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Case Report

^{18}F-FDG PET/CT in polyarteritis nodosa with multisystem involvement: utility in defining disease extent

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Abstract: Polyarteritis nodosa (PAN) is a rare systemic necrotizing vasculitis. It affects the medium-sized arteries. It attacks many parts of the body in very different ways. Therefore, it is difficult to diagnose and stage. ^{18}F-fluorodeoxyglucose (^{18}F-FDG) PET/CT combines anatomic and metabolic activity. This can show the complete extent of the disease in PAN very well. We report a 35-year-old man with fever, generalized myalgia, black stools and dry gangrene at the tip of his fingers. Laboratory tests revealed high C-reactive protein and high erythrocyte sedimentation rate. However, the anti-neutrophil cytoplasmic antibodies (ANCA) were negative. ^{18}F-FDG PET/CT identifies multisystem lesions. The doctor thought that these lesions were there, but they could not be found by regular scanning. These lesions include high activity of many muscles, more absorption of the middle artery of the calf, local absorption of the skin between the toes, and absorption of the stomach wall. Tissue samples from many places confirmed necrotizing arteritis. After treatment with corticosteroids and targeted antibiotics, the patient's condition improved.

Keywords: Polyarteritis nodosa, ^{18}F-FDG PET/CT, multisystem vasculitis, disease staging, diagnostic imaging

Introduction

Polyarteritis nodosa (PAN) is a rare systemic necrotizing vasculitis. It primarily affects medium-sized arteries, with occasional involvement of small arteries while sparing arterioles, capillaries, and venules [1, 2]. Clinically, PAN manifests as non-specific systemic symptoms such as fever and weight loss, which may involve the nervous system, skin, kidneys, gastrointestinal tract, etc. [3]. Diagnosis is still difficult. There are no specific blood markers. PAN resembles other inflammatory diseases.

^{18}F-FDG PET/CT can detect glucose metabolism abnormalities in inflammatory tissues. This is a key sign of active vasculitis. Therefore, this kind of scanning has become an important tool for evaluating systemic inflammatory diseases [4]. Severe PAN with multi-system involvement and hematological abnormalities is uncommon in young adults under 40 years old. These young patients make up only 8.7% of all PAN cases. And fewer than 5% of all PAN patients present with concurrent myopathy, pure red cell aplasia, and sepsis together [5]. Here, we report an unusual PAN case with multi-system involvement.

Case presentation

A 35-year-old man significant past medical history presented with a 2-week history of swelling and pain in his left jaw and right lower leg. These symptoms progressively worsened over the subsequent week. He also developed fever and melena. Physical examination revealed a tender left submandibular lymph node. It was about 1 cm wide. He had painful swelling in his right upper arm, buttock, and calf. He had dry gangrene of the skin between his toes on both feet. He also had skin discoloration on the tip of his nose. The doctor noted limb muscle wasting and weaker tendon reflexes. His muscle strength was 4/5 in all arms and legs. The right side was weaker than the left side.

Laboratory tests revealed markedly elevated C-reactive protein and erythrocyte sedimentation rate. Both anti-neutrophil cytoplasmic antibodies (ANCA) and antinuclear antibodies were negative. Microbial testing identified presence of penicillin-resistant epidermal staphylococcus in the wound fluid. It was also found that the level of Epstein-Barr virus DNA was 1.16×10^4 copies/mL. But the blood culture result is still negative. Blood tests confirmed secondary pure red cell aplastic anemia, accompanied by severe anemia and hypoproteinemia. Electromyography demonstrated peripheral neurogenic injury, predominantly affecting the lower extremities.

