



Case report

A rare case study of pyoderma gangrenosum with dilated cardiomyopathy and multiple cerebral infarct in malnourished children

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ABSTRACT

Pyoderma gangrenosum (PG) is an extremely rare case of sterile necrotic ulcerative disease associated with malnutrition as a predisposition factor. It is unclear, though, whether dilated cardiomyopathy, which affects blood flow and results in stenosis in the arteries, could play a role as an etiology. In this study, a case of pyoderma gangrenosum in a 10-year-old boy complicated by dilated cardiomyopathy, a previous history of cerebrovascular disease, and a malnourished condition were reported. The patient was reported to have exudative necrotic lesions in both legs. Lesions began as small, multiple, itchy lesions on both legs, which later became blisters and scuffed, and progressed into painful, peeled-off lesions with pus, bleeding, redness around lesions, and maggots within a month. A high fever was an accompanying symptom. The multidisciplinary team was involved to provide a comprehensive treatment for this patient. Antibiotics and necrotomy debridement were performed several times. Anticoagulant treatment was indicated as the coagulation markers were increased and echocardiography suggested thrombus in the left ventricle. The underlying condition that increases the risk of pyoderma gangrenosum should be corrected. The patient was discharged after a clinical improvement, although the continuation of outpatient monitoring was required. Our report suggests that a chronic condition of dilated cardiomyopathy that affects normal blood flow leads to malnutrition, the formation of thrombus, and stenosis of a peripheral artery, all of which contributed to pyoderma gangrenosum. Therefore, early surgical treatment, antibiotic administration, and anticoagulant treatment were recommended.

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1. Introduction

Pyoderma gangrenosum (PG) is an extremely rare case of chronic non-infectious dermatoses characterized by painful and necrotic ulceration with undermined border. Based on previous reports and retrospective case series, the global incidence of PG was approximately 3–10 cases per million population per year [1–4]. However, most investigations reported adult cases because the peak of the incidence was between 20 and 50 years of age, while 3–4 % were recorded among children. The most recent systematic review among children showed that the average age of all included patients of the study was 9.5 years with a standard deviation of 6.3 years [5]. It was also discovered that the diagnosis of PG in children is often delayed by an average of 2 months due to low prevalence and difficulty to exclude the differential diagnosis of other ulcerative diseases [5].

Severe malnutrition is common in developing countries and data indicates that malnourished children are at higher risk of dying once infected [6]. Additionally, dilated cardiomyopathy (DCM) is a phenotype of cardiomyopathy caused by idiopathic, familial, genetic mutations (primary) and inflammatory, congenital heart disease, oncologic, toxin-mediated, systemic, and nutritional disorders including protein-energy malnutrition (secondary) [7].

2. Case report

A 10-year-old boy came to the pediatric emergency department with a wound on both legs as the chief complaint. Three months prior to admission, the patient had swelling all over the body, all over the body, from the face, trunk, legs, hands, and small multiple itchy lesions on both legs, which later became blister and scuffed. Within a month of admission, the painful lesions were peeled off, with pus, bleeding, redness around the lesions, and maggots accompanied by high fever. A history of cardiomyopathy and four episodes of transient ischemic attack were also observed.

The patient was moderately ill and fully conscious, with a heart and respiratory rate of 148 beats per minute and 24 breaths per minute, a temperature of 36.8 °C, 25 kg weight, 132 cm height, and 16 cm arm circumference. The height and body mass index for age were -1.05 and -1.51 SD, respectively, classified as severe wasting. At the right lower extremity as shown in Fig. 1a, there were multiple ulcers with the smallest and largest sizes of 1 × 0.5 × 0.3 cm and 10 × 5x0.5 cm, respectively, as well as granulation, slough,

a. before

b. after treatment



Fig. 1. Extremity photo of patient.

Table 1
Progressivity of clinical manifestation.

Time Course	Feb-2021	Mar-2021	Apr-2021	May-2021
Clinical Manifestation	- Swelling all over the body - Small multiple itchy lesions	- Blister and scuffed lesions	- The painful lesions were peeled off (pus (+), bleeding (+), redness around the lesions (+), maggots (+) - High fever	Right lower extremity: Multiple ulcers sized of 1 × 0.5 × 0.3 cm and 10 × 5x0.5 cm, granulation (+), slough (+), erythema (+), pitting edema (+) at 1/3 distal leg, pus (+), exudate (+), necrotic tissue (+), the oxygen saturation of the 2nd, 4th, 5th fingers decreased to 78 %, 35 %, and 89 %. Left lower extremity: Multiple ulcers sized of 2 × 0.5 × 0.3 cm and 20 × 8x0.5 cm, granulation (+), slough (+), erythema (+), pitting edema (+) at 1/3 distal leg, pus (+), exudate (+), necrotic tissue (+), the oxygen saturation of the 1st, 2nd, 5th fingers decreased to 84 %, 91 %, and 92 %.

