

Characteristic Imaging Patterns of Muscle Involvement in Polyarteritis Nodosa: Case Report and Review of the Literature

Peikai Huang, Hailong Liu, Meng Zhang, Yanxia Chen, Jingzhi Ye, Jingfeng Liu, Jun Chen, Mengqiang Xiao

Department of Radiology, Zhuhai Hospital, Guangdong Hospital of Traditional Chinese Medicine, Zhuhai, Guangdong, China.

Abstract

Objective: Polyarteritis nodosa (PAN) is a rare disease with complex clinical manifestations that are difficult to diagnose. Imaging diagnoses of previously reported patients have focused on vascular manifestations. Magnetic resonance imaging (MRI) has been used to detect muscle involvement in PAN. Here, we reviewed imaging findings pertaining to muscle involvement in patients with PAN. **Methods:** Twelve articles concerning muscle involvement in PAN were published during the period 1980–2020; 21 patients, including our patient, were examined in this study. **Results:** Across the published articles, the male to female ratio was 1:1, the mean patient age was 40.76 ± 18.28 years, and there were 17 patients with calf involvement and 3 with thigh involvement. The T1WI and T2WI findings were both isointense in one patient, and the T1WI findings alone were isointense in seven patients. The T1WI findings were slightly hyperintense in five patients, and no T1WI images were available for the remaining seven patients. The T2WI signal was diffusely hyperintense in 10 patients, and “patchy villous hyperintense” in 9 patients. Among the 12 patients with enhanced images, most exhibited diffuse or cotton-like enhancement, while some showed involvement of the fascia and periosteum. The comprehensive imaging analysis of our patient included muscle and blood vessel MRI and computed tomography (CT) examinations. Our patient’s disease involved the calves and thighs, with T1WI isointensity and T2WI patch-like hyperintensity, as well as cotton-like enhancement centered on blood vessels. Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) examinations of the lower limbs showed beadlike changes in the arterial branches, and the lower leg arterial branches were obvious. Head MRA revealed stenosis and occlusion of the right middle cerebral artery. Additionally, the superior mesenteric artery was locally dilated around a tumor, with the greatest width being approximately 9 mm. Cerebral perfusion analysis indicated cerebral blood flow (CBF) was lower in the right cerebellar hemisphere. **Conclusions:** PAN should be considered in the presence of patchiness or diffuse muscle signal changes on MRI of the lower leg (or thigh), followed by vessel-centered cotton-like enhancement accompanied by fascial or periosteal enhancement. Our findings suggest that systemic examination of small and medium arteries in patients with PAN can aid early prevention and treatment.

Keywords: MRI-CT- Polyarteritis nodosa- Muscle

Asian Pac J Cancer Care, 6 (2), 209-215

Submission Date: 03/12/2021

Acceptance Date: 04/26/2021

Introduction

Polyarteritis nodosa (PAN) is uncommon in patients with systemic vasculitis, and has a low incidence of 1.6–2.4 per 1 million people worldwide [1-2]. In its early stages, PAN encompasses the majority of vasculitis manifestations. In recent years, in-depth research on vasculitis has revealed specific subtypes. Research concerning the pathogenic effect of antineutrophil

cytoplasmic antibodies (ANCA) in vasculitis has led to a distinction being drawn between ANCA-positive patients with vasculitis and patients with PAN. Furthermore, eosinophilic granulomatous polyangiitis is currently classified as ANCA-associated vasculitis, formerly known as Churg–Strauss syndrome. PAN with rheumatoid arthritis is now regarded as “rheumatoid arthritis vasculitis” [3-5].

Corresponding Author:

Dr. Mengqiang Xiao

Department of Radiology, Zhuhai Hospital, Guangdong Hospital of Traditional Chinese Medicine, 53 Jingle Road, Zhuhai City, Guangdong Province, China.

