



Tuberculosis and Takayasu arteritis: case-based review

Moritz K. Jansson¹ · Hilte F. Geerdes-Fenge¹ · Antje Kangowski² · Christian Kneitz² · Emil C. Reisinger¹

Received: 30 September 2018 / Accepted: 14 December 2018 / Published online: 3 January 2019
© Springer-Verlag GmbH Germany, part of Springer Nature 2019

Abstract

This report presents a case of co-occurrence of Takayasu arteritis (TA) and multiorgan tuberculosis (TB) in a 20-year-old female and provides a review of 18 previously reported cases of co-occurring TA and TB. All patients were between 9 and 24 years of age. Most reports describe a concomitant diagnosis of active TB and TA. TB lymphadenitis was described in 11 cases (57.9%), and microbiologically confirmed in 4 of these. All patients received antituberculous therapy and most received corticosteroids (89.5%). In our and two other cases, TA relapses necessitating additional immunosuppressive therapy were observed.

Keywords Vasculitis · Autoimmune disease · Takayasu arteritis · Tuberculosis · Tuberculosis, Lymph node

Introduction

Tuberculosis (TB) is caused by bacteria of the *Mycobacterium tuberculosis* complex and predominantly affects the lungs. Symptoms include fever, night sweats, fatigue, cough, and weight loss. In 2017, an estimated 10.0 million people (range 9.0–11.1 million) developed TB disease worldwide (5.8 million men, 3.2 million women and 1.0 million children). Extrapulmonary tuberculosis was present in 14% of TB incident cases [1].

Takayasu arteritis (TA) is a systemic granulomatous inflammation of the large and medium-sized arteries.

Arterial thrombosis, occlusion, and aneurysms with end-organ ischemia are common complications [2]. The leading causes of death in TA are heart failure, hemorrhage and pulmonary infection [3]. Young women of child-bearing age seem to be more frequently affected than older women or men of any age group [4]. Annual incidences vary between 1/1,000,000 per year in Europe, 2/1,000,000 per year in Japan, 2.2/1,000,000 per year in Kuwait and 2.6/1,000,000 per year in North America. Reported female:male ratios range from 1.2:1 in Israel to 6.9:1 in Mexico [2]. The pathogenesis of TA is poorly understood. Several genetic loci associated with increased susceptibility have been identified, the most robust of which is located within the human leukocyte antigen (HLA) class I region [5]. However, environmental factors are also thought to play a role. TB has been discussed repeatedly as a possible cause or trigger of TA because lesions in the two diseases show histological similarities [6].

This report presents a case in which TA co-occurred with pulmonary and multiorgan TB and analyses previously published cases of co-occurring TA and TB.

Case report

A 20-year-old HIV-negative female from Syria presented with recurrent tenderness of the entire left thorax, present for the last 9 months, increasing dyspnea, subfebrile temperatures, and weight loss of 10 kg over the last 12 months.

✉ Moritz K. Jansson
moritz.jansson@uni-rostock.de

Hilte F. Geerdes-Fenge
hilte.geerdes-fenge@uni-rostock.de

Antje Kangowski
antje.kangowski@klinikusued-rostock.de

Christian Kneitz
christian.kneitz@rheuma-schwerin.de

Emil C. Reisinger
emil.reisinger@uni-rostock.de

¹ Department of Tropical Medicine and Infectious Diseases, Rostock University Medical Center, Ernst-Heydemann-Str. 6, 18057 Rostock, Germany

² Department of Medicine, Rheumatology and Clinical Immunology, Klinikum Südstadt, Rostock, Südring 81, 18059 Rostock, Germany

The patient had arrived in Germany as a refugee from Syria 6 months before hospital admission and given birth to a healthy boy 1 month later. There were no other events in her past medical history, apart from iron deficiency anemia.

Clinical findings included reduced respiratory sounds at the base of the right lung, cachexia (body weight 48 kg, height 170 cm, body mass index 16.6 kg/m²), a difference of 14 mm Hg between the systolic blood pressures of the left arm (97/61 mmHg) and the right arm (111/73 mmHg), and bruits over the left subclavian artery and the common carotid arteries. The pulse of the left radial artery was weaker than the pulse of the right radial artery.

