CASE REPORT

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A fatal case of Norwegian scabies in a patient with diabetes mellitus

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ABSTRACT

Background: Diabetes mellitus (DM) is characterized by hyperglycemia due to a defect in insulin secretion, action, or both. Immunocompromised individuals such as those with DM tend to suffer from skin disorders, including crusted scabies (Norwegian scabies). However, studies regarding the incidence of Norwegian scabies are still limited. This article reports a case of an elderly diabetic patient suffering from crusted scabies.

Case Presentation: A 67-year-old male patient was referred to Dr. Soetomo Hospital with sepsis caused by suspected skin infection, hypertension, and type 2 DM. The patient presented with a chief complaint of recurrent skin diseases throughout the body and itching for the past three years. The itching became severe in the last three weeks prior to the hospital admission. The patient had a medical history of diabetes, tuberculosis, hypertension, and arrhythmia. Based on our assessments the patient was diagnosed with crusted scabies, septic shock, sepsis, pneumonia, pulmonary tuberculosis, hypoalbuminemia, type 2 DM, and erythroderma. The patient received oxygen, antibiotics, norepinephrine, albumin, ranitidine, metoclopramide, NovoRapid, ventolin nebules, rehydration, topical keratolytic cream, and scabicidal cream as therapies. However, on the 3rd day of the treatment, the patient suffered from type 1 respiratory failure and passed away the next day due to septic shock.

Conclusion: We reported a diabetic male patient suffering from crusted scabies, accompanied by pneumonia, pulmonary tuberculosis, hypoalbuminemia, and type 1 respiratory failure. Secondary infections had been developed at the time of presentation. Therefore, establishing an early diagnosis of Norwegian scabies will help prevent the disease's progression.

Keywords: Diabetes mellitus, crusted scabies, Norwegian scabies, hyperkeratosis, *Sarcoptes scabiei*.
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INTRODUCTION

Diabetes mellitus (DM) is a metabolic disease associated with the lack of effective insulin due to dysfunction of pancreatic beta cells, impaired glucose uptake in peripheral tissues in type-2 DM, or an absolute lack of insulin in type-1 DM. The primary disorder lies in carbohydrate metabolism and the secondary impairment lies in fat and protein metabolism.[1] In 2015, Indonesia was recognized as the seventh-highest rank of diabetic worldwide, with an estimated 10 million cases nationwide.[2] Furthermore, DM with complications is the third-highest cause of mortality in Indonesia and associated with long-term complications.[3,4]

Scabies infection is one of the complications occurring in patients with DM. Scabies is a skin disorder caused by *Sarcoptes scabiei* var. Hominis infection. Crusted scabies was described earlier by Boeck and Danielssen in Norway in 1848 among lepers, which was then referred to as Scabies Norvegi Boekiî in 1862. A severe condition of scabies occurs as widespread hyperkeratotic crusted lesions, hence the name "crusted scabies" is preferred to "Norwegian scabies".[5,6] Norwegian scabies is characterized by hyperkeratosis (skin hardening).[5,6] The worldwide incidence of scabies in 2014 affected approximately 130 million people as reported by the World Health Organization (WHO), while the International Alliance for the Control of Scabies (IACS) suggested that its incidence varied from 0.3% to 46% in the same year worldwide.[7]

Scabies is endemic in tropical regions and is commonly discovered in Indonesia. Despite its prevalence has been reportedly decreasing over the year in the country according to the Indonesian Ministry of Health, scabies disease, particularly crusted scabies, is still considered one of the concerning infectious diseases in Indonesia.[8] Furthermore, Norwegian scabies is more dangerous since their lesions often predispose patients to secondary infections.[9] Nevertheless, studies regarding the incidence of Norwegian scabies are still limited. This article reports a case of an elderly diabetic patient suffering from Crusted (Norwegian) Scabies.