Email: 714454688@qq.com

PAN is a segmentalized, necrotizing vasculitis that primarily involves small and medium arteries, as well as arterioles. It was defined at the Chapel Hill Conference in 2012. This rare disease may present with multiple organ involvement and complicated clinical manifestations, and is difficult to diagnose early [3]. The present report summarizes the typical magnetic resonance imaging (MRI) manifestations of patients with PAN, as described in the English-language medical literature. The shin exhibits patchy-like T1WI isointensity and T2WI hyperintensity, with diffuse or vascular foci. After enhancement, the shin exhibits cotton-like enhancement with vascular foci and focal accumulation in the periosteum and fascia. In our patient, plain and contrast-enhanced muscle MRI showed typical imaging manifestations, but plain and contrast-enhanced computed tomography (CT) revealed no abnormalities. The clinical manifestations of other extramuscular organs were normal, but computed tomography angiography (CTA) and magnetic resonance angiography (MRA) findings suggested stenosis of cranial vessels or aneurysmal dilation of the mesentery, and vascular lesions precluded clinical manifestations.

Case Report

Clinical manifestations and treatment process

The patient was a 55-year-old man who had a 1-year history of bilateral nodal erythema with swelling, but no itching desquamation, fever, or discomfort. He presented to the First Affiliated Hospital of Guangzhou University of TCM and the Fifth Affiliated Hospital of Sun Yat-sen University. He was first diagnosed with nodular erythema, and treated with hydroxychloroquine, prednisone, and Chinese medicine. These treatments did obviously relieve his symptoms, and he gradually developed nodal erythema on the wrist, left elbow, and thighs. Approximately 4 months later, he developed pain in both knee joints, accompanied by pain during exertion of the bilateral thigh muscles. He also experienced muscle fatigue, swelling and pain in the left ring finger, and limited fist clenching ability in both hands. However, there was no morning stiffness or skin tightness. Three months later, he developed nonspecific fever, primarily at night, with a maximum temperature of 39.0°C. This temperature returned to normal in the mornings. Based on the absence of other symptoms (e.g., oral and genital ulcers, sore throat, cough and expectoration, and abdominal pain), the patient was diagnosed with PAN. He was treated with prednisone acetate (30 mg/day) and methotrexate (12.5 mg/week). These treatments reduced the intermittent fever.

Laboratory examination

The initial laboratory findings were as follows: monocytes, 14.34%; absolute monocyte count, $1.19 \times 10^9/L$; PLT, $391.00 \times 10^9/L$; PT, 13.7 s; INR, 1.22 ↑; fibrinogen, 6.78 g/L; activated partial thromboplastin time, 33.8 ↑s; ESR, 44 mm/h; complement C3, 1.72 g/L; and high-sensitivity C-reactive protein, 107.73 mg/L. The patient had negative findings for cytoplasmic ANCA, formaldehyde-resistant perinuclear ANCA

(pANCA), formaldehyde-sensitive pANCA, glomerular basement membrane antibody, proteinase 3 target antigen, myeloperoxidase-resistant Sm antibodies, anti-SSB antibody, anti-SSA antibody, Ro-52 antibody, Scl-70 antibody, Jo-1 antibody, centromere-resistant double-stranded DNA antibody, and nucleosome-resistant antibody. The extremity electromyography (EMG) findings were as follows: abnormal bilateral median nerve F wave, bilateral ulnar nerve F wave, and bilateral tibial nerve H reflex; these findings suggested proximal nerve root involvement.

Follow-up laboratory findings were as follows: lymphocytes, 68.5%; monocytes, 10.8%; absolute monocyte count, $0.87 \times 10^9/L$; Hb, 128.00 g/L; HCT, 39.7%; MCH, 26.7 pg; RDW, 15.5%; MPV, 8.90 fl; PDW, 9.50 fl; ratio of large platelets, 16.3%; aspartate aminotransferase, 13.2 U/L; and 2019-nCoV nucleic acid, negative. The patient again had negative findings for cytoplasmic ANCA, formaldehyde-resistant pANCA, formaldehyde-sensitive pANCA, glomerular basement membrane antibody, proteinase 3 target antigen, myeloperoxidase-resistant Sm antibodies, anti-SSB antibody, anti-SSA antibody, Ro-52 antibody, Scl-70 antibody, Jo-1 antibody, centromere-resistant double-stranded DNA antibody, and nucleosome-resistant antibody.