A chest radiograph and computed tomography (CT) scan of the thorax revealed an extensive pleural effusion on the right side accompanied by atelectasis of the adjacent lung tissue, a minor pleural effusion on the left side, disseminated miliary-like opacities in both lungs and lymphadenopathy of the mediastinum and the hili (Fig. 1). A magnetic resonance tomography (MRT) of the cranium revealed seven small ring-like, contrast medium-absorbing cerebellar lesions of up to 7.3 mm in size, and at least 16 supratentorial lesions of up to 5 mm in size in both cerebral hemispheres, consistent with intracerebral tuberculoma (Fig. 2). The interferon-gamma release assay (QuantiFERON-TB-gold-in-tube) was positive. Because of the high likelihood of pulmonary and extrapulmonary TB with cerebral tuberculoma and intrathoracic lymphadenopathy, antituberculous treatment was started with weight-adjusted daily oral doses of isoniazid/pyridoxine, rifampin, pyrazinamide and ethambutol. After

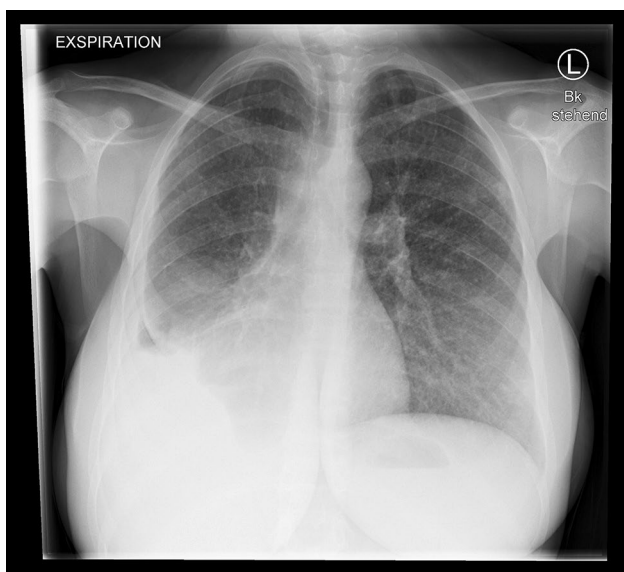


Fig. 1 A chest radiograph reveals extensive pleural effusion on the right side accompanied by atelectasis of the adjacent lung tissue, disseminated miliary-like opacities in both lungs and lymphadenopathy of the hili

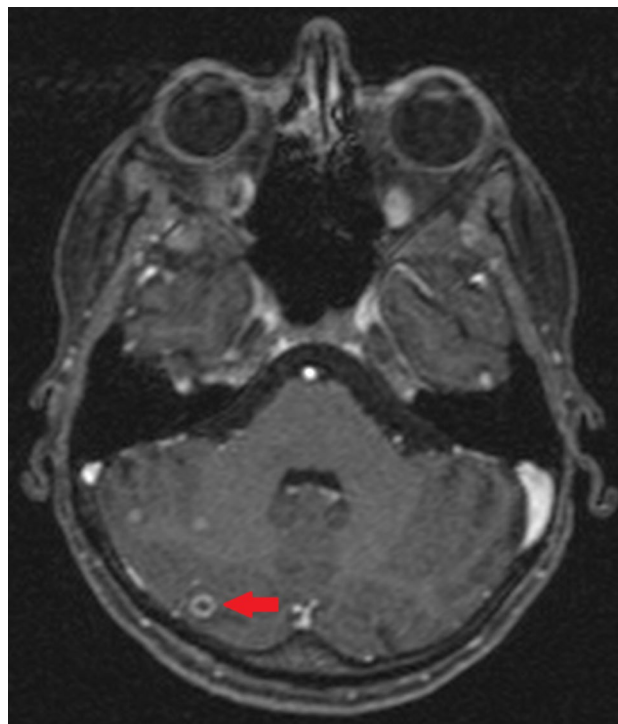


Fig. 2 An MRT scan of the cranium revealed seven small ring-like, contrast medium-absorbing cerebellar lesions of up to 7.3 mm in size (arrow), and at least 16 supratentorial lesions of up to 5 mm in size in both cerebral hemispheres, consistent with intracerebral tuberculoma

three weeks of culture, the bronchoalveolar lavage (BAL) grew *Mycobacterium tuberculosis*.