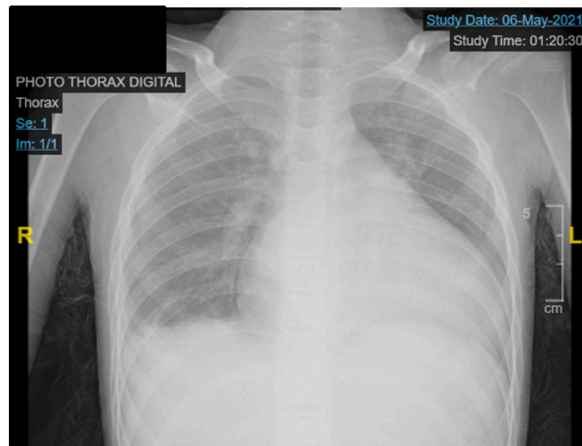
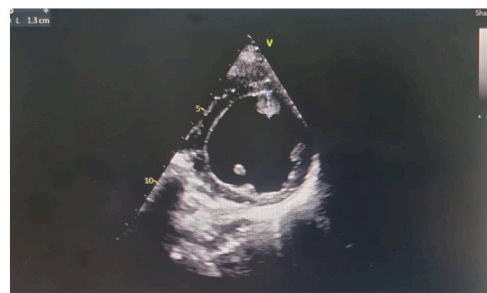
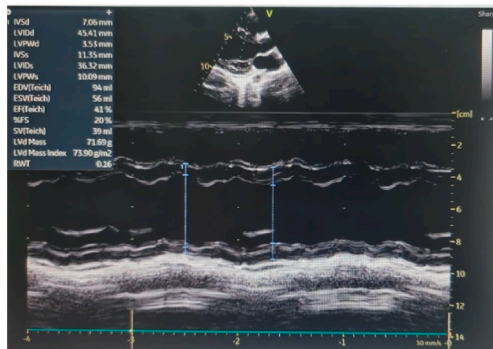


Fig. 2. Chest X-Ray of the patient.



(a)



(b)



(c)

Fig. 3. Echocardiography.

erythema, and pitting edema at level 1/3 of the distal leg, as well as pus, exudate, and necrotic tissue. The oxygen saturation of the second, fourth, and fifth fingers of the right lower extremity decreased to 78 %, 35 %, and 89 %, respectively. In the left lower extremity, multiple ulcers were found with the smallest and largest sizes of $2 \times 0.5 \times 0.3$ cm and $20 \times 8 \times 0.5$ cm, respectively, as well as granulation, slough, erythema, and pitting edema at level 1/3 of the distal leg, as well as pus, exudate, and necrotic tissue. Furthermore, the oxygen saturation of the first, second, and fifth fingers of the left lower extremity also decreased to 84 %, 91 %, and 92 %, respectively. The pulsation and sensory function for both lower extremities were within normal limits and the motoric function was limited due to pain. The progression of the illness is shown in Table 1.

The laboratory test showed 10.0 d/dL Hb, 30.6 % Ht, total leukocyte of $11.23 \times 10^3/\text{mm}^3$, 0 % basophil, 1 % eosinophil, 0 % band neutrophil, 52 % segmented neutrophil, 35 % lymphocyte, and 12 % monocyte. While prothrombin, aPTT, and INR times were 11.6, 21.0, and 1.06 seconds, respectively. The total protein level was 5.4 g/dL, 1.59 g/dL albumin, 3.8 g/dL globulin, and an albumin/



(a) Right lower leg



(b) Left lower leg

Fig. 4. X-Ray of right and left lower legs.