CASE PRESENTATION

A 67-year-old male was referred to the emergency unit of Dr. Soetomo Hospital with sepsis *et causa* suspected skin infection with hypertension and type-2 DM. The results of blood test indicated: hemoglobin 10.6 g/dL, hematocrit 36.6%, mean corpuscular concentration (MCV) 97.6, mean corpuscular hemoglobin (MCH) 28.2, mean corpuscular hemoglobin concentration (MCHC) 28.9, leukocyte 33,960/mm^3, platelet 287,000/mm^3, non-reactive HIV, blood urea nitrogen (BUN) 25 mg/dL, serum creatinine of 1.24 mg/dL, sodium 135 mmol/L, potassium 3.8 mmol/L, and chloride 101 mmol/L (Table

1). Thorax imaging exhibited fibrotic lesions in the right suprahilar, whereas heart was within normal limits (Figure 1). Before referring to the Dr. Soetomo Hospital, the patient had been treated with ringer lactate with sodium chloride infusion 25 mEq, paracetamol infusion 1 g, intravenous ranitidine, intravenous ondansetron, and intravenous NovoRapid 3x4 units.

At presentation, the patient complained of ~3-year recurrent skin diseases throughout the body and itching that comes and goes. The itch had become intense in the past three weeks before the hospital admission and spread throughout the body causing scratching wounds. The itchy areas thickened, developed into crusty plaque, and became infected (bleeding and purulent). The patient used to do self-medication whenever the itch appeared. Other members of the family also experienced less severe itching all over the body. The patient also complained of shortness of breath, high fever for three days, decreased appetite, weakness, nausea, and vomiting every meal. Coughing and drastic weight loss were denied. The patient tended to be drowsy and was difficult to communicate. The patient had a history of DM since 2004, for which he received metformin 500 mg and glibenclamide 5 mg. The patient also had a history of tuberculosis and underwent treatment twice (in 2010 and 2015) at a public medical center for six months and stopped. A history of hypertension and arrhythmia was also reported five years ago but did not undergo any therapy. There was no history of jaundice, transfusion, or surgery.

Physical examination indicated general weakness, Glasgow Coma Scale (GCS) of 3-5-6, blood pleasure of 80/60 mmHg, heart rate of 110 x/minute, respiratory rate of 24x/second, and temperature of 37.8°C. Head and neck assessment exhibited dyspnea, normal jugular venous pressure (JPV), no anemic conjunctiva, no scleral icterus, and no cyanosis. Chest examination revealed symmetrical movements. No intercostal or supraclavicular retractions were found. A regular single S1 and S2 were detected. Murmur, gallops, or pericardial friction rub was not heard. Lung examination exhibited vesicular breathing over both of the hemithorax, decreasing breath sound in the upper right hemithorax, and rhonchi and wheezing in the right hemithorax. The results of the abdominal examination showed flat symmetric, normal bowel sounds, and non-palpable liver and spleen. Examination of the extremities showed a warm dry red acral and edema was not found. Hyperkeratosis, plaques, and erythematous macules were found throughout the body (Figure 2A and B). The lesions also formed fissures. The crusts

Table 1. The results of the patient's laboratory test.

Lab parameters (unit)	Results			
	Initial test	Day 2	Day 3	Day 4
Blood test				
Hemoglobin (g/dL)	10.6			
Hematocrits (%)	36.6			
Mean corpuscular volume (fl)	97.6			
Mean corpuscular hemoglobin (pg)	28.2			
Mean corpuscular hemoglobin concentration (g/dL)	28.9			
Leukocytes (/mm^3)	33,960			
Platelets (/mm^3)	287,000			
HBsAg	Non-reactive			
Blood urea nitrogen (mg/dL)	25			
Serum creatinine (mg/dL)	1.24			
Sodium (mmol/L)	135			
Potassium (mmol/L)	3.8			
Chloride (mmol/L)	101			
Random blood glucose (mg/dL)	221	163	193	203
Serum glutamic oxaloacetic transaminase (U/L)	163			
Serum glutamic pyruvic transaminase (U/L)	101			
Albumin (g/dL)	2.31	2.5	2.0	
Total bilirubin (mg/dL)	1.49			
Direct bilirubin (mg/dL)	0.9			
HbA1c (%)			11.9	
Fasting blood glucose (mg/dL)				504
Blood gas analysis				
pH		7.48	7.3	
pCO_2 (mmHg)		35	32	
pO_2 (mmHg)		73	47	
HCO_3 (mEq/L)		26.1	16.1	
BE (mmol/L)		2.6	-10.2	
SaO_2 (%)		96	78	