An MRT scan of the thoracic aorta revealed concentric mural thickening of the aortic arch and supra-aortic branches, a long stenosis of the left common carotid artery, a proximal stenosis of the right common carotid artery, and another stenosis of the middle section of the left subclavian artery. The vertebral arteries were normal (Fig. 3). Thickening and stenosis of the tunica intima of the extracranial sections of the carotid arteries and stenosis of the left internal and right external carotid arteries were revealed by duplex ultrasound. A diagnosis of concomitant TA was made. Prednisolone [initially 250 mg once daily (OD)] and oral acetylsalicylic acid (100 mg OD) were added to the treatment regimen, with prednisolone then gradually tapered to 7 mg OD.

The patient showed clinical and radiological signs of remission within the first year of follow-up with complete resolution of cerebellar and supratentorial lesions after 12 months of antituberculous therapy. No neurological symptoms were observed at first presentation or within the follow-up period. A follow-up duplex ultrasound showed no progression of carotid or subclavian arteritis. The bruits over the left subclavian artery and common carotid arteries persisted. In the twelfth month of follow-up, an MRT scan of the affected blood vessels showed vessel wall thickening

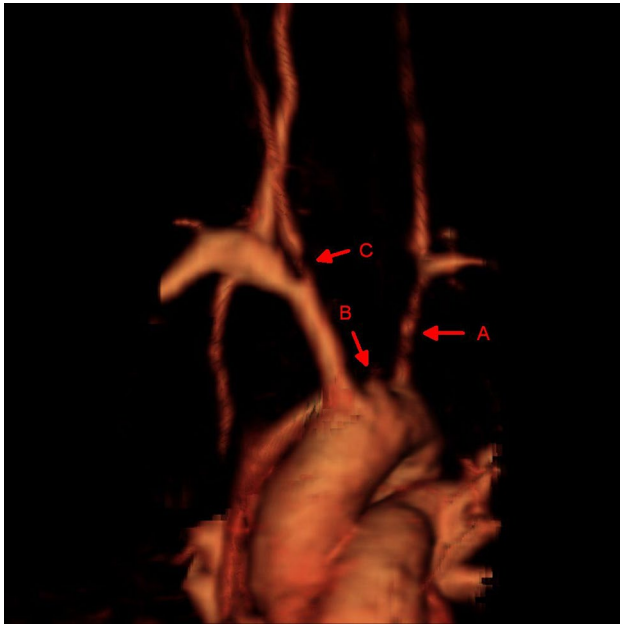


Fig. 3 An MRT scan of the thoracic aorta revealed concentric mural thickening of the aortic arch and supra-aortic branches, stenosis of the middle section of the left subclavian artery (A), a long stenosis of the left common carotid artery (B) and a proximal stenosis of the right common carotid artery (C). The vertebral arteries were normal

and contrast medium enhancement along the thoracic aorta, consistent with radiological signs of active TA [7]. Immunosuppressive therapy was supplemented with a weekly dose of methotrexate (initially 15 mg subcutaneous administration [sc.], then 20 mg sc.) which, after an initial phase of remission, did not prevent a further relapse 6 months later, when an MRT scan showed the previous radiological signs of active vasculitis in the affected vessels and the descending aorta in particular. We decided to terminate methotrexate therapy and opt for anti-interleukin-6 agents, starting a weekly dose of tocilizumab (162 mg sc.). Seven weeks into the adjusted immunosuppressive therapy regimen the patient was in a good general state. A follow-up MRT scan is planned in a few months.

Search strategy

A systematic search of the literature on the electronic databases Pubmed and Scopus was conducted using the search terms “Takayasu’s arteritis AND tuberculosis” and “large vessel vasculitis AND tuberculosis” in October 2018. Criteria for inclusion in this study were reports in English or German language of patients with a diagnosis of TA (meeting the American College of Rheumatology [ACR] 1990 or EULAR/PRINTO/PRES classification criteria [8, 9]) and TB (previous active TB diagnosis and treatment or clinical

evidence of active TB supported by tuberculin sensitivity testing/interferon-gamma release assay/acid-fast staining/culture/radiological imaging). The search yielded 78 case reports, 17 of which (describing 18 cases) met the inclusion criteria. The current case was also included for analysis.