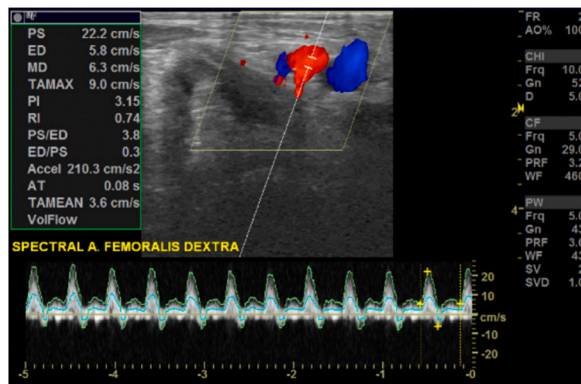


Fig. 5. USG colored Doppler of lower extremity.

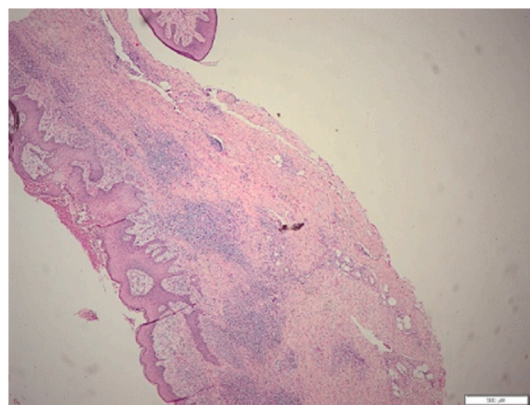
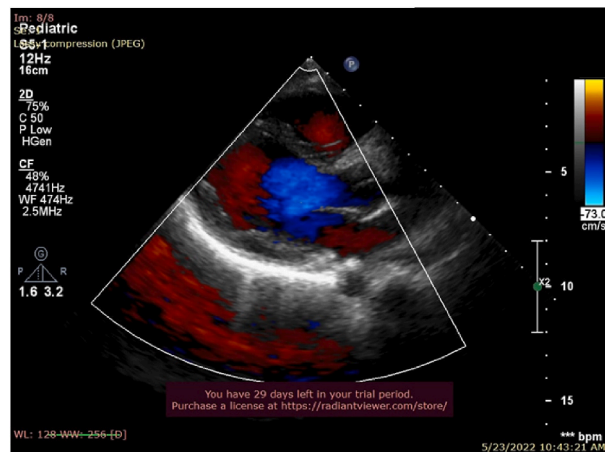


Fig. 6. Biopsy of lesion.

globulin ratio of 0.42. Furthermore, CRP was 7.11 mg/L, 161.0 mg/dL fibrinogen, 3.95 mg/L d-Dimer, 29 U/L CK-MB 29, and 0.02 ng/mL troponin I. The thoracic X-ray with anteroposterior projection (AP) showed cardiomegaly with a 78 % cardiothoracic ratio as indicated in Fig. 2. Echocardiography showed that all chambers were dilated with a 32 % reduced ejection fraction and thrombus measuring 14 × 27 mm in the left ventricle, as well as thrombus in the right ventricle as shown in Fig. 3. Foot X-ray as illustrated in Fig. 4, showed soft tissue irregularity, suggestive right foot ulcer, and lytic sclerotic lesion at the left calcaneus bone of the left foot. Furthermore, USG Color Doppler in Fig. 5 revealed mild to moderate stenosis at the level of the bilateral dorsalis pedis artery.

Angiography and MRI revealed thrombosis in the left sinus transversus, multiple infarctions in the left juxtacortical and fronto-temporal lobes, and a chronic infarction in the right subcortical parietal lobes and left cerebellum caused by stenosis of the right cerebral media artery. The patient suggested repeating the MRI examination and passing through the test with venography. The results showed multiple small ovals and linear white matter images, also in bilateral juxtacortical frontoparietal areas suggestive of multiple sclerosis. However, these discoveries still require further examination. The biopsy of the lesion indicated the possibility of PG as presented in Fig. 6. Pus culture showed the growth of *Pseudomonas aeruginosa* and *Citrobacter freundii* which are resistant to ampicillin, ampicillin/sulbactam, and cefazolin.

The patient was diagnosed with PG in bilateral legs and feet, dilated cardiomyopathy, hypoalbuminemia, and severe malnutrition. A combination of cefotaxime and metronidazole, with analgesic, furosemide, and albumin was administered along with necrotomy debridement within 24 hours after admission was discovered. On the fifth day, prothrombin, INR, and aPTT time were increased to 67.8, 7.39, and 48.5, respectively, the coagulation disorder was marked, and warfarin was administered. Furthermore, the patient experienced necrotomy debridement three times and had a positive outcome as illustrated in Fig. 1b. One month after the last debridement, there was an improved thrombus at the left ventricle in the echocardiography examination and coagulation marker, with a clinically improved skin lesion. Nutrition status also increased with weight gain to 28 kg and the patient was discharged from the hospital but continued with outpatient treatment. After one year, echocardiography evaluation was carried out twice, and showed



(a)



(b)

Fig. 7. Echocardiography evaluation.

