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Case Report

A 6- Month-Old-Child with Sweet Syndrome in Tanzania: A Case Report

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Abstract: ***Background*:** Sweet syndrome or acute febrile neutrophilic dermatosis is a recurrent and rare skin disease caused by the release of cytokines, with diverse possible etiologic causes mainly being associated with infections, malignancy and drugs, with a reported incidence of 0.08 pediatric cases Historically, the Von den Driesch diagnostic criteria has been used to diagnose sweet syndrome. ***Case Presentation*:** We report a 6 month-old African female infant who presented with features of sweet syndrome. This case illustrates the typical presentation, diagnosis, and treatment outcome of this highly misdiagnosed condition. ***Conclusion*:** Due to the limited resources and knowledge about this under-reported disease in resource –constrained settings, the characteristic manifestation of sweet syndrome can be easily missed and thus requires a high index of suspicion for earlier diagnosis and management.

Keywords: Sweet Syndrome, Acute Febrile Neutrophilic, Tanzania.

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BACKGROUND

Sweet Syndrome is a febrile neutrophilic dermatosis, characterized by the onset of painful erythematous-violaceous lesions that may be located mainly on limbs, trunk, face and neck. [1,2] The disease is universally distributed among the races. In adults, women are more commonly affected, mainly with the classical (idiopathic), pregnancy related sweet syndrome and drug-induced forms. The first episode generally occurs between the ages of 30 and 50 years [1-3]. Cases associated with neoplasms, on the other hand, are equally distributed between the genders, while cases described in children are rare (8% of the total), equally distributed between the genders and are preceded by one to three week by infectious diseases of the upper respiratory tract (streptococci), gastrointestinal tract (salmonellosis and yersiniosis), and inflammatory bowel disease [1-4].

CASE PRESENTATION

A 6 –month- old African female infant presented to Muhimbili National Hospital, Dar es salam, Tanzania with complaints of recurrent convulsions which started 8 hours post-delivery, jerk like rhythmic movements involving upper and lower limbs with starring of eyes lasting for 2 to 3 minutes. The convulsion could occur 3 to 5 times per day with similar presentation but with varying durations, with interval of 4 to 6 hours apart. She was started on phenobarbitone (loading dose 20mg/kg then mentainance dose of 5mg/kg/day) the convulsions were well controlled, last event was 8 weeks prior to admission.

Progressive skin lesion since age of 10 weeks which were acute onset, small rash with blister on the fore head, upper and lower limbs bilaterally which characterized by erythematous plaques some filled with pus, raised borders and central necrosis, some bleeds when doing general body cleanness. Rashes were none itching sparing oral cavity, trunk, palms and soles of feet.

Fever started 2 weeks prior to admission which was gradual onset, high grade, continuously with nonspecific in periodicity, temporary relieved by oral paracetamol given 6 hourly (at Hospital) accompanied by reduced breastfeeding frequencies, baby use to breast feed less as compared to previously hence expressed breast milk were calculated to support her nutrition intake. Fever lasted for 8 days. There were no history of runny nose or cough, ear ache or pain, no joint pain or swelling, no history of jaundice neither bleeding tendencies, baby had normal bowel and urination habits.

Baby was born via spontaneous vagina delivery (SVD) with birth weight of 3.8kg and Apgar score of 7 and 10 in 1st and 5th minutes respectively, was able to breastfed within 1 hour post-delivery. She is a fourth born in a family of 3 living children, first born is a 10 –

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year-old male child alive and doing well, 2nd born was a male child died at age of 2 years due to recurrent convulsion for 1 month and was diagnosed to have Brain tumor (these ones shared father). The 3rd born is female aged 6 years who has her own father different from the 1st and the index (4th born). Child had received all vaccines according to immunization vaccination development programme(IVPD) and has normal developmental mile stone per age.

Mother is a 36 years graduate who was diagnosed to have syphilis during 2nd pregnancy, treated with intramuscular medication for 5 days (no documentation), but during 4th pregnant at gestation age (GA) of 20/40 had rashes appearing on hands and face, hard small masses around the genitalia ,with foul smelling vagina discharge, whitish in nature treated with oral antibiotic and ant fungal vaginal pessaries for 1/52 Venereal Disease Research Laboratory(VDLR) which is the screening for syphilis was negative though at 28/40 had history of febrile illness repeated VDRL was positive and treated with intramuscular medication(not documented) once a day for 3 days. At gastation age of 38 weeks VDRL was positive mother was not treated as she was in labor and even after delivery was not treated. Otherwise she had normal weight gain during index pregnant, normotensive with negative HIV status.