^{18}F-FDG PET/CT was performed for comprehensive disease staging. Coronal maximum intensity projection (MIP) images showed diffuse skeletal muscle hypermetabolism, most prominent in the extremities and right iliopsoas muscle (SUVmax 3.14-9.19) (**Figure 1A**). Linear radiotracer uptake along medium-sized vessels, forming the characteristic "vascular tree" pattern, had been described in most reported cases [6-8]. The "leopard sign", defined by multiple focal hotspots in skin and muscles, had also been documented [9]. In this patient, however, the FDG distribution pattern suggested predominant small-vessel rather than classic medium-vessel involve-

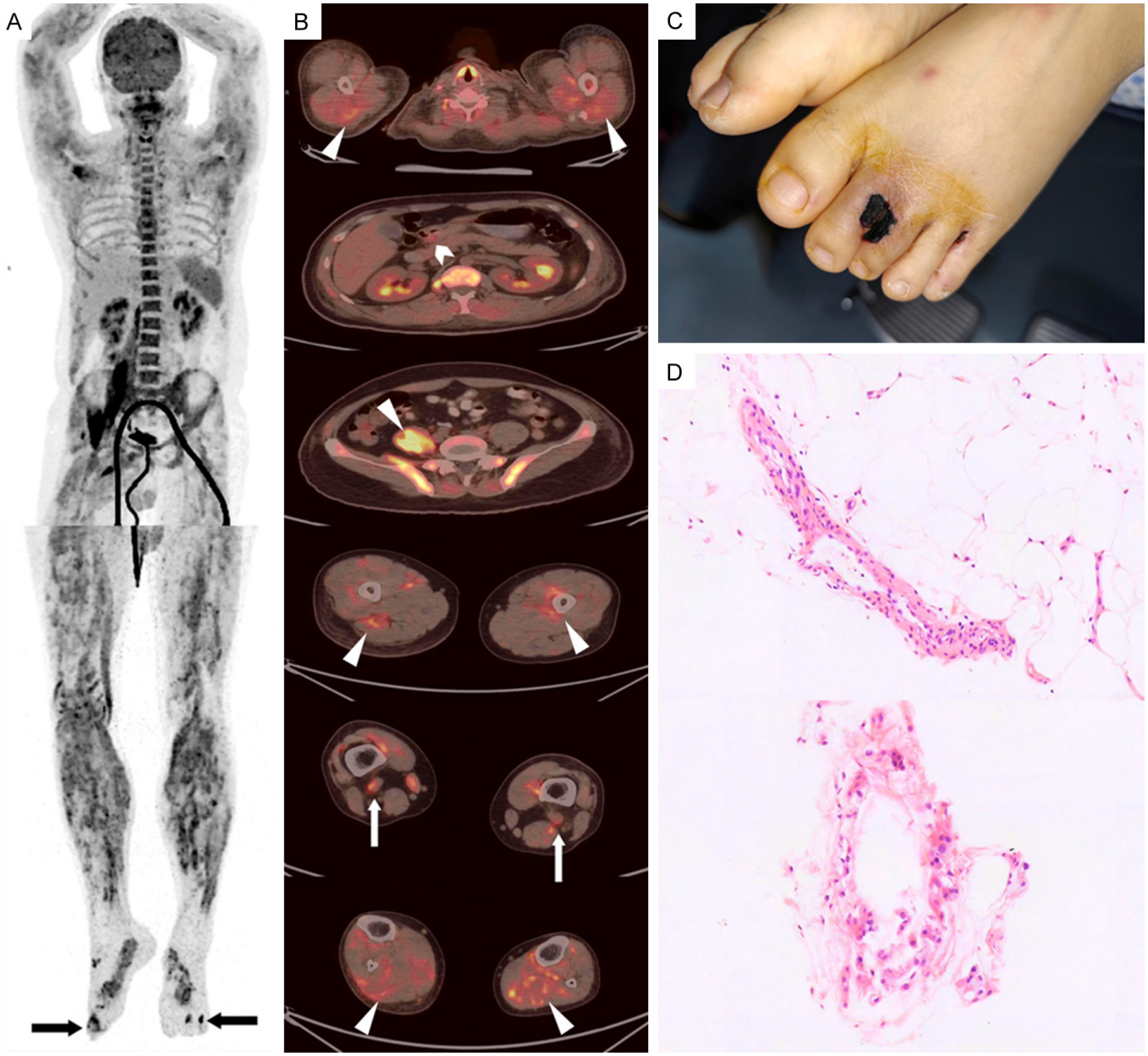

Figure 1. ^{18}F-FDG PET/CT imaging, a clinical photo, and histopathological features. A. MIP shows widespread high activity in muscles. This is most notable in the arms, legs, and right iliopsoas muscle. B. Fused axial PET/CT image shows a "dirty muscle sign". There is activity in the connective tissue inside and between muscles (arrowheads). There is more tracer along the popliteal artery (white arrows). There is focal activity in the toe web skin (black arrow). And there is activity in the stomach wall (white V-shaped arrow). C. Clinical photograph of bilateral toe web dry gangrene, correlating with the PET/CT findings. D. Tissue slide (hematoxylin-eosin stain, ×400) shows necrotizing arteritis. There are white blood cells in the vessel wall and in the tissue around the vessel.