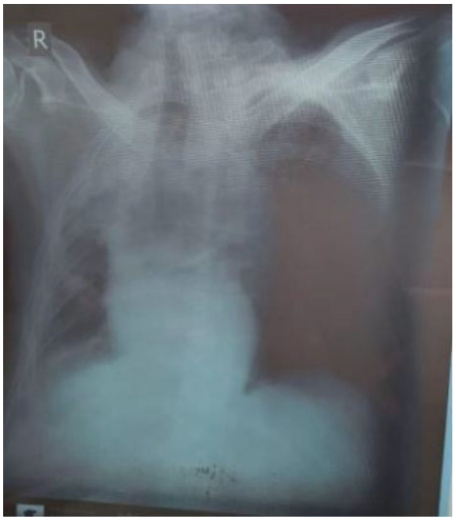

Figure 1. Chest x-ray revealed the presence of fibrotic lesions in the right suprahilar of the lung.

were found on the palms, elbow extensors, scalp, ears, and both feet. They were cream, grey, and yellow-brown in color and were firmly adherent. The undersurface was observably smooth, red, moist, and velvety when the crust was removed. There was a sign of inflammation on the soles and toes (Figure 2C and D).

The results of laboratory examination indicated a high level of leukocytes, high level of random blood sugar, low level of albumin, negative for hepatitis B surface antigen (HBsAg), and liver damage (Table 1). Urine test showed glycosuria, but negative for leukocytes, nitrite, protein, ketone, bilirubin, erythrocytes, and epithelial cells. pH (5) and urobilinogen (0.2 mg/dL) were within normal limit. The urine was normal in color (dark yellow) and clear.

Based on the anamnesis, physical examination, and laboratory findings, the patient was initially diagnosed with septic shock + sepsis + suspected pneumonia + inactive old pulmonary tuberculosis + type-2 DM + hypoalbuminemia + corrected hypokalemia + suspected crusted + erythroderma. The patient was then suggested to undergo a skin scraping test and lung imaging three days following antibiotic therapy.

The patient was treated with a simple mask oxygen 6 liter per minute (lpm), a diabetic diet of 2100 Kcal/day, NaCl 0.9% 500 mL/hour, sodium chloride infusion 0.9% 1500 mL/24 hours, norepinephrine drip 50 Nano, ceftriaxone injection 1 gram/12 hours, albumin drip 20% in 4 hours, ranitidine injection 50 mg/12 hours, metoclopramide injection/8 hours, sub-cutaneous NovoRapid injection 4 units/8 hours after meal, cetirizine 10 mg/12 hours orally, and sodium fusidate cream.

On the next day of treatment, weakness, shortness of breath, nausea, and declined appetite were still observed. The patient tended to be sleepy, while the hands were found uncontrollably scratching all over the body. GCS was 3-5-6. Vitals signs showed blood pressure of 80/40 mmHg, heart rate of 110 x/minute, respiratory rate of 24x/second, and temperature of 37.2°C. A low albumin level after correction and a high level of random blood sugar was still recorded. Blood gas analysis indicated decreased pO_2 (Table 1). The result of the skin scraping was positive for *Sarcoptes scabie*, indicative of crusted scabies. The patient was then diagnosed with septic shock + sepsis + pneumonia + pulmonary tuberculosis + type-2 DM + hypoalbuminemia + corrected hypokalemia + crusted scabies. The same therapies were continued along with the addition of ventolin nebules/8 hours and an increased dose of norepinephrine drip (100 Nano). Permethrin cream 5% was also prescribed twice a week (applied all over the body at night and rinsed off in the morning). Albumin therapy was stopped and meropenem of 1 gram/12 hours was injected to replace the ceftriaxone.