Examination on the day of admission, she was alert with no emergency sign, febrile at 39.0^0c, had some palmar pallor with normal oral mucosal membrane, ear and throat were normal, no dysmorphic features, no lymphadenopathy, not jaundiced, not cyanotic no lower limb edema (on subsequent days had edematous limbs more on the lesions site). Heart rate of 96b/minutes, Respiratory rate 32 breath/mins SPO_2 98% on room air Weight 6kg, Length 64cm 75 percentile) weight/length =Lies between -2 and -3 SD, OFC=40cm (85percentile) AF =Couldn't be measured due to skin lesion all over the head. (Figure 1) Had normal systemic examination.

Figure 1: showed scalp and fore arm lesions on the day of admission on which topical antibiotics (unknown) was applied

Local examination: Has vesico-purpular lesions more on the face, upper and lower limbs sparing the trunk. New ones**:** Well defined with tender erythematous plaque and nodules which are hard and pale at the center (dermal edema), on the lower limbs are of annular appearance, While Old ones**:** Hyerkeratinized with crusted plaque these lesions vary in size ranging 0.3x0.5 cm while largest 3cm x 7 cm **(Figure 2 below)**.

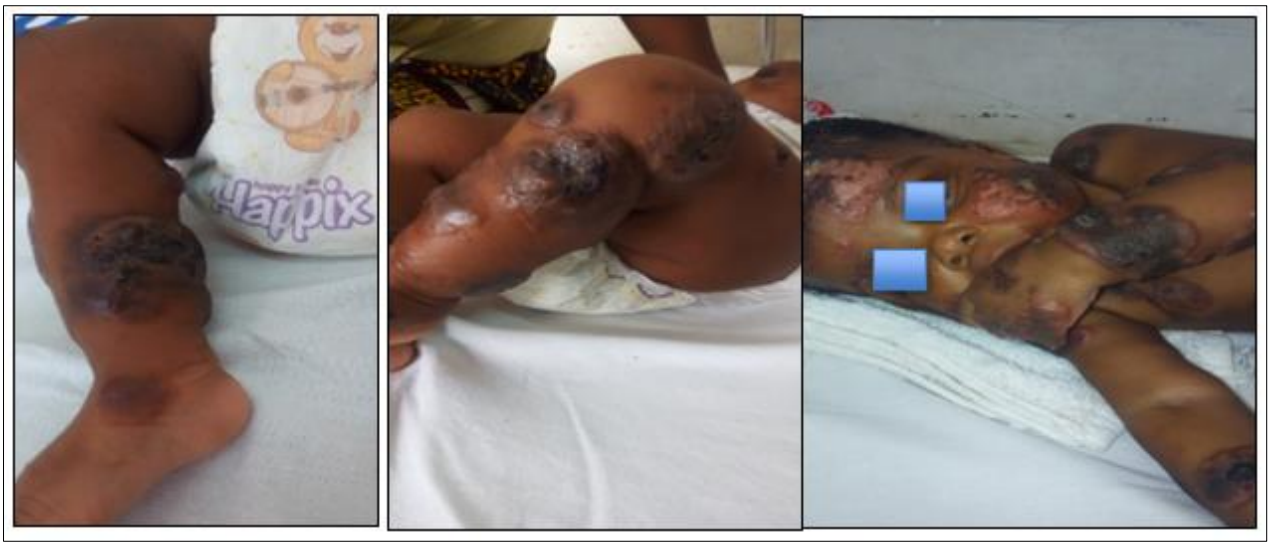

Figure 2: showed lesions on subsequent days when child was admitted at MNH

Based on history and physical examination, we had provisional diagnosis of congenital syphilis complicated to neuro syphilis with differentials of tuberculous gumma, granuloma annulare, septicemia secondary to infected skin lesions and febrile neutrophilia dermatosis.

Several investigations were done to confirm the diagnosis and its complications as shown in the table below.

Tab 1

Investigation	**Day 1**	**Day 7**	**Day 15**	**Day 20**
Crp	205	188	123.4	13.9
wcc	15.7	13.9	7.2	
Neutrophil	19.9(88.4)	15.6(81.7)	12.2(61.3)	
Hb(gm/dl)	7.4	8.02	10.4	
Platelets	794	324	266	
ALT(IU/L)	23		13	
AST(IU/L)	22		21	
LDH(IU/L)	965	736	490	231
Createnine	34.3		32.3	
Serum ironUG/DL)	40			
Feritin	894.12		102	
Serum transferitin	1.24			
Serum albumin	33		34	
VDRL	POSITIVE			

CRP=C reactive protein, WBC=White blood cell, HB=Hemoglobin, ALT=Alanine transaminase, AST=Aspartate transaminase, LDH=Lactate dehydrogenase, VDRL=Venereal Disease research laboratory.

