A CASE REPORT ON SWEET SYNDROME IN K/C/O HYPOTHYROIDISM

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ABSTARCT:

Sweet syndrome was first defined in 1964 as a condition in which an acute febrile neutrophilic dermatosis occurs. It is a group of non- infectious disorders that are represented by neutrophilic infiltrations of the epidermis, dermis or hypodermis. A 40-year-old female patient reported with chief complaints of skin lesions over face, upper chest, back, legs for 20 days. The vital signs such as temperature, BP and pulse were normal with presence of right-side Inguinal Lymphadenopathy. O/E- M/E ill-defined erythematous papules, plaques, nodules, vesicles with crystal present. Particularly in this case acute neutrophilic dermatosis, skin lesions overs face, back upper chest and legs suggests that patient is suffering from sweet syndrome. The patient was diagnosed based on clinical pattern and physical Examination. Generally, patients with sweet syndrome are given systemic glucocorticoid therapy. With addition to this colchine, dapsone and potassium iodide are also equally effective.

KEY WORDS: sweet syndrome, febrile neutropenic dermatosis

INTRODUCTION:

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is a rare and distinctive inflammatory skin condition characterized by the sudden onset of fever, painful skin lesions, and an abundance of mature neutrophils (a type of white blood cell) in the affected tissues. It was first described by Dr. Robert Douglas Sweet in 1964, hence the name "Sweet syndrome"¹ Sweet syndrome typically presents as tender, red or purple skin lesions that appear.³ abruptly and are often located on the arms, legs, face, or neck. These lesions may initially resemble hives but rapidly evolve into painful nodules or plaques. Other symptoms can include fever, fatigue, joint pain, and general malaise. In some cases, internal organs, such as the eyes, lungs, liver, and kidneys, may be affected.² The exact cause of Sweet syndrome is not fully understood, but it is believed to be associated with abnormal immune system responses. It can occur as an idiopathic condition, meaning there is no identifiable underlying cause, or it can be triggered by various factors, including infections (such as respiratory or gastrointestinal infections), malignancies (particularly hematologic cancers), certain medications (such as granulocyte colony-stimulating factor), or autoimmune diseases.³ The diagnosis of Sweet syndrome is primarily clinical, based on the characteristic appearance of the skin lesions and the associated symptoms. However, a biopsy of the affected skin may be performed to confirm the diagnosis by showing dense infiltration of neutrophils in the dermis. Other tests, such as blood work and imaging studies, may be conducted to identify any underlying triggers or associated conditions.⁴ Treatment of Sweet syndrome involves addressing the underlying cause, if identified, and managing the symptoms. Systemic corticosteroids are the mainstay of therapy and often lead to rapid improvement of symptoms. In cases where corticosteroids are not well-tolerated or contraindicated, other immunosuppressive medications may be used. Additionally, supportive measures such as pain relief and wound care may be employed to promote healing.⁵ The immunohistopathological studies depicted that most cells of the infiltrate showed immunoreactivity for CD15, CD43, CD45, CD68, MAC386, HAM56, and lysozyme which is undeviating to the monocytic- histocytic immunopurified. The rigorous myeloperoxidase activity was found in many cells with histocytic appearance.

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CASE REPORT:

A 40-year-old female patient reported with chief complaints of skin lesions over face, upper chest, back, legs for 20 days. The vital signs such as temperature, BP and pulse were normal with presence of right-side Inguinal Lymphadenopathy.

O/E- M/E ill-defined erythematous papules, plaques, nodules, vesicles with crystal present.

The patient was also suffering from Hypothyroidism for 18 years and is currently on T. Thyroxine 100mcg (10D). The patient had normal physical and mental status with normal gait.

The patient was relatively asymptomatic before 20 days after which developed red raised skin lesion along with some fluid filled lesions, first over face gradually increasing in size and spreading over upper chest, back and legs. Itchy flaky scalp was noted. Lesions are associated with mild to moderate intensity itching which is relieved by medications. Patient had a travel history to Ujjain before 1.5 month. The patient had vegetarian food, her appetite was decreased, with normal bowel movements.