ment. Fused axial images revealed a pronounced "dirty muscle sign", more conspicuous than previously reported [6], with radiotracer accumulation in intra- and intermuscular connective tissues (SUVmax 9.78, right psoas major muscle). Uptake was also seen along the bilateral popliteal arteries and their branches (SUVmax 4.34), consistent with vascular wall inflammation, without large-vessel involvement or aneurysm formation. Focal hypermetabolism was noted in the bilateral toe webs (SUVmax 3.59-5.48), corresponding to clinically evident dry gangrene (**Figure 1C**), and in the gastric antral wall (SUVmax 2.57), which prompted gastroscopy and confirmed a large ulcer with inflammatory necrosis (**Figure 1B**). Reactive hypermetabolism was present in bilateral cervical and axillary lymph nodes (SUVmax 2.53-3.87), spleen (SUVmax 3.56), and bone marrow (SUVmax 5.39).

Targeted biopsies of hypermetabolic lesions in the right upper arm, left buttock, and left lower leg revealed extensive neutrophilic infiltration of small- to medium-sized vessel walls, accompanied by fibrinous exudation and necrotizing arteritis. These findings are pathognomonic for PAN [10] (**Figure 1D**). Pre-treatment magnetic resonance imaging (MRI) of the lower extremities (**Figure 2**) was obtained on day 15. Coronal and axial T2-weighted sequences showed extensive subcutaneous and muscu-