The patient was found anxious on the 3rd day of the treatment. Narrow breathing, low food intake, and nausea were observed. Blood pressure was 100/60 mmHg and heart rate was 98x/minute. Laboratory findings revealed decreased albumin level (2.0 g/dL), increased HbA1c (11.9%), and increased random blood glucose (193 mg/dL), indicative of hypoalbuminemia and type-2 DM. Blood gas analysis showed abnormal results (Table 1), suggesting that the patient suffered from type 1 respiratory failure. The patient was referred to anesthesiology and was assessed with respiratory failure; however, the patient's family refused for ventilator treatment in the Intensive Care Unit (ICU). Tube feeding and reservoir mask oxygen 12 lpm were given, along with an increased dose of norepinephrine drip (150 Nano). Further, the exact same medication as day 2 was continued.

On the next day, the patient's consciousness declined (GCS from 2-3-4 à 1-1-1). Blood pressure was 80/60 mmHg and heart rate 98x/minute. Fasting blood sugar was high (504 mg/dL), thus intravenous insulin was injected 3 times. Random blood glucose was 203 mg/dL. Further, at 10.15 a.m., the patient stopped breathing along with a cardiac arrest. The patient's family refused cardiopulmonary resuscitation (CPR) and the patient was confirmed died due to septic shock. Direct microscopic examination of the patient palmar's scraping sample of the lesions indicated the presence of a number of mites, eggs, and feces (Figure 3). Urine and blood culture assay exhibited no growth of aerobic and anaerobic organisms.

DISCUSSION

DM is the most common inherited metabolic disorder affecting people worldwide. DM can be diagnosed based on plasma glucose criteria: either fasting blood sugar or 2-hour postprandial blood sugar, glucose tolerance test, or A1C criteria.[1,10] The patient had been diagnosed with type-2 DM since 2004, but did not undergo regular treatment. During the current hospitalization, increased levels of random blood sugar (504 mg/dL) and A1C (11.9%) were observed in the patients, confirming the presence of type-2 DM.

Individuals with a weakened immune system such as DM and human immunodeficiency (HIV), neurological diseases, mental disorders, the elderly, and the disabled, often suffered from skin infections such as Norwegian scabies.[6,8,11] This disease is characterized by a thickened skin layer containing thousands to millions number of mites and scabies eggs. Unlike the usual scabies infection, symptoms of itching and rash may not be present.[12,13] Itching, if present, will disappear on its own over time. Norwegian scabies does not usually appear acutely erupted as in classic scabies.[12] The plaque consists of parakeratotic crusts that vary in thickness (3 to 15 mm). The plaque is cream, yellow-brown, yellow-green or grey in color and adheres tightly and firmly. When the lesion is very large and thick, the surface

may rupture into fissures. When the crust is removed, the undersurface appears to be smooth, red, moist, and soft. Such crusts are seen on the palms, elbow extensors, scalp, ears, soles, and feet. Norwegian scabies can also appear as a psoriasiform skin infection and a warty dermatosis. Norwegian scabies can cause, but rarely, erythroderma.[14] Diagnosis is based on the clinical findings and demonstration of mites. The presence of mites, eggs, and mites' faeces confirms scabies' diagnostic.[15]