CLINICAL ASSESMENT:

The patient was presented with multiple ill-defined hyperpigmented, erythematous papules, plaques, nodules, vesicles with crystal over face, upper chest, back and leg. No other lesion, papules, nodules or erosion were over any other parts of the patient's body. All the examination of the patient was performed under sufficient day light with proper exposure.



Figure 1.1: Lesions seen over neck.



Figure 1.2: Lesions seen over both hand.



Figure 1.3: Papules seen over BL hand.



Figure 1.4: Lesions seen over of face.

GENERAL EXAMINATION:

Patients was conscious, cooperative and oriented to time and place

Temperature: 97⁰F Rt axilla

Pulse: 86 beats/min, regular Rt radial artery normal force, tension, volume and normal arterial wall thickness

RR: 14/min, thoracoabdominal regular

BP: 120/80 mm/Hg in Rt brachial artery in supine position

SPO2: 98% on RA

Rt Inguinal Lymphadenopathy

- Scalp: diffuse fine scaling
- Lesions over both B/L legs
- Back and buttocks: mild defined erythematous papules, nodules, plaques, vesicles with crusted lesions over back
- Upper chest lesions, erythematous papules, nodules, plaques, vesicles
- Both UL and LL: mild defined erythematous papules, nodules, plaques, vesicles

VITALS:

Blood pressure (BP): 120/80 mm/Hg, Respiratory Rate (RR): 14/min, Pulse Rate (PR):86 beats/min, RBS: 111 mg/dl, Oxygen Saturation (Spo₂): 98%

PATIENTS HISTORY OF PRESENT ILLNESS:

- **PRESENT HISTORY:** The patient was relatively asymptomatic before 20 days after which developed red raised skin lesion along with some fluid filled lesions and patients was currently suffering from skin lesions over face, upper chest, back, legs for 20 days. Lesions are associated with mild to moderate intensity itching. with presence of right-side Inguinal Lymphadenopathy. O/E- M/E ill-defined erythematous papules, plaques, nodules, vesicles with crystal present
- **PAST HISTORY:** The patient had normal physical and mental status with normal gait. K/C/O Hypothyroidism since 18 years. And was on medication- tab. Thyroxine-100mcg (10D)
- FAMILY HISTORY: H_x of diabetes (uncle), No history of autoimmune disease
- **DIET:** vegetarian diet
- **OBSTETERIC HISTORY:** $G_2P_0A_2L_0$, miscarriage (1st- 2month and 2nd 2.5 months)

| LABORA | TORY INVESTIG | GATION: |
|--------|---|---------|
| | the second se | |

| 5.00 | OBTAINED (7 th | OBTAINED | REFERENCE | |
|---------------|---------------------------|-----------------------|----------------|-----------------------|
| 1 march 10 | Aug) | (8 th Aug) | | INTERFERENCE |
| Haemoglobin | 11.30 g/dl | 10.92 g/dl | 11-15g/dl | Normal |
| WCB | 6300/cmm | 5300/cmm | 4000-10000/cmm | Normal |
| Platelets | 360000/cmm | 301000/cmm | 150000- | Normal |
| 1 san | | | 410000/cmm | |
| Neutrophils | 70% | 67% | 40-80% | Normal |
| Lymphocytes | 28% | 28% | 20-40% | Normal |
| Eosinophils | 1% | 4% | 1-6% | Normal Normal |
| Monocytes | 1% | 1% | 2-10% | Decreased |
| Sodium | 135 mEq/L | | 135-145 mEq/L | Normal Normal |
| Potassium | 3.8 mEq/L | - | 3.5-5.1 mEq/L | Normal Normal |
| Total | 1 mg/dl | - | 0.1-1.2 mg/dl | Normal Norm al |
| Bilirubin | | | | |
| Direct | 0.4 mg/dl | - | 0-0.4 mg/dl | Normal 🛛 👘 |
| Bilirubin | | | | |
| Indirect | 0.6 mg/dl | - | 0.1-0.8 mg/dl | Normal |
| Bilirubin | | | | |
| Total Protein | 6.8 mg/dl | OPEN ACC | 6-8 mg/dl | Normal |
| Albumin | 4.2 g/dl | _ | 3.2-5g/dl | Normal |
| Globulin 💛 | 2.6g/dl | - | 2.3-3.6g/dl | Normal 🧷 |
| A/G ratio | 1.62 | - | 1-2 | Normal |
| SGPT | 15 IU/L | - | 0-40 IU/L | Normal |
| SGOT | 25 IU/L | - | 0-37 IU/L | Normal |
| ALP | 81 U/L | - | 28-111 U/L | Normal |
| Urea | 26 mg/dl | - | 14-40 mg/dl | Normal |
| Creatinine | 1.02 mg/dl | - | 0.6-1.2 mg/dl | Normal |
| RBC | - | 3.98 x 10*6 | 3.8-4.8 x 10*6 | Normal |
| PCV | - | 33.11% | 36-46% | Normal |
| MCV | - | 83.09 fL | 83-101 fL | Normal |
| МСН | - | 27.4 pg | 27-32 pg | Normal |
| MCHC | - | 32.97 g/dl | 31.5-4.5g/dl | Normal |
| RDWcv | - | 14.78% | 11.6-13.7% | Increased |
| ESR | - | 48 mm | 0-12mm | Increased |
| Sr. CRP | - | 41.2 mg/L | 0-6 mg/L | Increased |