Imaging findings

MRI of both lower limbs revealed swelling of both lower legs, as well as large abnormal shadows scattered in the calf muscle groups on both sides. T1WI showed mainly uneven hypointensity, and a few slightly hyperintense “internal shadows”. Fat-saturated T2WI and T2WI showed large shadows with blurred edges. Occasional hypointense signals on T1WI and hyperintense signals on T2WI were

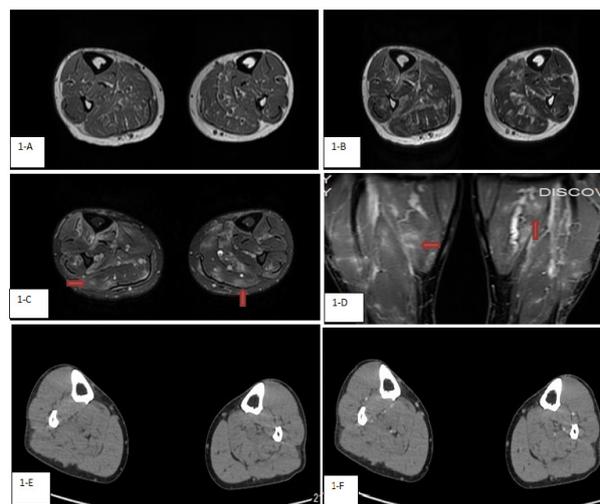


Figure 1. 1-A, T1WI image; 1-B, T2WI image; 1-C, fat-saturated T2WI image; 1-D, fat-saturated T1WI image; 1-E, Plain CT scan; 1-F, enhanced CT scan. On MRI, large abnormal shadows were scattered in bilateral calf muscle groups, with a few slightly hypointense signals on T1WI, hyperintense shadows on T2WI and fat-saturated T2WI, and blurred edges and cotton-like enhancement.

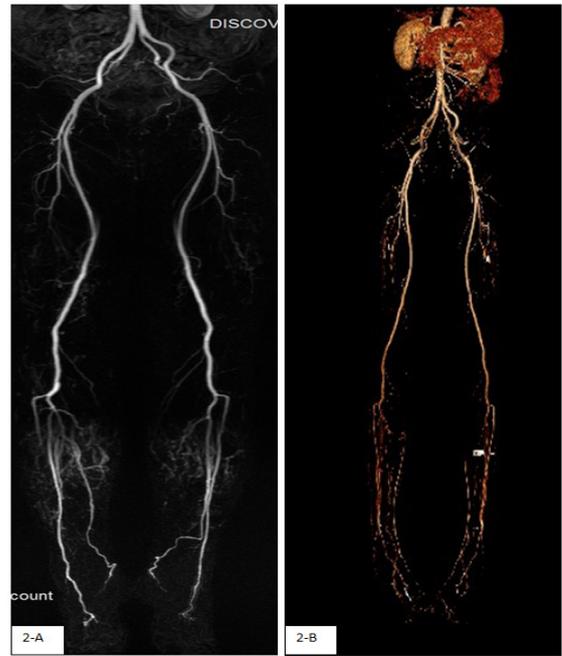


Figure 2. 2-A, MRA of Lower Extremity Vessels; 2-B, CTA of Lower Extremity Vessels. MRA and CTA showed beadlike changes in the branches of lower extremity arteries, as well as obvious branches of lower leg arteries.

evident in the middle tibiofibular region on both sides, along with cotton-like enhancement centered on blood vessels (Figure 1A–D). CT of both lower extremities revealed no abnormalities (Figure 1E, F).

Bilateral lower limb MRA revealed normal findings in the abdominal aorta, bilateral common iliac arteries, bilateral external iliac arteries, bilateral internal iliac arteries, bilateral femoral arteries, bilateral popliteal arteries, bilateral anterior tibial, and posterior tibial arteries and their branches, although they had slightly rough walls. The vessels of the lower limb arteries showed beadlike changes, and the branches of the bilateral calf artery were obvious (Figure 2A).