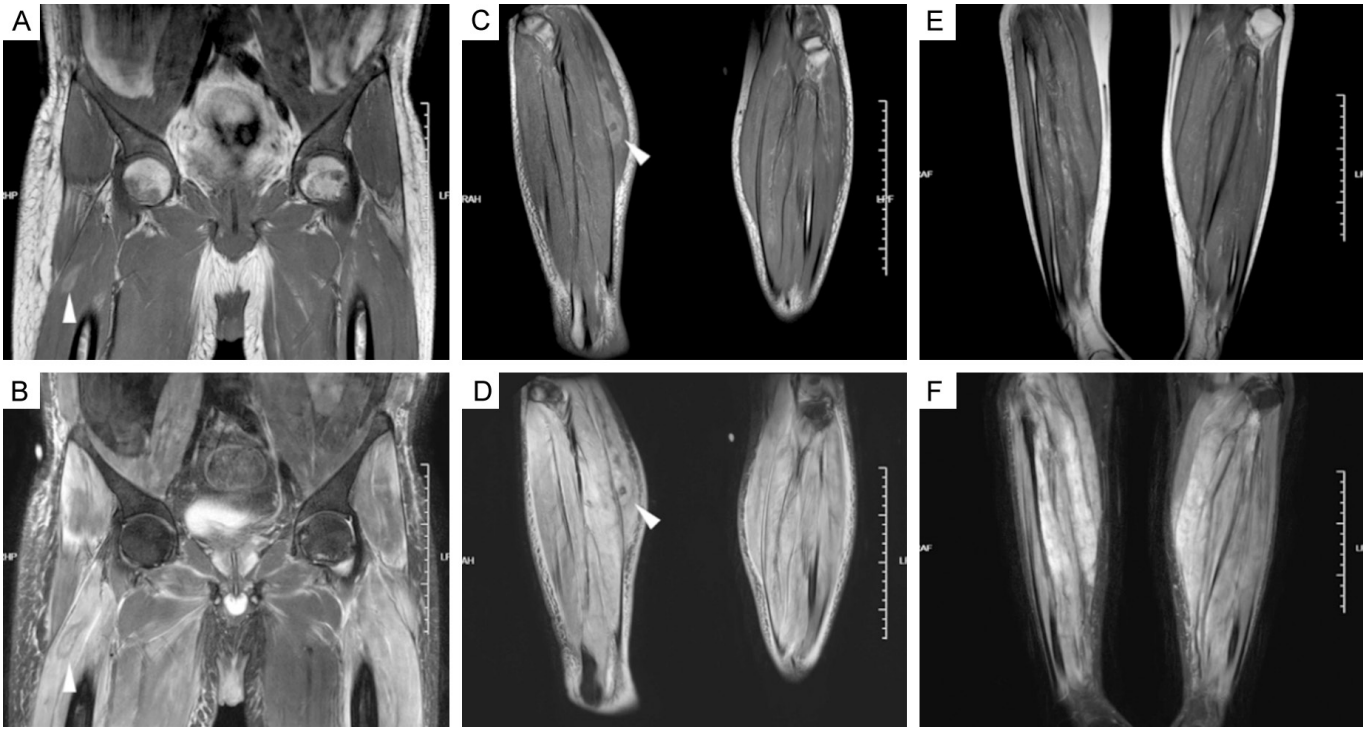

Figure 2. Pre-treatment and post-treatment lower extremity MRI scans. There was a 25-day gap between the two scans. A-D. Pre-treatment MRI shows a lot of swelling under the skin and inside the muscles. There is bleeding inside a muscle (arrowhead). This matches a burst blood vessel. E, F. After treatment, MRI showed a significant decrease in swelling and bleeding.

lar edema (**Figure 2A**, **2B**), sagittal T1-weighted images revealed intramuscular hemorrhage (**Figure 2C**), and fat-suppressed T2-weighted sequences demonstrated well-demarcated edematous foci (**Figure 2D**). Repeat MRI on day 40, 25 days later, showed marked resolution of edema, hemorrhage, and inflammatory signal abnormalities (**Figure 2E**, **2F**), confirming a favorable response to therapy.

In accordance with the 2021 American College of Rheumatology/Vasculitis Foundation (ACR/VF) guidelines for PAN management [11], the patient was diagnosed with multisystem-involved PAN complicated by sepsis, severe immunodeficiency, critical illness-related myopathy/neuropathy, and an A1-stage gastric ulcer (Forrest IIb classification). Treatment followed guideline recommendations and included broad-spectrum antimicrobial therapy and sequential corticosteroid therapy. Markedly elevated infectious markers directed initial clinical attention toward sepsis early in the course. Empirical antimicrobials were started pending cultures. Once sensitivity data returned, linezolid, vancomycin, and meropenem were used in sequence. The fever and severe muscle pain have not disappeared. Blood culture remained consistently negative. Subsequently, the wound was debrided, and the skin lesions improved. These results indicated that the staphylococcal infection was well controlled.

PET/CT was performed at this time. The special metabolic pattern described above appeared. Then a muscle biopsy confirmed necrotizing arteritis. This shifted the diagnosis toward an underlying immune system problem. A trial of low-dose IV steroids was started. This gave quick relief of symptoms. Then the dose was raised slowly. Steroids were the only immunosuppressive agent used during the hospital stay.

The patient improved significantly over 44 days. Fever and melena resolved completely. Swelling in his extremities decreased substantially. He was sent home with mild lasting weakness in his right arm and leg. After going home, he got mycophenolate mofetil at a follow-up clinic visit. This was for long-term maintenance. At the last follow-up several months later, he was still stable. He had no recurrence of vasculitis or infection.

Discussion

PAN is a rare systemic necrotic vasculitis. It primarily affects medium-sized arteries, with occasional involvement of small arteries while sparing arterioles, capillaries and venules. Its effects on many parts of the body vary from person to person [1]. This difference, coupled with the lack of specific blood markers, makes early diagnosis and accurate staging difficult. According to 2021 ACR/VF guideline, PAN typically presents with negative ANCA, which is consistent with our case [11]. In this case, the concomit penicillin-resistant epidermal staphylococcus infection made the diagnostic problem more serious. This is because bacteria can simulate or aggravate PAN-related inflammation [12]. The patient's potential vascular inflammation may cause systemic inflammatory storms

and immune system problems. Then this led to a second infection. Blind biopsy often misses the target of this disease. This is because the PAN lesion is patchy and only affects the short blood vessel segment [13]. Therefore, this increases the demand for imaging tools that can guide tissue sampling and improve biopsy results.