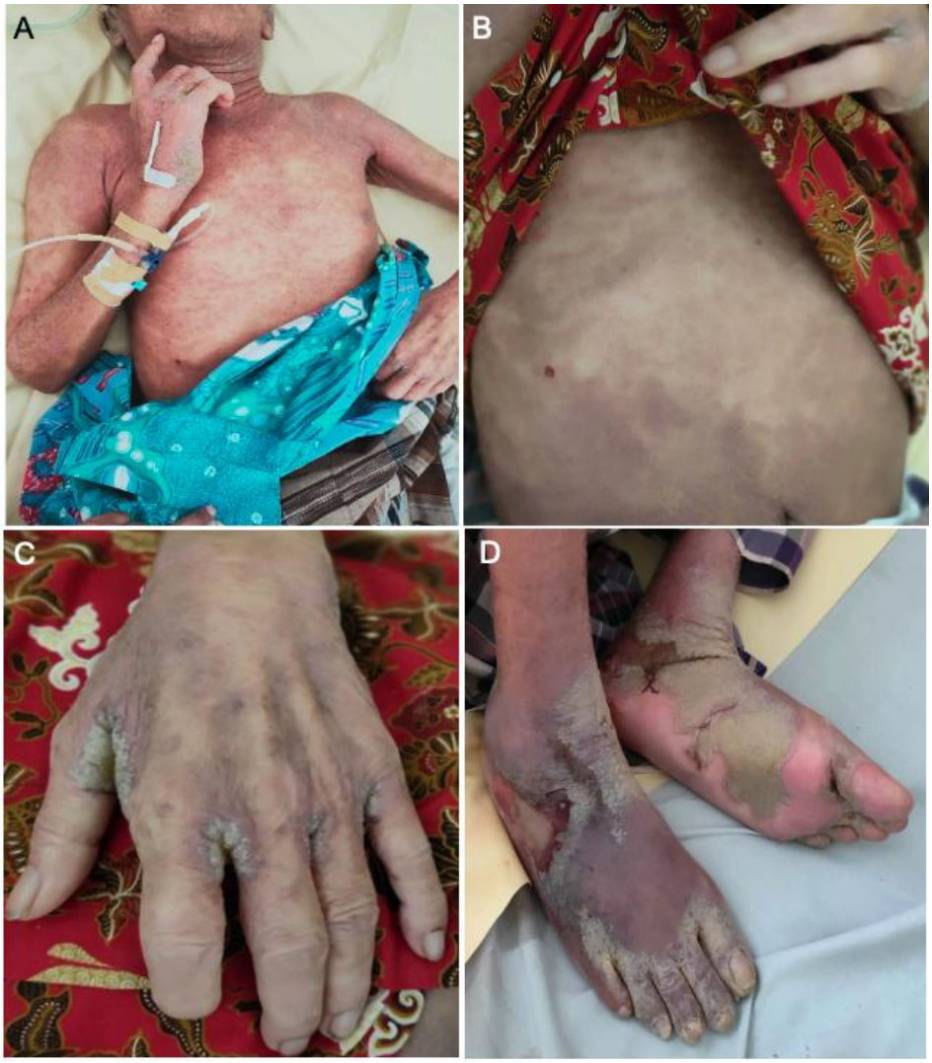

Figure 2. Hyperkeratotic crust throughout the body with cream, grey, or yellow-brown crusts which are firmly adherent (A and B). The lesions over the extensor surface showed fissuring with undersurfaces are smooth, red, moist, and velvety when the crusts are removed (C and D).

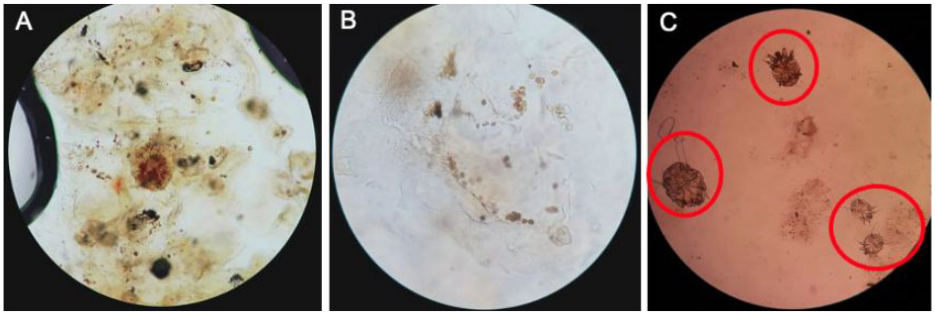

Figure 3. Direct microscopic investigation of the scraping samples showed the presence mites, eggs, and feces. The samples were prepared using immersion oil (A-B) and 10% potassium hydroxide (C).