Table 2
Supporting examination results.

Day of Hospitalization	Day 1	Day 2	Day 5	Day 6	Day 10	Day 11
Laboratory Result	Hb: 10.0 d/dL Ht: 30.6 % Leu: $11.23 \times 10^3/\text{mm}^3$ Basophil: 0 % Eosinophil: 1 % Band neutrophil: 0 % Segmented neutrophil: 52 % Lymphocyte: 35 % Monocyte: 12 % PT: 11.6 s aPTT: 21.0 s INR: 1.06 s Total protein: 5.4 g/dL Albumin: 1.59 Globulin: 3.8 Albumin/globulin ratio: 0.42 CRP: 7.11 mg/L Fibrinogen: 161.0 mg/dL d-Dimer: 3.95 mg/L CK-MB: 29 U/L Troponin I: 0.02 ng/mL	–	Hb: 10.7 d/dL Ht: 30.9 % Leu: $12.87 \times 10^3/\text{mm}^3$ Basophil: 0 % Eosinophil: 1 % Band neutrophil: 1 % Segmented neutrophil: 76 % Lymphocyte: 16 % Monocyte: 6 % PT: 17.7 s aPTT: 31.8 s INR: 1.26 s Albumin: 2.17 CRP: 4.33 mg/L d-Dimer: 2.75 mg/L	PT: 67.8 s aPTT: 48.5 s INR: 7.39 s	PT: 11.7 s aPTT: 26.3 s INR: 1.07 s	Hb: 9.5 d/dL Ht: 28.4 % Leu: $12.32 \times 10^3/\text{mm}^3$ Tr: $517 \times 10^3/\text{uL}$ Basophil: 0 % Eosinophil: 1 % Band neutrophil: 0 % Segmented neutrophil: 72 % Lymphocyte: 16 % Monocyte: 11 % Total protein: 6 g/dL Albumin: 2.29 Globulin: 3.7 Albumin/globulin ratio: 0.62 CRP: 2.29 mg/L
Blood Culture	–	Growth of bacteria was not identified	–	–	–	–
Pus Culture	–	Growth of <i>Serratia marcescens</i> and <i>Citrobacter freundii</i> that are resistant to ampicillin, ampicillin/sulbactam, and cefazolin	Growth of <i>Pseudomonas aeruginosa</i> and <i>Citrobacter freundii</i> that are resistant to ampicillin, ampicillin/sulbactam, and cefazolin	–	–	–
Gram Staining	Gram negative rod: 4+ Gram positive coccus: 2+	Gram negative rod: 3+ Gram positive coccus: 2+	Gram negative rod: 1+ Gram positive coccus: 1+	–	–	–
Echocardiography	–	Dilatation of all chambers with 32 % reduced ejection fraction and thrombus in the left ventricle	–	–	–	–
Doppler USG	Mild to moderate stenosis at the level of bilateral dorsalis pedis artery	–	–	–	–	–
Thorax X-Ray	Cardiomegaly (78 % CTR)	–	–	–	–	–
Pedis X-Ray	Soft tissue irregularity, suggestive right foot ulcer, and lytic sclerotic lesion at the left calcaneus bone of the left foot	–	–	–	–	–
Histopathology	–	–	–	–	–	–
Angiography MRI	–	–	–	–	–	–
Day of Hospitalization	Day 14	Day 16	Day 19	Day 20	Day 22	Day 26
Laboratory Result	Hb: 8.4 d/dL Ht: 25.8 % Leu: $12.69 \times 10^3/\text{mm}^3$ Tr: $707 \times 10^3/\text{uL}$ Basophil: 0 % Eosinophil: 3 % Band neutrophil: 0 % Segmented neutrophil: 58 % Lymphocyte:	–	–	–	Hb: 12.1 d/dL Ht: 36.5 % Leu: $10.05 \times 10^3/\text{mm}^3$ Tr: $782 \times 10^3/\text{uL}$ Basophil: 0 % Eosinophil: 4 % Band neutrophil: 0 % Segmented neutrophil: 55 % Lymphocyte: 27 % Monocyte: 14 % PT: 13.1 s aPTT: 30.2 s INR: 0.92 s	Hb: 11.8 d/dL Ht: 24.8 % Leu: $7.84 \times 10^3/\text{mm}^3$ Tr: $805 \times 10^3/\text{uL}$ Basophil: 0 % Eosinophil: 3 % Band neutrophil: 0 % Segmented neutrophil: 51 % Lymphocyte: 37 % Monocyte: 9 % PT: 13.3 s aPTT: 28.3 s INR: 0.93 s CRP: 0.48