^{18}F-FDG PET/CT played a key role in this case. It provides both anatomical and metabolic information [14]. In this case, it can accurately locate a wide range of high muscle activity, arterial inflammation, toe fin injury and gastric wall involvement. This drawing guided targeted biopsy and confirmed necrotising arteritis. Therefore, it avoids the well-known problem of plaque PAN blind sampling [15, 16]. The patient is younger than usual (under 40 years old) and has other rare health problems. These factors increase the value of PET/CT in tracking disease transmission and guiding treatment.

In this case, the "dirty muscle signs" are very prominent. The results show that FDG accumulates diffusely in tissues inside and between muscles. This model broadens the known PAN PET/CT range. It also distinguishes PAN from the classic "vascular tree" pattern [6-8] and "leopard sign" [9]. This sign reflects a potential disease of inflammation of the whole wall of blood vessels with the accumulation of white blood cells. It conforms to the guideline description of middle and medium artery disease [11, 17]. It shows the changes of extravascular inflammation that routine angiography often misses [8].

PET/CT also links metabolic problems with actual organ damage. It guides the confirmation of the gastric extent of the gastric ulcer. It also independently confirmed the toe fin gangrene [18]. Structural scans such as CT angiography (CTA) and MRI are good at detecting aneurysms or stenosis. But they can't find mild inflammation of the vascular wall or extravascular lesions. PET/CT found that the patient had gastric wall vasculitis. But the first abdominal CT didn't find it. This finding is very important because intestinal involvement is one of the most serious problems of PAN [19]. PET/CT can check vascular diseases and extravascular diseases at the same time. Therefore, it reduces repeated scans and makes the diagnostic path simpler [20]. MRI shows that the swelling has decreased after treatment. However, the semi-quantitative parameters (SUVmax, metabolic volume) given by PET/CT can track disease activity and predict the long-term course of vascular inflammatory diseases [21]. These numbers can guide the gradual reduction of personalized steroids. The current guidelines openly say that this is necessary.

PET/CT can distinguish between vasculitis lesions and infectious lesions by observing the uptake pattern. This ability is the key to balancing immunosuppressive drugs [11] with correct antibiotics against epidermal staphylococcus [22]. Accordingly, this contributes to favorable clinical outcomes for patient. Some studies object to attributing high activity of broad muscles to reactive or infectious muscle diseases. The "dirt muscle signs" of tracer accumulation in tissues inside and between the muscles are described as a specific pattern of PAN vasculitis muscle disease [23]. This pattern is different from the proximal and strong, even uptake seen in idiopathic inflammatory muscle diseases [7].

In juvenile dermatomyositis, muscle FDG uptake tends to be symmetrical, diffuse and linear, mainly affecting the proximal limb muscles, without skin or subcutaneous involvement [7]. Its distribution is completely different from the perivascular and fascia patterns observed in this case. Septic muscle clots, in comparison, usually show up as uneven, random focal spots. They do not have the organized pattern around blood vessels seen here [7]. The muscle uptake in our patient also occurred together with line-shaped FDG buildup along the popliteal arteries and their branches. This group of findings indicated medial arterial vasculitis rather than primary muscle infection. Targeted muscle biopsy confirmed necrotic arteritis, and the entire blood vessel wall was inflamed. This provides organizational evidence.

After starting steroid and cyclophosphamide combined treatment, repeated PET/CT showed a significant decrease in muscle and blood vessel SUVmax. This matches the clinical improvement of the patient. But infectious muscle diseases are not expected to react so quickly only to immunosuppression. There are both EB virus infection and penicillin-resistant epidermal staphylococcus in the blood. But both of these situations are usually not related to this special pattern around the blood vessels and muscle cover. Reactive muscle diseases caused by serious diseases usually show milder and more dispersed absorption. It has no organized "dirty muscle" appearance [24]. This level of diagnostic accuracy is very valuable. This is because FDG absorption itself is not specific. It can be seen in infection, cancer and other inflammatory diseases. By observing PET/CT results, bacterial culture results and patients' reactions to treatment, this problem can be effectively reduced [25].