CTA of the bilateral lower limbs revealed normal findings in the abdominal aorta, renal artery, celiac axis and its branches, superior mesenteric artery, bilateral common iliac arteries, bilateral intenal iliac arteries, bilateral external iliac arteries, shallow bilateral femoral artery, deep artery, popliteal artery, peripheral artery, and posterior tibial artery and its branches. The vessels exhibited rough walls, with a double leg artery and its branches, the local change a beaded samples, superior mesenteric artery local expansion in tumor samples, at its widest point is about 9 mm, bilateral popliteal artery, double pretibial, posterior tibial artery and its branches development shallow light, double leg visible early show great saphenous vein, small saphenous vein, abnormal muscle did not see reinforcement (Figure 2B).

Cranial MRA revealed right middle cerebral artery stenosis and occlusion, but the other intracranial vessels were normal (Figure 3). Brain perfusion imaging and head MRI showed that cerebral blood flow (CBF) was

Table 1. Diagnosis and Treatment Process

Diagnosis and treatment	May 2018	March 2019	March 2020	March 2020 (follow-up)	July 2020
	No venous abnormalities were observed in either lower limb.	Erythema nodosum on both legs was suspected by the First Affiliated Hospital of Guangzhou University of TCM and the Fifth Affiliated Hospital of Sun Yat-sen University, but the appropriate treatment was unclear.	Subcutaneous nodule biopsy findings were consistent with ANCA-negative erythema nodosum and fever. Multidisciplinary consultation led to a diagnosis of PAN. Arthritis pain was relieved by prednisone acetate and methotrexate treatment, and his body temperature returned to normal.	Mesenteric arteriovenous CTA showed a slightly wider localized superior mesenteric artery. Ultrasound showed no abnormalities in the bilateral renal arteries, carotid arteries, or vertebral arteries.	Prednisone acetate and methotrexate treatments yielded joint pain relief and normal temperature. Injection of recombinant human tumor necrosis factor receptor II antibody led to substantial lower limb pain and fatigue reduced, but no fever.

Table 2. Literature and Statistical Table of Basic Information of this Case

Reference	Gender	Age	Position	T2-Weighted STIR Images	T1-Weighted Images	Contrast Enhancement	Fascial Lesion	Periosteal Lesion
Nakamura et al. [17]	F	38	leg	Diffuse increase	Not available	Not available	Not available	Not available
Yang et al. [18]	M	26	leg	Diffuse increase	Not available	Present	Not available	Not available
Hofman et al. [19]	F	36	leg	Diffuse increase	Not available	Not available	Not available	Not available
Révelon et al. [20]	M	43	leg	Patchy increase	Not available	Cotton-wool appearance	Present	Not available
Ahmed et al [21].	F	36	leg	Diffuse increase	Not available	Not available	Present	Not available
Gallien et al. [22]	F	41	leg	Patchy increase	Not available	Present	Not available	Present on bone scintigraphy
Gallien et al. [22]	F	45	leg	Diffuse increase	Within normal limit	Not available	Not available	Not available
Mac Donald and Blake [23]	M	20	leg	Patchy increase	Not available	Not available	Present	Present on bone scintigraphy
Esteva-Lorenzo et al. [24]	M	56	leg	Diffuse increase	Decrease	Not available	Absent	Present
Reading et al. [25]	M	38	thigh	Diffuse increase	Not available	Not available	Present	Absent
Kang, Y [26]	F	42	leg	Diffuse increase	Slight hyperintensity	Cotton-wool appearance	Absent	Absent
	M	7	leg	Diffuse increase	Within normal limit	Cotton-wool appearance	Absent	Present
	M	64	leg	Diffuse increase	Within normal limit	Diffuse enhancement	Present	Absent
	F	20	leg	Within normal limit	Within normal limit	Absent	Present	Absent
	M	7	leg	Patchy hyperintensity	Within normal limit	Cotton-wool appearance	Present	Present
	M	30	leg	Patchy hyperintensity	Slight hyperintensity	Cotton-wool appearance	Absent	Absent
	F	56	leg	Patchy hyperintensity	Within normal limit	Cotton-wool appearance	Absent	Present
	F	49	leg	Patchy hyperintensity	Within normal limit	Cotton-wool appearance	Absent	Present
Masahiro Aoshima [27]	F	78	leg	Patchy high signal Adipoid signal	Adipoid signal	Not available	Not available	Not available
Hiroshi Takei [28]	M	69	thigh	Discretely granular	Slightly high signal	Cotton-wool appearance	Absent	Absent
This case	M	55	leg thigh	Patchy hyperintensity	Within normal limit	Cotton-wool appearance	Absent	Absent

