** With H₂O₂, Normal Saline & Betadine, Followed By 2% Xylocaine + Anti-Septic / Antibiotic Ointment Sterile Dressing, With Adequate Gauze For Soakage.

Usually Very Little Or Upto No **LA** Is Needed, Confirming Hypo-Anaesthesia Caused By Disease.

Appropriate Resuscitative Fluid Supplementation, Is Recommended, In View Of Considerable Loss By Fasciotomies Soakage Wound (Upto 1-2 Litres In 24 Hours, Depending Upon Surface Area, Disease Extent, & U/L Or B/L Involvement).

Management Of Pre-Existing & Or Precipitated Co-Morbidities- Diabetes, HTN, Deranged Renal Function, C-Reactive Proteins, HB%, Serum Proteins, Electrolytes, Minerals, Blood Gas Chemistry Monitoring, During Different Stages Of Ensuing Toxicemia, Septicemia Etc.,

Are The **Important Determinants For Overall Result Out Come**.

With Proper Regular Sterile Dressings, ? Supersaturated Hydrogen-Per-Oxide Use Needed Change For Antibiotic Therapy Depending Upon C & S Report,

Appropriate Nutritional Supplementation Support(Oral +_ Parenteral),

Needed Blood Transfusion/s,

Fasciotomy Wound Usually Start Healing With Secondary Intention By Healthy Granulation, With/Without Needed **Meticulous Cleaning & Debridement Of Wound**, In About 2-3 Weeks Time.

The Pain Of Local Infection May Cause The **Voluntarily Immobilisation**, In Affected Areas Of The Body.

Discretely Supervised, Timely, Appropriate Splintage & Proper Physiotherapy (Both Passive And Active Movements), Play A Very Important Role In Prevention Of Joint Stiffness, Scar Contracture,

Deformity Development & Other Disfigurements,

By Achieving The Goal To Attain A Position That Opposes The Forces Of Contracture, Provide Safe Joint Alignment, And Maintain Tendon Balance Without Causing Stretch Or Pressure Injuries To The Peripheral Nerves Or Skin.

The Complete Healing Duration However, Can Be **Shortened By**, 'Distantly Placed **Approximation Sutures**', Subsequent Skin Grafting, Plastic Reconstructive Repairs.

(VAC) Vacuum-Assisted Closure Device Use- Lately, Many Surgeons Worldwide Have Started Using VAC Therapy For Fast And Effective Wound Closure. A Vac Device Consists Of A **Sterile, Open-Cell Foam Sponge Placed In The Wound**, The Size Of Which Is Adjusted To The **Wound Size**.

This Is **Covered By Transparent Adhesive Drape** To Create An **Airtight Environment**.

The **Sponge Is Connected To A Portable Vacuum Pump** By Means Of **Non-Collapsible Tubing**.

Evacuation Is Applied To The Sponge Using The Pump, Which Provides **Continuous Negative Pressure**.

The Vac Device **Improves Wound Healing By Providing Microstrain &**

Preventing Infection D/T Seroma Formation

(HBO)Hyperbaric Oxygen Therapy - Role In NSTI Is **Controversial**, As **No Proven Benefit** For The Patient And **Can Delay Resuscitation And Surgical Debridement**.

HBO Involves The Use Of Oxygen At 2-3 Times Atmospheric Pressure With

Proposed Benefits Of **Bacteriocidal Effects, Improved Polymorphonuclear Lymphocyte Function,** & **Enhanced Wound Healing**.

HBO Should Be Considered As A **Treatment Adjunct & Not Replacement**

For Surgical Debridement & Medical Therapy.

(IVIG) Intravenous Immunoglobulin Therapy- Postulated To **Bind Exotoxin Produced By Streptococcal And Staphylococcal Species, Potentially Delaying The Onset Of The Systemic Inflammatory Response And Sepsis**. The Use Of IVIG Has **Not Been Definitively Established**, But Can Be Considered For

Hemodynamic Unstable, Critically Ill Patients, Clostridial Myonecrosis(Gas Gangrene), Streptococci Resulting In

Streptococcal Toxic Shock Syndrome,

Intravenous Immunoglobulins Might Play A Therapeutic Role.

CONSULTATIONS AND TRANSFER- Without Delay For Of Laboratory Or Radiology Results. Surgical Subspecialty Consultation - Urology In Cases Of Fournier Gangrene, Consultation With An Infectious Diseases Specialist & If Facility For Handling The Aggressive Care, Monitoring, And Serial Surgical Debridement Not Available, Arrangements For Transfer Are Made.

However, Patients Should Not Be Considered For Transfer Until They Remain Hemodynamically Stable.