Other investigations which were done including X ray of the long bones which were all normal, electrolytes were within normal ranges. Random blood glucose was 6.1 mom/l, lumber puncture was clear macroscopic, high protein (1.09), pleocytosis predominant lymphocyte with glucose of 3.71 and positive for VDRL. Treponemal hemoagglutination test (TPHA) and Fluorescant treponemal antibody absorption (FTA) were negative also human immunodeficiency virus test (HIV), Hepatitis A and BsAg, Toxoplasmosis IgM, Rubera IgM, CMV IgM were negative.

Urinalysis was normal, urine culture and blood culture were no growth within 7 days, Pus swab for culture and sensitivity showed polymorphs lymphocyte +2, while Ziehl-Neelsen (ZN) stain was negative, echocardiogram was normal while cranial ultrasound was not done due to multiple wet painfully lesions on the head including area for acoustic windows.

A cutaneous incisional biopsy was done on a lesion on a left lower limb which was analyzed at Muhimbili national Hospital (MNH) and Dublin. The results at MNH showed Keratinized epidermis with inflammatory infiltrates of mixed types of cells mainly Lymphocytes and numerous macrophages engulfed cellular debris, while results from Dublin showed Irregular and focally pseudo-epitheliomatous epidermal

hyperplasia Neutrophil infiltrates in epidermis within hair follicles, no features of neoplastic process, ZN was negative hence the diagnosis of deep seated fungal infection, perforating disorder and histiocytic (though should not involve epidermis)and Sweet syndrome (though rarely ulcerate) were reached with the final diagnosis of congenital neuro-cutaneous syphilis complicated to sweet syndrome was made. Image below show the histopathological slide view

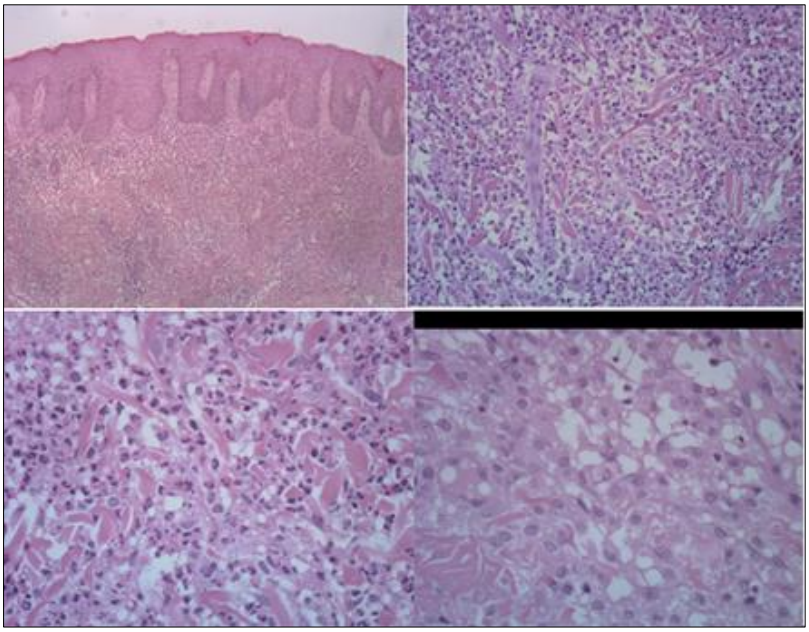

Figure 3: showed morphological images in different power view (from low to high power field) of the tissue biopsy done in Dublin showed Irregular and focally pseudo-epitheliomatous epidermal hyperplasia Neutrophil infiltrates in epidermis within hair follicles, no features of neoplastic process, ZN stain was negative

Child was treated with Benzathine penicillin G 150,000 iu intramuscular once a day for 10/7, Phenobabitone tabs 30 mg nocte oral 1/12,Paracetamol 90 mg iv 8 hourly 24 hours then tabs 125 mg oral 8 hourly 5 days ,Blood transfusion 120 mil of whole blood 2 times in interval of 2 days and the control was 8.02g/dl, Folic acid 5mg once a day for 1/12 ,Daily dressing with application of mupirocine creamVitamin A 50,000U(D1, D2 and D14), Prednisolone 2.5 mg 8 hourly 14 days then medication was tapped to 2.5 mg bid 7/7 followed by 2.5 mg once a day 7/7,Vitamin D and calcium 5mils (400iu) once a day for 2/12,Omeprazole 10 mg once a day for 1/12. After stopping the prednisolone we started mometasone furoate 1% on feeding child received EBM: 165mils @3hourly.