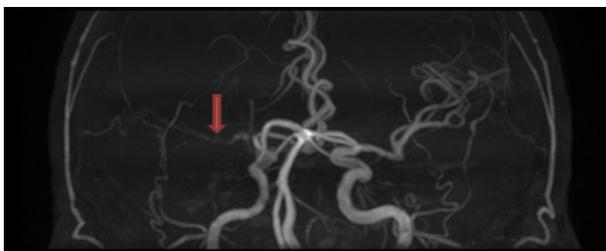


Figure 3. Right Middle Cerebral Artery Stenosis and Occlusion (arrow). The remaining intracranial vessels were normal

lower in the right cerebellar hemisphere than in the left cerebellar hemisphere at DLP1.5, although both sides were equivalent at DLP2.5. These findings were indicative of ischemic changes in the right cerebellar

hemisphere (Figure 4).

EMG of both lower extremities revealed an abnormal tibial nerve H reflex on both sides, which implied proximal nerve root involvement.

Diagnosis and treatment process

See Table 1.

Pathological examination

Skin biopsy of the left leg revealed findings consistent with erythema nodosum.

Discussion

PAN is a relatively rare rheumatic immune disease with various clinical manifestations and no specific serological markers. Therefore, it is difficult to diagnose

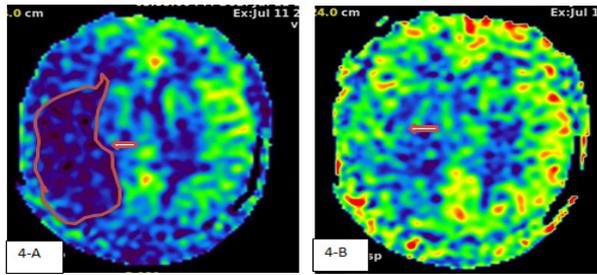


Figure 4. 4- A, Cerebral Perfusion of CBF at DLP1.5; 4-B, Cerebral Perfusion of CBF at DLP2.5. Magnetic resonance perfusion imaging indicated that CBF of the right cerebellar hemisphere decreased at DLP1.5, compared with that of the left cerebellar hemisphere. CBF was similar on both sides at DLP2.5 (arrows).

and clinicians have insufficient knowledge of this disease. The pathogenesis of PAN remains unclear. Some clinicians have suggested that it is heredity, where deletion of the autosomal recessive *CecRL* gene can lead to PAN [6]. Furthermore, patients with familial Mediterranean fever may present with PAN [7]. Viral infection, such as hepatitis B virus, hepatitis C virus, and human immunodeficiency virus, is an important potential etiology [8].

Patients with PAN may have nonspecific symptoms such as fever, weight loss, joint pain, and muscle pain. The corresponding organs or tissues may exhibit ischemia or bleeding due to narrowing or blockage of inflamed arteries, or microaneurysm rupture. PAN combined with cardiovascular lesions has the following imaging characteristics: extensive artery involvement, most commonly in the aorta and secondary branches; and diverse arterial lesions, primarily in the gut and lower extremities, including the renal artery and its branches. Coronary artery involvement is also common. The reported incidence of cardiac involvement of PAN ranges from 4% to 65% [9]. Vascular lesions of PAN disease show segmental changes, mainly in vascular branches, along with immune complex deposition and granuloma formation. Extensive inflammatory cell infiltration (mainly lymphocytes) is present in the lesions. Fibrosis-like necrosis is a common manifestation of active lesions, often accompanied by neutrophil infiltration [10-13].