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Table 2 (continued)

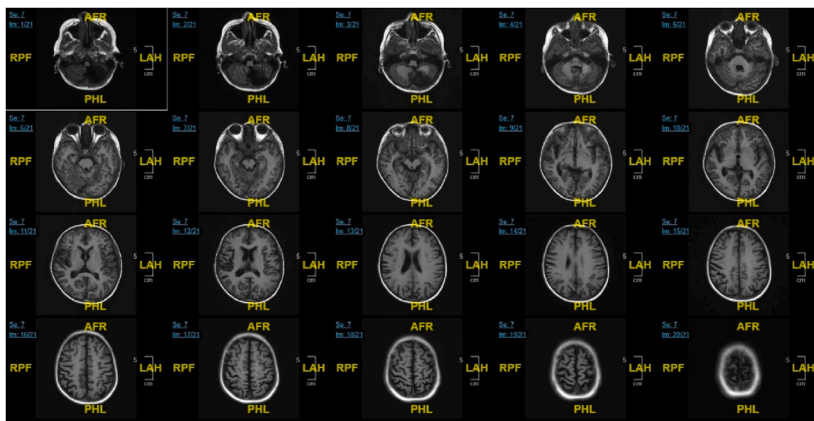
Day of Hospitalization	Day 14	Day 16	Day 19	Day 20	Day 22	Day 26
	29 % Monocyte: 10 % PT: 11.1 s aPTT: 21.6 s INR: 1.01 s Total protein: 5.8 g/dL Albumin: 2.51 Globulin: 3.3 Albumin/globulin ratio: 0.76					
Blood Culture	–	–	–	–	Growth of bacteria was not identified	–
Pus Culture	–	Growth of <i>Pseudomonas aeruginosa</i> that are resistant to cefazolin, ceftazidime, tigecycline, aztreonam	Growth of <i>Pseudomonas aeruginosa</i> that are resistant to cefazolin, ceftazidime, tigecycline, aztreonam	–	–	–
Gram Staining	–	Negative	–	–	–	–
Echocardiography	–	–	–	–	Suggestive to dilated cardiomyopathy, thrombus at left ventricle and non compaction of left ventricle	–
Doppler USG	–	–	–	–	–	–
Thorax X-Ray	–	–	–	–	–	–
Pedis X-Ray	–	–	–	–	–	–
Histopathology	–	–	–	Possibility of pyoderma gangrenosum	–	–
Angiography MRI	–	–	–	–	–	Thrombosis on left sinus transversus, multiple infarctions at left juxtacortical, and frontotemporal lobes chronic infarct in right subcortical parietal lobes and left cerebellum (caused by stenosis of the right cerebral media artery)

normal cardiac function with improved ejection fraction and no thrombus, as presented in Fig. 7.