Conclusion

This case shows the multiple uses of ^{18}F-FDG PET/CT in PAN. As a whole-body imaging modality, PET/CT enables comprehensive assessment of systematic lesions across multiple organs. It guides targeted biopsy to obtain a clear diagnosis. And it links metabolic findings to patient symptoms. The "dirty muscle sign" broadens the known PET/CT imaging spectrum of PAN and underscores the necessity of identifying characteristic FDG uptake patterns. In patients with suspected ANCA-negative multisystem vasculitis, PET/CT offers a one-stop approach to optimize both diagnosis and therapeutic planning. Prospective studies are warranted to validate PET/CT as a surrogate marker of disease activity and to develop standardized interpretation criteria for PAN.

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Disclosure of conflict of interest

None.

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References

[1] Jennette JC, Falk RJ, Bacon PA, Basu N, Cid MC, Ferrario F, Flores-Suarez LF, Gross WL, Guillevin L, Hagen EC, Hoffman GS, Jayne DR, Kallenberg CG, Lamprecht P, Langford CA, Luqmani RA, Mahr AD, Matteson EL, Merkel PA, Ozen S, Pusey CD, Rasmussen N, Rees AJ, Scott DG, Specks U, Stone JH, Takahashi K and Watts RA. 2012 revised international Chapel Hill consensus conference nomenclature of vasculitides. Arthritis Rheum 2013; 65: 1-11.

[2] Lie JT. Systemic and isolated vasculitis. A rational approach to classification and pathologic diagnosis. Pathol Annu 1989; 24 Pt 1: 25-114.

[3] Pagnoux C, Seror R, Henegar C, Mahr A, Cohen P, Le Guern V, Bienvenu B, Mouthon L and Guillevin L; French Vasculitis Study Group. Clinical features and outcomes in 348 patients with polyarteritis nodosa: a systematic retrospective study of patients diagnosed between 1963 and 2005 and entered into the French Vasculitis Study Group Database. Arthritis Rheum 2010; 62: 616-626.

[4] Yukishima T, Ohmura SI, Yonezawa H, Katayama M, Otsuki Y, Miyamoto T and Ogawa N. Possible usefulness of fluorodeoxyglucose positron emission tomography in diagnosing polyarteritis nodosa: a case report and literature review. Mod Rheumatol Case Rep 2025; 9: 137-142.

[5] Eleftheriou D, Dillon MJ, Tullus K, Marks SD, Pilkington CA, Roebuck DJ, Klein NJ and Brogan PA. Systemic polyarteritis nodosa in the young: a single-center experience over thirty-two years. Arthritis Rheum 2013; 65: 2476-2485.

[6] Fagart A, Machet T, Collet G, Quéméneur T, Ben Ticha R, Verstraete M, Le Gouellec N, Demailly F and Rousselin C. Fluorodeoxyglucose positron emission tomography-computed tomography findings in a first series of 10 patients with polyarteritis nodosa. Rheumatology (Oxford) 2022; 61: 1663-1668.

[7] Triaille C, Benali S, Barsalou J, Haddad E, Kokta V, Kraus R, Lambert R, Morin MP, Samaan K, Turpin S and De Bruycker JJ. F(18)-FDG PET-CT findings in juvenile-onset polyarteritis nodosa: a first series and literature review. J Clin Med 2025; 14: 3012.

[8] Brown PJ, Huang S and Martinez F. 18 F-FDG PET/CT in the evaluation of polyarteritis nodosa. Clin Nucl Med 2024; 49: e682-e684.

[9] Shimizu M, Inoue N, Mizuta M, Ikawa Y and Yachie A. Leopard skin appearance of cutaneous polyarteritis nodosa on 18F-fluorodeoxyglucose positron emission tomography. Rheumatology (Oxford) 2016; 55: 1090.

[10] Hernández-Rodríguez J, Alba MA, Prieto-González S and Cid MC. Diagnosis and classification of polyarteritis nodosa. J Autoimmun 2014; 48-49: 84-89.