The MRI manifestations of 21 patients (including our patient, as shown in Table 2) described in 12 English-language articles [17-28] were retrospectively analyzed: there were 17 patients (80.9%) with calf involvement, 3 (14.3%) with thigh involvement, and 1 (4.8%) with both thigh and calf involvement. The patients included 11 males and 10 females (mean age, 40.76 ± 18.28 years; range: 7–78 years). The T1WI and T2WI findings were both isointense in one patient, while the T1WI findings alone were isointense in seven patients. The T1WI findings were slightly hyperintense in five patients, and no T1WI images were available for the remaining seven patients. The T2WI signal was diffusely hyperintense in 10 patients, and “patchy villous hyperintense” in 9 patients. Among the 12 patients with enhanced images, most exhibited diffuse or cotton-like enhancement, and some patients had involvement of the

fascia and periosteum. Our patient showed early disease manifestations in the calves, and late manifestations in the thighs. MRI revealed that the thigh and calf muscles were involved, with T1WI isointensity and T2WI patch-like hyperintensity, as well as cotton-like enhancement centered on blood vessels. Enhanced muscle scans did not show abnormalities (Figure 1). Focal accumulation was indicative of vascular stenosis. The bilateral leg artery and its branches showed localized beadlike changes with many lesions. The wall of the profunda femoris artery was rough, with slightly localized stenosis and few lesions. It also showed aneurysmal dilation of the superior mesentery, as well as middle cerebral artery stenosis and occlusion, and ischemic compensatory changes in the right cerebellar hemisphere. However, no clinical symptoms were evident (Figures 2-4).

Currently, diagnosis of PAN is made using the American College of Rheumatology 1990 classification [14]. However, due to improvements in our understanding of vasculitis, this standard now clearly has limitations, especially because the ANCA antibody test does not reliably detect microscopic polyangiitis. PAN was defined at the Chapel Hill Conference in 2012 and determined to have no association with ANCA [3]. Some researchers have proposed combining the American College of Rheumatology, Laham Standard, and Chapel Hill Conference standards, such that granulomatous polyangiitis is ruled out first. If eosinophilic granulomatous polyangiitis, granulomatous polyangiitis, and microscopic polyangiitis can be excluded, then PAN may be the appropriate diagnosis [15].

Glucocorticoids are the currently preferred treatment, combined with immunosuppressants as necessary. Surgical treatment may be necessary in patients with serious complications, such as gastrointestinal perforation, visceral rupture, ischemia, or bleeding. In a retrospective database analysis conducted in 2011, the French Vasculitis Research Group found that factors associated with 5-year mortality in patients with PAN included age > 65 years, renal insufficiency (serum creatinine $\geq 150 \mu\text{mol/L}$), symptomatic cardiac insufficiency and, especially, severe gastrointestinal involvement (e.g., perforation, hemorrhage, and/or pancreatitis) [16].

In Conclusion, in cases with patchiness or diffuse muscle signal changes in the lower leg (or thigh) on MRI, cotton-like or diffuse enhancement centered on blood vessels may be accompanied by fascial or periosteal enhancement. PAN should be considered in the differential diagnosis, and vascular CTA or MRA examinations should be added to facilitate the diagnosis. In our patient, only the skin and lower leg showed clinical symptoms, but the profunda femoris artery, celiac trunk, and cranial vessels were abnormal. Therefore, patients with PAN should undergo examinations of small and medium arteries throughout the body, to ensure early detection of diseased vessels. This may prevent or reduce damage to target organs. If focal accumulation is suspected, MRI and MRA examinations are recommended.