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PROVISIONAL DIAGNOSIS: SWEET SYNDROME?

SWEET SYNDROME LIKE ERYTHEMA NODOSUM LEPROSUM?

FINAL DIAGNOSIS: SWEET SYNDROME IN K/C/O HYPOTHYROIDISM

PLAN OF ACTION:

| SR.NO | DRUG | DOSE | ROUTE | FREQUENCY |
|-------|-----------------------|--------|-------|----------------|
| 1 | Inj. dexamethasone | 1.5cc | iv | OD in morning |
| 2 | Inj. Cefotaxime | 1 gm | iv | 12 hry |
| 3 | Inj. pantoprazole | 40 mg | iv | 12 hrly |
| 4 | Tab. CPM | 4 mg | oral | 1-1-1 |
| 5 | Tab. diclofenac | 50 mg | oral | SOS |
| 6 | Tab. MVBC/FE/FA | 500/5 | oral | 1-0-1 |
| | | mg | | |
| 7 | Tab. Ca ⁺⁺ | 500 mg | oral | 1-0-1 |
| 8 | Fusidic acid cream | | LABD | 3-4 time a day |
| 9 | Betapic cream | | LABD | 3-4 time a day |
| 10 | Tab. Eltroxin | 100 mg | oral | ODin morning |
| 11 | Tab. PCM | 500 mg | oral | SOS |

DISCUSSION:

The syndrome is an acute febrile neutrophilic dermatosis. It is a group of non-infectious disorders that are represented by neutrophilic infiltration of the epidermis, dermis or hypodermis. It can usually develop with or without vasculitis. The aetiology of the syndrome is idiopathic or secondary to an underlying disorder, localized or generalized, and may or may not have extracutaneous manifestations ^[1]. The immunohistopathological studies depicted that most cells of the infiltrate showed immunoreactivity for CD15, CD43, CD45, CD68, MAC386, HAM56, and lysozyme which is undeviating to the monocytic- histocytic immunopurified. As the patient in this case is recovering with the medication therapy only. The further investigation of the lesions was done. The size and area covered by the lesions were decreased along with its intensity. The severity of the infection needs to be assessed and analysed based on the cause and invasion of the organism. In some cases organisms causes severe infection or deeply invaded and in such cases patients do not respond well to the therapy as they are resistant to that therapy so in such cases alternative regimens are used.

CONCLUSION:

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The patients in this case has received the therapy based on culture and sensitivity testing and the laboratory testing and has adhere to the medications so that they are less severe to the case and improving the recovery rate. the patients also had hypothyroidism history so for that also patient was taking medication daily. sweet syndrome refers to the symptomatic treatment and relief from the lesions, papules, vesicles and scares that are -resent all over the body.

CONFLICT OF INTEREST:

Authors have no conflict of interest.

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