Literature review and analysis

Most reports (15; 83.3%) were published in or after the year 2000. The mean age of patients presenting with co-occurrence of TA and TB was 16.8 (range 9–24), the female:male ratio was 3.8:1. The main presenting symptoms were fever (52.6%), weight loss, claudication of extremities (each 31.6%), cough, dyspnea and headaches (each 26.3%). One patient presented with a cervical mass, one with a reticulated rash, one because of a syncope episode and one with convulsion. Two patients had previously been treated for pulmonary TB (1 and 2 years earlier, respectively) and one had a previous diagnosis of papulonecrotic tuberculid before the first evidence of TA was found. In all other cases (16; 84.2%) diagnoses of active TB and TA were made simultaneously at presentation or a few weeks apart (Table 1). In two of these cases the patients had been treated for TB several years earlier. Another case report described a history of hypertension, possibly an early symptom of TA, in a young woman years before a concomitant diagnosis of TA and TB was made. The most frequently observed angiographic lesion types according to the angiographic classification of TA lesions proposed in 1994 [10] were type V (6; 31.6%) followed by types IV and I (4; each 21.1%) (Table 1). In five cases (26.3%) TA was associated with heart failure. TB limited to the lungs was only described in four cases (21.1%). Extrapulmonary TB (EPTB) was reported in 14 cases (73.7%), in three of which evidence of both pulmonary TB and EPTB was found. There was one case of intestinal TB, one case of renal TB and one case of papulonecrotic tuberculid. In one case tuberculous arteritis was suspected but could not be confirmed. Eleven reports (57.9%), including our case, described evidence of TB lymphadenitis (Table 1). In seven of these cases biopsies were performed. In three cases, biopsy showed caseating tubercles suggestive of TB lymphadenitis. In 4 cases, bacteriological confirmation by culture and/or Ziehl–Neelson staining was successfully performed from biopsy tissue of two cervical, one supraclavicular and one hilar lymph node [11–14].

Corticosteroids were used in 17 cases (89.5%). In one case, concomitant Crohn’s disease led to the use of sulfasalazine and azathioprine without corticosteroids. All patients but one received both antituberculous and immunosuppressive therapy. Pantel et al. described an improvement in symptoms of TA and a return of pulses solely upon administration of antituberculous therapy [12]. Two reports described TA