IV. RESULTS

Patients Seeking Medical Opinion, **Immediately /Within 2-4 Days Of Disease Onset, By Described Medical Therapy Including Anti-Allergic Drug, For 5-7-10 Days, With Careful Monitoring & As Needed Immediate Follow Up Advise, Were Completely Relieved, In About 75% Of The Cases, (Disease Process Cessation Control, In Clinical Stage -1(Early) & Upto Initial Clinical Stage-2(Intermediate), In Early Reporting Category Patients).**

Remaining About 25% Patients, Not Completely Controlled By Described

Treatment Protocol Including Conservative Measures &

Late Presentations Of 1-2 Weeks Disease Onset With Progressive Disease, Manifesting As Localized Infection, Inflammation, Oedema, Fascio-Cutaneous Changes, With/Without Generalized Toxicemia /Septicemia, Deranged Body Bio-Chemistry Features Were,

Admitted & Managed With Parenteral Medications, Appropriate Fluid Therapy, With Electrolytes Correction, Oral / Parenteral Nutritional Support &

Needed Surgical Interventions (Fasciotomies, Wound Debridement Etc),

Responded To Treatment & Could Be Discharged Within 1-2 Weeks Of Hospital Stay, With Explained Awareness Regarding Medications, Nutrition (Protein, Mineral Rich Foods Supplements), Proper Sterile Dressings, Physiotherapy Exercises &

Preparation For Needed Skin Grafting, Plastic Reconstructive Repairs, As Per Patients' Compliance.

About 5-10% Patients, Late Presentations Of Severe Progressive Disease,

Without Previous Treatment Or Not Proper Inadequate Management,

In Different Stages Of Septicemia, Deteriorating/ Clinical, Laboratory, Radiodiagnostic Monitors,

Confirmed High Virulence Resistant Pathogens, **Co-Existing Controlled/ Uncontrolled Co-Morbidities** e.g Diabetes, Hypertension, COPD, Hepatico-Renal Dysfunction, HbsAg, HCV, HIV For Aids Etc.,

Were Advised Referral To Higher Specialized Centres For Immunotherapy, Hyper-Baric Oxygen Therapy, Better Critical Care Facilities Etc. After Preliminary Management &

Supportive Measures Ensured Safe, Secure Transport,

Otherwise, Continued Management At The Same Centre,

With Prognosis Explained Treatment Risk Consent,

Recorded Increased Morbidity And About 1-2% Mortality.

V. CONCLUSION

In Clinical Stage -1(Early) & Upto Initial Clinical Stage-2(Intermediate),

Reporting Early To Clinician Category Patients,

(i.e **Clinically Evident Acute Inflammatory Response Phase –**

Usually, Initial 3-5-7 Days After Disease Process Onset, In All Types),

Addition Of Meticulously Titrated Dosage Duration Schedule Of Anti-Allergic Medication Therapy,

Along With The Medical Therapy, Properly Instituted Supportive Measures,

Controlled Necrotizing Soft-Tissue Disease Process Severity,

Conversion To Next Clinical Stages (Infections - Cellulitis - Fascitis _ _ _.)

Associated Complications, Sequelae Of The Disease Process, **Safely, In About 75% Cases.**

The Remaining About 25% Cases, Responded To Variable Extents, Needing Further Treatment.

Late Reporting Patients- 1-2 Week Of Onset, **In Different Stages Extent Of Progressivity- Fascitis,**

Cellulitis..., With Different Status Features Of 'PND' Shock Syndrome,

After Providing Information Regarding Available Management At Higher Specialized Centres,

e.g Immunotherapy, Hyper-Baric Oxygen Therapy, Better Intensive Care Units Etc.

After Preliminary Management & **Supportive Measures Ensured Safe, Secure Transport,**

Were Planned Referral (About ¼ Patients),

Otherwise, Continued Management At The Same Centre, With Prognosis Explained Treatment Risk Consent,

Recorded Comparative Increased Morbidity And About 1-2% Mortality.

However, **Management Of Pre-Existing & Or Precipitated Co-Morbidities –** Monitored Control Of

Diabetes, HTN, Deranged Renal Function, C-Reactive Proteins, HB%, Serum Proteins, Electrolytes, Minerals,

Blood Gas Chemistry, During Different Stages Of Ensuing Progressive Toxicemia, Septicemia Due To PF

Shock Syndrome, **Time Of Seeking Medical Attention & Standard Of Appropriate Care Are The**

Important Determinants For Overall Result Out Come, With Regard To Mortality & Morbidity Aspects.

CONFLICT OF INTEREST STATEMENT

The Author Declares That The Research Was Conducted In The Absence Of Any Commercial Or Financial Relationships, That Could Be Construed As A Potential Conflict Of Interest.

ACKNOWLEDGEMENTS

With Gratitude For Rendered Moral Support & Sincere Thanks To Study Material Resources- Print & Electronic Media.

Dr.(Mrs.) Poonam Sahni M.B.B.S, D.G.O(Gold Medalist)

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