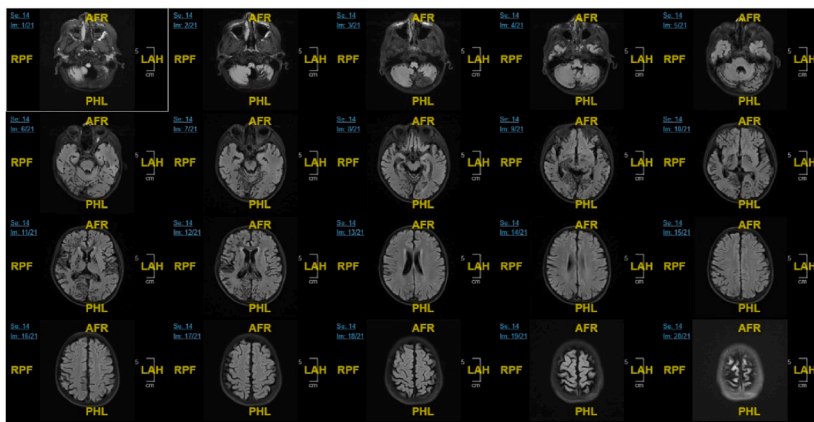
3. Discussion

Pyoderma Gangrenosum is a rare case of reactive, non-infectious, inflammatory neutrophilic dermatosis characterized by the rapid progression of painful erythematous pustules or nodules into hemorrhagic necrotic plaques, or ulcers with defined violaceous borders [8–10]. Although the etiology of PG is not clearly understood, it can be idiopathic or occur with systemic diseases such as inflammatory bowel disease (IBD), rheumatoid arthritis (RA), leukemia, IgA gammopathy, immunodeficiency states or as part of PAPA syndromes (pyogenic arthritis, PG, and acne) [11]. In this study, a rare case of PG without a history of an associated underlying disease was reported. A systematic review of pediatric PG showed that more than half of the cases occurred without presenting an underlying systemic disease [5]. As a part of a heterogeneous group of neutrophilic dermatoses, PG is characterized by the existence of sterile neutrophilic infiltrates. Neutrophils could be infiltrated into dermis and manifest as a typical cutaneous lesion. Meanwhile, aseptic neutrophil foci can deposit anywhere in the body and act as extracutaneous manifestations, which most commonly involved pulmonary and ocular organs [11,12].

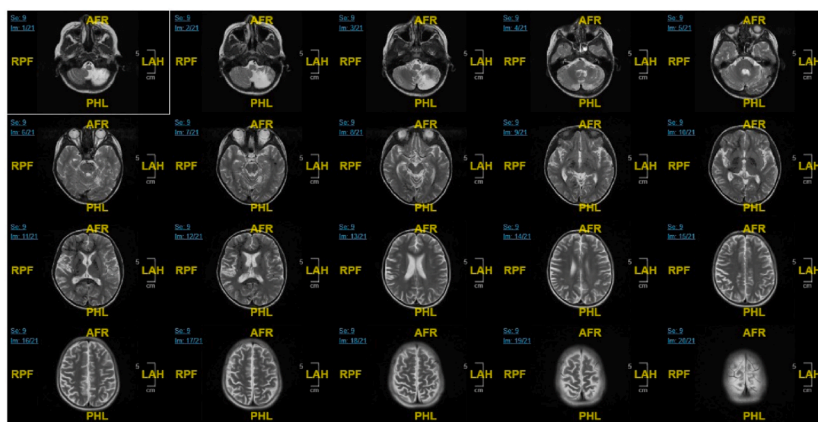
Pathogenesis of PG, specifically in children, was not clearly defined but it might be complex and multifactorial. Investigations showed that neutrophil dysfunction, immune dysregulation or abnormalities of inflammation, and genetic susceptibility play a critical role in the basic PG pathogenesis induced by minor trauma. Proinflammatory and neutrophil chemotactic factors were also found to be



(a)



(b)



(c)

Fig. 8. Second examination of head MRI.