In this case, the patient had a complaint of an extensive skin disease since the past three years. The itch became intense and spread throughout the body three weeks prior to the hospital admission, resulting in the formation of scratching wounds. The itchy area was found thickened, crusted, plaque, bleeding, and festering due to secondary infection. Hyperkeratotic lesions were discovered all over the body and formed fissures. The crusts were cream, grey, and yellow-brown in color which were firmly adherent. The undersurface was observably smooth, red, moist, and velvety when the crust was removed. The crusts were found throughout the body (palms, elbow extensors, scalp, ears, and feet) and indicated inflammation. A number of mites, eggs, and feces were also detected during a direct microscopic investigation of scraping samples, confirming the diagnosis of Norwegian scabies.

In patients immunocompromised, the number of mites can rapidly increase. An impaired immune response, lack of pruritus, or physical inability of patients to scratch might be assumingly associated with the rapid growth of the mites. Clinically, eruption might occur due to the thickening and crusting of the skin.[15] Pruritus in the elderly may be associated with senile pruritus, and the unusual presentation of scabies in elderly could delay the diagnosis.[12]

Despite the patient in our case having experienced a wide range of skin diseases for the past 3 years, the disease was not properly diagnosed. The patient often underwent self-medication to overcome the ailments. In addition, unobvious itching also obscured the diagnosis. At the time of hospital admission, a secondary infection had been developed. A decreased immune response led to hyperinfestation and absence of itching. DM-related peripheral neuropathy might have also been associated with the absence of itching in the patient, resulting in a worse disease progression.

Treatment of Norwegian scabies is considerably intricate due to hyperkeratotic

skin, nail involvement, and high parasite burden. Immunocompromised condition is another factor challenging the treatment of this disease. Several therapies such as the use of topical keratolytic agents, scabicidal agents, and oral ivermectin have been suggested to treat the disease. Keratolytic substances including a 5-10% salicylic acid in petrolatum and 40% urea are useful to remove hyperkeratotic skin and increase drug penetration. Crust removal can also be accomplished by soaking the body in a hot tub. These therapies can help decrease the mite burden and increase the efficacy of topical scabicides.[12] Furthermore, isolating crusted-scabies patients is critically important to prevent scabies outbreaks. Adequate care must be given to dying or paralyzed patients to prevent relapse. Norwegian scabies may become complicated due to secondary bacterial infection (*Staphylococcus aureus*). If septicemia occurs, aggressive treatments with broad-spectrum antibiotics should be performed.[5]

To date, no available method has been developed for measuring the severity of Norwegian scabies that can be used to predict treatment duration. However, Joshua S. Davis and team have developed a grading scale based on the skin examination to help classify the disease severity (mild, moderate, or severe), which later can be used to predict the duration of treatment. The levels of disease severity are determined based on clinical assessment of four main indicators: the distribution of crusting; the degree (depth) of crusting; the degree of skin cracking and pyoderma; and the number of previous episodes.[16] Based on this grading scale, we found our patient had grade 3 (severe) Norwegian scabies. Therefore, permethrin cream of 5% (which was applied all over the body at night and rinsed off in the morning) was given twice a week. In addition, sodium fusidate cream was also prescribed and the infected lesion was treated and compressed. Despite all the therapies given, the patient was found in a state of septic shock that led to the patient's death.

CONCLUSION

A patient with Norwegian scabies, along with pneumonia, pulmonary tuberculosis, hypoalbuminemia, and type 1 respiratory failure was reported. Secondary infections had been developed at the time of presentation and four days after admission the patient passed away due to septic shock. Secondary infections contribute to the increment of mite growth that worsens the disease's progression. This case highlights that establishing an early diagnosis guideline of Norwegian scabies is important to be developed in order to help early diagnose and to prevent the mortality.

PATIENT CONSENT

Written informed consent provided by the family member of the patient.

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DISCLOSURE OF CONFLICTS OF INTEREST

There is no conflict of interest.

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None.

AUTHOR CONTRIBUTION

NAR and AC contributed in clinical assessments of the patients, data collection, writing the manuscript. SWM supervised the clinical assessments and data collection and revised the manuscript. All authors have read the final manuscript.

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