References

- Watts RA, Jolliffe VA, Carruthers DM, Lockwood M, Scott DGI. Effect of classification on the incidence of polyarteritis nodosa and microscopic polyangiitis. *Arthritis & Rheumatism*. 1996 07;39(7):1208-1212. <https://doi.org/10.1002/art.1780390720>
- Selga D, Mohammad A, Sturfelt G, Segelmark M. Polyarteritis nodosa when applying the Chapel Hill nomenclature—a descriptive study on ten patients. *Rheumatology*. 2006 08 09;45(10):1276-1281. <https://doi.org/10.1093/rheumatology/keq091>
- 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 CGM, Lamprecht P, Langford CA, Luqmani RA, Mahr AD, Matteson EL, Merkel PA, Ozen S, Pusey CD, Rasmussen N, Rees AJ, Scott DGI, Specks U, Stone JH, Takahashi K, Watts RA. 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. *Arthritis & Rheumatism*. 2012 Dec 27;65(1):1-11. <https://doi.org/10.1002/art.37715>
- Lin, H.Y, W.F. Fagan, P.E. Jabin. *Polyarteritis and relate disorders Kelley's textbook of rheumatology*. saunders company. 2009;:1539-47.
- John S sergent. *Polyarteritis and relate disorders Kelley's textbook of rheumatology*[M]-8th ed WB. saunders company. 2009;:1539-47.
- Navon Elkan P, Pierce SB, Segel R, Walsh T, Barash J, Padeh S, Zlotogorski A, Berkun Y, Press JJ, Mukamel M, Voth I, Hashkes PJ, Harel L, Hoffer V, Ling E, Yalcinkaya F, Kasapcopur O, Lee MK, Klevit RE, Renbaum P, Weinberg-Shukron A, Sener EF, Schormair B, Zeligson S, Marek-Yagel D, Strom TM, Shohat M, Singer A, Rubinow A, Pras E, Winkelmann J, Tekin M, Anikster Y, King M, Levy-Lahad E. Mutant Adenosine Deaminase 2 in a Polyarteritis Nodosa Vasculopathy. *New England Journal of Medicine*. 2014 03 06;370(10):921-931. <https://doi.org/10.1056/nejmoa1307362>
- Standing ASI, Eleftheriou D, Lachmann HJ, Brogan PA. Familial Mediterranean fever caused by homozygous E148Q mutation complicated by Budd-Chiari syndrome and polyarteritis nodosa. *Rheumatology*. 2010 Dec 11;50(3):624-626. <https://doi.org/10.1093/rheumatology/keq405>
- Pagnoux C, Seror R, Henegar C, Mahr A, Cohen P, Le Guern V, Bienvenu B, Mouthon L, Guillevin L. 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 & Rheumatism*. 2010 02;62(2):616-626. <https://doi.org/10.1002/art.27240>
- Cohen, R.D , D.L. Conn, , D.M. Ilstrup. Clinical features, prognosis, and response to treatment in polyarteritis. *Mayo Clin Proc*. 1980;55(3):146-55.
- Bae YD, Choi HJ, Lee JC, Park JJ, Lee YJ, Lee EB, Song YW. Clinical Features of Polyarteritis Nodosa in Korea. *Journal of Korean Medical Science*. 2006;21(4):591. <https://doi.org/10.3346/jkms.2006.21.4.591>
- Coll-Vinent B, Cebrin M, Cid MC, Font C, Esparza J, Juan M, Yage J, Urbano-Mrquez, Grau JM. Dynamic pattern of endothelial cell adhesion molecule expression in muscle and perineural vessels from patients with classic polyarteritis nodosa. *Arthritis & Rheumatism*. 1998 03;41(3):435-444. [https://doi.org/10.1002/1529-0131\(199803\)41:3<435::aid-art9>3.0.co;2-9](https://doi.org/10.1002/1529-0131(199803)41:3<435::aid-art9>3.0.co;2-9)
- Cid M, Grau JM, Casademont J, Campo E, Coll-Vinent B, López-Soto A, Ingelmo M, Urbano-Márquez A. Immunohistochemical characterization of inflammatory cells and immunologic activation markers in muscle and nerve biopsy specimens from patients with systemic polyarteritis nodosa. *Arthritis & Rheumatism*. 1994 07;37(7):1055-1061. <https://doi.org/10.1002/art.1780370711>
- Lie J. Systemic and isolated vasculitis. A rational approach to classification and pathologic diagnosis. *Pathol Annu*. 1989;24 Pt 1:25-114.
- Lightfoot RW, Michel BA, Bloch DA, Hunder GG, Zvaifler NJ, McShane DJ, Arend WP, Calabrese LH, Leavitt RY, Lie JT, Masi AT, Mills JA, Stevens MB, Wallace SL. The American college of rheumatology 1990 criteria for the classification of polyarteritis nodosa. *Arthritis & Rheumatism*. 2010 08 17;33(8):1088-1093. <https://doi.org/10.1002/art.1780330805>