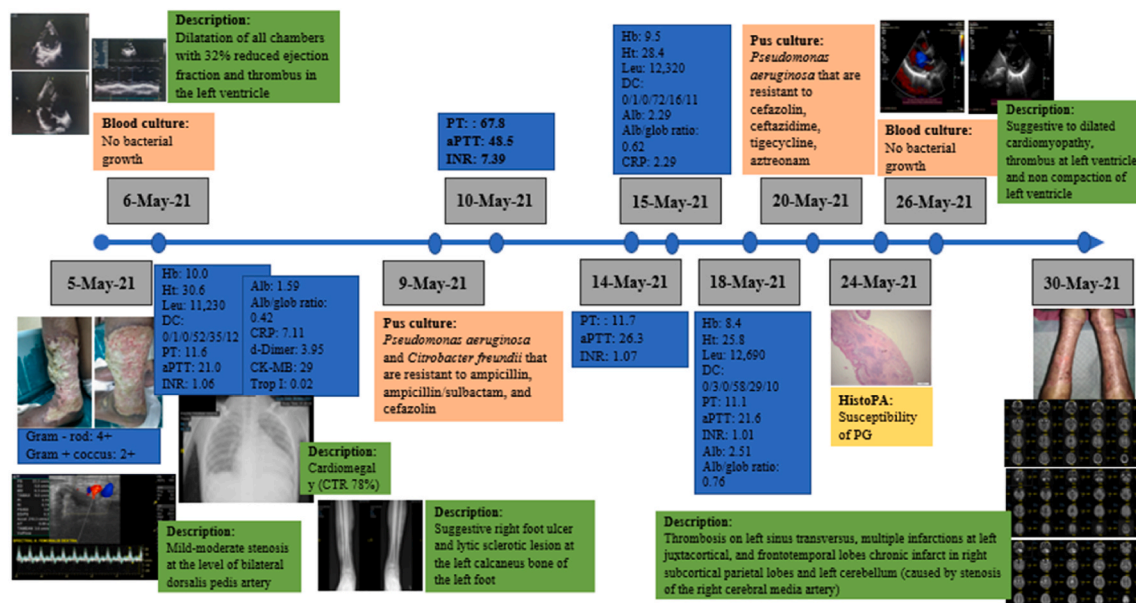


Fig. 9. Course of illness.

upregulated within skin lesions, which overexpressed cytokines and other inflammatory mediators such as interleukin (IL)-8, IL-17, tumor necrosis factor (TNF)-, chemokines 1, 2, 3, and 16, and matrix metalloproteinase (MMP) 2 and 9. In addition, the increase of IL-1 β and its receptor suggested the activation of inflammasomes revealing the autoinflammatory process. As the inflammation progressed, the central epidermal layer died and exposed an underlying ulcer. Highly exudative ulcer formed due to a dense neutrophilic infiltrate. Inflammatory cells embedded in deeper tissue showed reddish-violaceous border as a hallmark of PG. Zone of erythema, peripheral to the lesion border, contains perivascular lymphocytes which are found in histological examination [8–11].

Schoch et al. conducted a retrospective review of 13 pediatric patients which included unequivocal PG [13]. The results showed that all patients manifested an ulcerative subtype of PG that is concordantly found in this study. Ulcerative subtype was the most common or classical in all age groups and the lesions might have an initial presentation as pustules, and become ulcerated with severe necrosis at a later stage [5,8]. In this study, ulcerative lesions on both legs are presented as the most commonly affected body sites due to potential trauma [10]. A similar report by Schoch et al. found that the lower extremities represented 77 % of the area that is most commonly affected by PG lesions [13]. Kechichian et al. also discovered that the lower extremities were frequently affected in a localized state following disseminated ulcerative lesions [5].

Diagnosis of PG is still referred to as a “diagnosis of exclusion” because there are still no validated or established clinical or pathological criteria for diagnosing PG. Su et al. proposed the criteria to fulfill two major and minor measures for diagnosing PG that were widely used in previous investigations [14]. Major criteria consist of: (1) Rapid progression of a painful, necrolytic cutaneous ulcer with an irregular, violaceous, and undermined border (margin expansion of 1–2 cm per day, or a 50 % increase in ulcer size within 1 month), (2) Other causes of cutaneous ulceration have been excluded. The minor criteria are as follows: (1) a history suggestive of pathergy (ulcer development at sites of minor cutaneous trauma) or a clinical finding of cribriform scarring; (2) systemic diseases associated with PG (IBD, arthritis, IgA gammopathy, underlying malignancy); (3) histopathologic findings including sterile dermal neutrophilia, mixed inflammation, and lymphocytic vasculitis; and (4) treatment response (rapid response to systemic steroid treatment; generally responds to a dosage of 1 mg/kg to 2 mg/kg per day, with a 50 % decrease in size within 1 month).

New diagnostic criteria had been established by an international panel of experts using a two-step approach to improve the tools used for minimizing diagnostic delays and misdiagnoses [10]. The criteria is categorized as a major and four minor based on indicative biopsy result as well as histology, history, clinical examination, and response to therapy, respectively. Histological features from the biopsy sample were depicted as predominantly neutrophilic infiltrates dermal edema, suppurative inflammation, sometimes sterile abscess formation, and perivascular or periadnexal lymphocytes.

Cultures and infection screening, routine blood chemistry, urinalysis, imaging, and biopsy should all be included in the supporting examination to rule out other possibilities. The coagulation screen was used in the laboratory to look for thrombotic causes of ulceration. Our study results showed abnormal coagulation parameters, as shown in Table 2. The activation of coagulation can be triggered by increased tissue factors due to inflammatory responses. According to a previous report, when the PT ratio is < 1.2 and fibrin degradation products < 10 mg/L, diseases other than infection-associated coagulation disorders should be considered [15]. Meanwhile, this study showed a low level of albumin which might be affected by the patient’s malnutrition status.