Child's mother was investigated, both VDRL, and TPHA were positive hence a treatment of Benzathine penicillin G 2.4 mu im weekly for 3/52 was provided.

DISCUSSION

Sweet syndrome The Sweet Syndrome (SS) is an acute, febrile neutrophilic dermatosis, first described by Robert Douglas Sweet, in (1964) [1]. It is characterized by the acute onset of fever, erythematous-violaceous cutaneous lesions, usually painful, located mainly on limbs, trunk, face or neck [2]. The inflammatory edema may impart the aspect of lesions with vesiculations, but palpation shows that actually the lesions are solid, somewhat softened; the name given to this is pseudovesiculation. As the lesion regresses, there may be a central lightening of the lesion color, giving it a target aspect similar to erythema multiforme [1, 2]. Women are more frequently affected and seem to be particularly involved by the idiopathic or drug induced forms [1-4]. Of these, our patient had fever, extremities, face, scalp erythematous-violaceous painful lesion.

In 1986 Su and Liu proposed diagnostic criteria for SS, which was modified by Von den Driesch in 1994.[6] which based on 2 major criteria and 2 minor criteria, Major criteria are, (1) sudden onset of painful erythematous or violaceous plaques or nodules; (2) a predominantly neutrophilic dermal infiltrate, without leukocytoclastic vasculitis while minor one includes (1) fever or prior infection; (2) arthralgia, conjunctivitis or malignancy; (3) leukocytosis;(4) favorable response to use of corticosteroid therapy. Histopathology shows visible perivascular neutrophilic nodular infiltrates, with necrophilia cariorrexis. Even though there is no primary vasculitis, blood vessels may be secondarily involved in immunologic response, an infrequent finding.[6,8,12]Laboratory alterations include peripheral leukocytosis with neutrophilia and elevated speed of erythrocyte sedimentation or C-reactive protein, particularly in cases where SS associated with malignancy, leukopenia, anemia and thrombocytopenia have been reported [3-10].

We thus confirm our diagnosis of SS based on both major and minor criteria, elevated C - reactive protein and histological findings from our patient. The recommended treatment is corticosteroid therapy, either oral (30 to 60mg/day for 4 to 6 weeks in adult patient) or intralesional.[1-6] with rate of recurrences up to 30 - 50% of cases, depending on the etiology. Our patient responded well to oral and topical corticosteroid, being followed closely in her Renal profile and opthalmology review every three months, Neuro-developmental assessment, BP, BWT and RBG monthly, Height every six months. Currently child has 5 years doing well and still we are doing follow up annually.

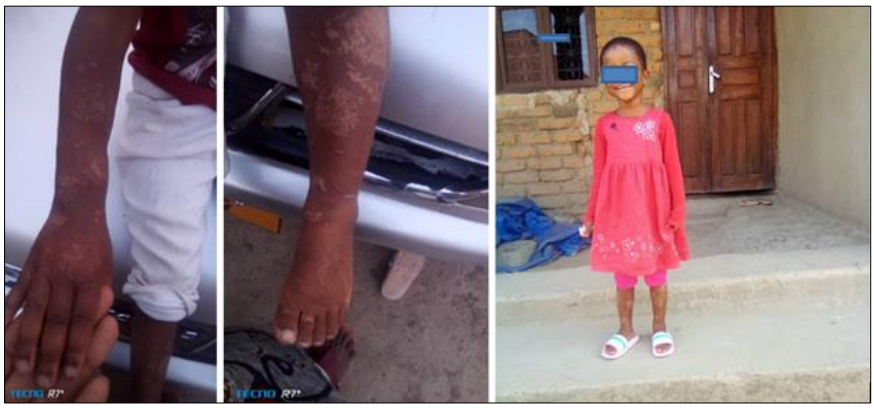

Figure 4: Child at age of 3 years

Figure 5: Child at age of 5 years

CONCLUSION

The incidence of pediatric SS in industrialized is reported to be 8% of all cases, to our knowledge, there is no incidence reported in Africa, however several cases for adult with SS has been reported Our case implicates the characteristics manifestation of SS can be clearly misdiagnosed and thus under reported. We should have a high index of suspicion and use the clinical and investigative modalities to make a diagnosis of SS.

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Authors' Contributions

AM and JS admitted and attended to the child. They also prepared the manuscript. EK is the lecturer Pediatric neurologist, together with LM, PS reviewed the case and provided corrections and inputs to this case report while PS provided assistance for sample to be analyzed in Dublin. All authors read and approved the final manuscript.

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Declaration

Ethics approval and consent to participate: Not applicable.

Consent for Publication

Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying images. A copy of the

written consent is available for review by the Editor-in-Chief of this journal.

Competing Interests: The authors declare no competing interest.

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