[11] Chung SA, Gorelik M, Langford CA, Maz M, Abril A, Guyatt G, Archer AM, Conn DL, Full KA, Grayson PC, Ibarra MF, Imundo LF, Kim S, Merkel PA, Rhee RL, Seo P, Stone JH, Sule S, Sundel RP, Vitobaldi OI, Warner A, Byram K, Dua AB, Husainat N, James KE, Kalot M, Lin YC, Springer JM, Turgunbaev M, Villa-Forte A, Turner AS and Mustafa RA. 2021 American college of rheumatology/vasculitis foundation guideline for the management of polyarteritis nodosa. Arthritis Care Res (Hoboken) 2021; 73: 1061-1070.

[12] Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJ, Gorbach SL, Hirschmann JV, Kaplan SL, Montoya JG and Wade JC. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the infectious diseases society of America. Clin Infect Dis 2014; 59: 147-159.

[13] Hughes LB and Bridges SL Jr. Polyarteritis nodosa and microscopic polyangiitis: etiologic and diagnostic considerations. Curr Rheumatol Rep 2002; 4: 75-82.

[14] Slart RHJA, Nienhuis PH, Glaudemans AWJM, Brouwer E, Gheysens O and van der Geest KSM. Role of (18)F-FDG PET/CT in Large Vessel Vasculitis and Polymyalgia Rheumatica. J Nucl Med 2023; 64: 515-521.

[15] Bosch P, Bond M, Dejaco C, Ponte C, Mackie SL, Falzon L, Schmidt WA and Ramiro S. Imaging in diagnosis, monitoring and outcome prediction of large vessel vasculitis: a systematic literature review and meta-analysis informing the 2023 update of the EULAR recommendations. RMD Open 2023; 9: e003379.

[16] Horomanski A and Forbess LJ. The role of imaging in diagnosis and monitoring of large vessel vasculitis. Rheum Dis Clin North Am 2023; 49: 489-504.

[17] Stanson AW, Friese JL, Johnson CM, McKusick MA, Breen JF, Sabater EA and Andrews JC. Polyarteritis nodosa: spectrum of angiographic findings. Radiographics 2001; 21: 151-159.

[18] Schmidt WA. Imaging in vasculitis. Best Pract Res Clin Rheumatol 2013; 27: 107-118.

[19] Ebert EC, Hagspiel KD, Nagar M and Schlesinger N. Gastrointestinal involvement in polyarteritis nodosa. Clin Gastroenterol Hepatol 2008; 6: 960-966.

[20] Zerizer I, Tan K, Khan S, Barwick T, Marzola MC, Rubello D and Al-Nahhas A. Role of FDG-PET and PET/CT in the diagnosis and management of vasculitis. Eur J Radiol 2010; 73: 504-509.

[21] Slart RHJA; Writing Group; Reviewer Group; Members of EANM Cardiovascular; Members of EANM Infection & Inflammation; Members of Committees, SNMMI Cardiovascular; Members of Council, PET Interest Group; Members of ASNC; EANM Committee Coordinator. FDG-PET/CT(A) imaging in large vessel vasculitis and polymyalgia rheumatica: joint procedural recommendation of the EANM, SNMMI, and the PET Interest Group (PIG), and endorsed by the ASNC. Eur J Nucl Med Mol Imaging 2018; 45: 1250-1269.

[22] Guillevin L, Pagnoux C, Seror R, Mahr A, Mouthon L and Toumelin PL; French Vasculitis Study Group (FVSG). The five-factor score revisited: assessment of prognoses of systemic necrotizing vasculitides based on the French Vasculitis Study Group (FVSG) cohort. Medicine (Baltimore) 2011; 90: 19-27.

[23] Ruffer N, Haase I, Xiong T, Prinz F, Koehler D, Kötter I and Krusche M. Temporal arteritis and vasculitic myopathy in polyarteritis nodosa. J Neurol 2025; 272: 496.

[24] Selva-O'Callaghan A, Gil-Vila A, Simó-Perdigó M, Trallero-Araguás E, Alvarado-Cárdenas M and Pinal-Fernandez I. PET scan: nuclear medicine imaging in myositis. Curr Rheumatol Rep 2019; 21: 64.

[25] Meller J, Sahlmann CO and Scheel AK. 18F-FDG PET and PET/CT in fever of unknown origin. J Nucl Med 2007; 48: 35-45.