- Watts R, Lane S, Hanslik T, Hauser T, Hellmich B, Koldingsnes W, Mahr A, Segelmark M, Cohen-Tervaert JW, Scott D. Development and validation of a consensus methodology for the classification of the ANCA-associated vasculitides and polyarteritis nodosa for epidemiological studies. *Annals of the Rheumatic Diseases*. 2006 08 11;66(2):222-227. <https://doi.org/10.1136/ard.2006.054593>
- Guillevin L, Pagnoux C, Seror R, Mahr A, Mouthon L, Toumelin PL. The Five-Factor Score Revisited. *Medicine*. 2011 01;90(1):19-27. <https://doi.org/10.1097/md.0b013e318205a4c6>
- Nakamura T, Tomoda K, Yamamura Y, Tsukano M, Honda I, Iyama K. Polyarteritis nodosa limited to calf muscles: a case report and review of the literature. *Clinical Rheumatology*. 2003 05 01;22(2):149-153. <https://doi.org/10.1007/s10067-002-0688-8>
- Yang SN, Cho NS, Choi HS, Choi SJ, Yoon E, Kim DH. Muscular Polyarteritis Nodosa. *JCR: Journal of Clinical Rheumatology*. 2012 08;18(5):249-252. <https://doi.org/10.1097/rhu.0b013e318262e3dd>
- Hofman DM, Lems WF, Witkamp TD, V.D.Putte SCJ, Bijlsma JJJ. Demonstration of calf abnormalities by magnetic resonance imaging in polyarteritis nodosa. *Clinical Rheumatology*. 1992 09;11(3):402-404. <https://doi.org/10.1007/bf02207202>
- Révelon G, Rahmouni A, Jazaerli N, Godeau B, Chosidow O, Authier J, Mathieu D, Roujeau J, Vasile N. Acute swelling of the limbs: magnetic resonance pictorial review of fascial and muscle signal changes. *European Journal of Radiology*. 1999 04;30(1):11-21. [https://doi.org/10.1016/s0720-048x\(98\)00119-3](https://doi.org/10.1016/s0720-048x(98)00119-3)
- Ahmed S, Kitchen J, Hamilton S, Brett F, Kane D. A case of polyarteritis nodosa limited to the right calf muscles, fascia, and skin: a case report. *Journal of Medical Case Reports*. 2011 09 12;5(1). <https://doi.org/10.1186/1752-1947-5-450>
- Gallien S. Magnetic resonance imaging of skeletal muscle involvement in limb restricted vasculitis. *Annals of the Rheumatic Diseases*. 2002 Dec 01;61(12):1107-1109. <https://doi.org/10.1136/ard.61.12.1107>
- MacDonald WBG, Blake MP. Periostitis and Localized Myositis in Polyarteritis Nodosa. *Clinical Nuclear Medicine*. 2004 Nov;29(11):703-705. <https://doi.org/10.1097/00003072-200411000-00006>
- Esteve-Lorenzo FJ, Ferreira JL, Tardaguila F, de la Fuente A, Falasca G, Reginato AJ. Case report 866. *Skeletal Radiology*. 1994 Oct;23(7):572-576. <https://doi.org/10.1007/bf00223096>
- Reading P, Hudgson P, Johnson M, Jenkins A. A sartorial challenge. *The Lancet*. 1999 09;354(9183):996. [https://doi.org/10.1016/s0140-6736\(99\)05342-8](https://doi.org/10.1016/s0140-6736(99)05342-8)
- Kang Y, Hong SH, Yoo HJ, Choi J, Park JK, Park J, Kang

- HS. Muscle Involvement in Polyarteritis Nodosa: Report of Eight Cases With Characteristic Contrast Enhancement Pattern on MRI. *American Journal of Roentgenology*. 2016 02;206(2):378-384. <https://doi.org/10.2214/ajr.15.14774>
27. Aoshima M, Fukuchi K, Tatsuno K, Ito T, Tokura Y. Ectopic Adipose Tissue with Vasculitis in the Calf Muscle Explaining Systemic Symptoms in Leg-limited Cutaneous Polyarteritis Nodosa. *Acta Dermato Venereologica*. 2016;96(1):142-143. <https://doi.org/10.2340/00015555-2163>
28. Takei H, Hanaoka H, Kaneko Y, Yamaoka K, Sasaki A, Takeuchi T. Intriguing Findings of the Muscle on Magnetic Resonance Imaging in Polyarteritis Nodosa. *Internal Medicine*. 2016;55(21):3197-3200. <https://doi.org/10.2169/internalmedicine.55.7110>



This work is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.