Thrombi and infection for the corresponding extremity was seen in this patient as it was overlapping condition generating the lesions. Doppler USG examination during admission showed mild-to-moderate stenosis at bilateral dorsalis pedis artery. Despite thrombus was not depicted, infectious process proved by the result of repeated pus culture from the lesions which revealed the growth

of *Pseudomonas aeruginosa* that are resistant to several antibiotics. The result was considered to determine the correct regimen of antibiotic use for treating this patient.

The management of PG in children was not fully different from adults and should begin crucially with early recognition as well as treatment of underlying systemic disease. However, the diagnosis is often delayed because it excludes other differential diagnoses with limited definitive signs and symptoms, as reported in this study. Pain control, local wound care, topical and systemic drug therapy were required as modalities of PG treatment [4,8,10,16]. Systemic drug therapy is indicated to treat more extensive lesions with high-dose corticosteroids (prednisone 1–2 mg/kg/d) because of the underlying inflammatory process and rapidity of response in 2–3 days [3]. Some ulcers required prolonged treatments to fully resolve of skin lesions, thereby allowing other immunomodulator treatments to be used. Maronese et al. showed that additional use of systemic immunomodulators has been associated with better healing of PG lesions [17]. Marzano et al. obtained a different result, where a patient with Klinefelter's syndrome who developed bullous PG resistant to immunosuppressive regimens died following the occurrence of septicemia [18]. Currently, biological agents can be used as an alternative therapy in the case of PG resistance to systemic corticosteroid or immunomodulator therapy [17]. This study revealed the growth of *Pseudomonas aeruginosa* and *Citrobacter freundii* which are resistant to ampicillin, ampicillin/sulbactam, and cefazolin from ulcerative pus culture. It was discovered that there is bacterial colonization in delayed treatment of ulcerative PG and inadequate wound care [9]. Therefore, the patient was in malnourished with an immunosuppressive condition to impede PG wound healing and also received antibiotics as well as necrotomy debridement to eradicate bacterial infection.

Dilated cardiomyopathy (DCM) is characterized by LV dilation and systolic dysfunction as identified in this patient. Several studies revealed that intracardiac thrombi could be a complication of DCM [19,20]. Factors implicated in the formation of thrombi in DCM include low-velocity swirling of blood, abnormal endocardial surfaces, atrial fibrillation, and a hypercoagulable state [21]. Thrombi complicate between 14 % and 23 % of DCM among children [20,22,23]. The most common site for thrombi in DCM is the apex where blood flow is generally slower [24]. Falk et al. noted that thrombosis was significantly more common in patients with a fractional shortening of 10 % or less (12 of 15 patients) than in those with a fractional shortening of 11 % or more (three of 10 patients; $P < 0.02$) [23]. The left ventricular thrombi can lead to decrease of LV ejection fraction and embolization of cerebral blood vessel [25]. McCrindle et al. also found left ventricular ejection fraction (LVEF) to be significantly low in dilated cardiomyopathy cases with intracardiac thrombus (mean LVEF $21 \pm 9\%$) as compared to the other group (mean LVEF $28 \pm 15\%$) [26]. The risk for ICT and systemic thromboembolism increases in case of a LVEF $<20\%$ [20,23,27]. This study suggested that chronic condition of DCM affecting circulatory flow led to malnourished condition, the formation of thrombi, and stenosis of a peripheral artery, all of which contributed to PG.

In this case, we found some interesting things in the head MRI result that showed thrombosis and chronic infarct. However, in the second examination of the head MRI, the result showed the patient had multiple sclerosis which shown in Fig. 8. This could be two different things between multiple sclerosis and PG or maybe there are some correlations that need to be studied more for future studies. Fig. 9 showed the course of his illness.

4. Conclusion

To the best of our knowledge, this case emphasizes the need for physicians' awareness of PG, especially in malnourished children with a cardiac complication that could affect the blood and cause peripheral artery stenosis, in combination with the risk of infection and coagulation disorder. Early surgical intervention and antibiotics are crucial when PG is diagnosed. Furthermore, the underlying condition that increases the risk of PG should be corrected.

Declarations

Author contribution statement

All authors listed have significantly contributed to the investigation, development and writing of this article.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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