Table 1 Co-occurrence of TA and TB: Cases from the literature review

Study	Sex	Age	Ethnicity	TB manifestation	Angio-graphic lesion type ^a	TA treatment	Timing of TA and TB diagnosis
Al-Aghbari, 2010 [15]	f	18	Yemeni	Cervical TB lymphadenitis	V	CS, Aspirin	Concomitant
Almohammadi, 2007 [11]	f	16	Canadian	Hilar and mediastinal TB lymphadenitis	IV	P	Concomitant (evidence of TA and TB 5 years before presentation)
Almohammadi, 2007 [11]	f	17	Canadian	Hilar, paratracheal, portohepatic lymphadenopathy; bone, pulmonary	V	P	Concomitant (latent TB 4 years earlier)
Baijal, 2015 [16]	f	21	Indian	Intestinal	I	Sulphasalazine, Aspirin	Concomitant
Moura, 2015 [17]	f	23	Brazilian	Pulmonary, suspicion of tuberculous arteritis	Iib, P+	P	Concomitant
Khemiri, 2016 [18]	f	12	Tunesian	Hilar lymphadenitis, pulmonary	I	P, MTX, Aspirin	Concomitant
Walters, 2013 [19]	f	16	Haitian	Pulmonary	V, C+	P, MTX, Rituximab, Aspirin	Concomitant
Duzova, 2000 [20]	m	12	Turkish	Left kidney	V	P, Cyclophosphamide	Concomitant
Pantell, 1981 [12]	f	12	Afro-American	Cervical TB lymphadenitis	Iib	–	Concomitant (pulmonary TB in early childhood)
Kontogiannis, 2000 [21]	f	14	Caucasian	Papulonecrotic tuberculide	IV	P, MTX	TB many years earlier
Zaki, 2011 [22]	m	9	Indian	Abdominal TB lymphadenopathy	IV	P	Concomitant
Modi, 2000 [13]	f	22	Indian	Supraclavicular TB lymphadenitis	I	CS	Concomitant
Mukherjee, 2013 [23]	f	14	Indian	Pulmonary	IV	P, MTX, AZA, Cyclophosphamide	Concomitant
Yang, 2013 [14]	f	9	Taiwanese	Cervical TB lymphadenitis	Iib/III	Methylprednisolone	TB 2 months earlier
Wensing, 1987 [24]	f	24	German	Pulmonary	I	P	TB 2 years before TA
Sivashanmugam, 1981 [25]	m	23	Indian	Pulmonary	V	P	TB 1 year before TA
Ahasan, 2009 [26]	f	20	Bangladeshi	Axillary TB lymphadenitis	V	CS	Concomitant
Reshkova, 2016 [27]	m	18	Bulgarian	Axillary TB lymphadenitis	Iia	Methylprednisolone	Concomitant
current case	f	20	Syrian	Hilar and mediastinal lymphadenitis; pulmonary, intracerebral	Iia	P, MTX, aspirin, tocilizumab	Concomitant

f female, m male, CS Corticosteroids, P, Prednisolone, MTX Methotrexate

^aAngiographic TA lesion types according to the 1994 classification. Type I, branches from the aortic arch; Type Iia, ascending aorta, aortic arch and its branches; Type Iib, ascending aorta, aortic arch with its branches and thoracic descending aorta; Type III, the thoracic descending aorta, abdominal aorta and/or renal arteries; Type IV, only the abdominal aorta and/or renal arteries; Type V, the combined features of both type Iib and IV. Involvement of coronary or pulmonary arteries is indicated as C (+) or P (+), respectively

relapses upon corticosteroid tapering during/after antituberculous therapy, necessitating the administration of methotrexate and, in one case, subsequently rituximab. Methotrexate was used in five cases (26.3%), cyclophosphamide in two cases, azathioprine in one case (Table 1). The time of

follow-up varied drastically from a few weeks to four years. Remission from active TA was described in all patients. TA manifestations in one patient led to nephrectomy and one received a splenorenal bypass. No patient died in hospital or during follow-up.

Discussion

Our patient presented with unspecific symptoms that had been present for almost 1 year. Unspecific symptoms were described in most of the reviewed reports. The patient fulfilled the criteria for multiorgan TB and the ACR 1990 criteria for Takayasu arteritis with type IIa angiographic lesions [8]. Angiographic lesions were heterogeneous in the reviewed cases, the most frequently observed lesions were also described as the three most common lesion types in a study of Indian and Japanese patients (types I, IV and V) [10].

Immunosuppressive treatment is required to prevent irreversible vessel damage in TA patients. Corticosteroid monotherapy led to remission in eleven reviewed cases of co-occurring TA and TB (57.9%), compared to a remission rate of up to 60% in cases of TA alone [2]. However, relapses frequently occur when a dose reduction is attempted, as observed in our case and two of the reviewed cases [18, 19]. Other treatment options for TA include methotrexate, cyclophosphamide, azathioprine, mycophenolate mofetil, tacrolimus hydrate, leflunomide, anti-TNF agents and tocilizumab [4]. A possible exacerbation of TB during treatment of TA with immunosuppressive agents has to be taken into consideration as anti-TNF agents are well known for their potential to reactivate latent TB [28]. When methotrexate failed to prevent TA relapses, rituximab was successfully used in one case [19] and tocilizumab was used in our case.

Eleven retrospective studies analyzed by Lim et al. have observed much higher frequencies of TB (history of TB, positive tuberculin skin test or active TB) in cases of TA compared to the general population in the respective countries or control groups [29]. A retrospective study by Lupi-Herrera et al. showed that 21% of TA patients had TB lymphadenitis at presentation or in their histories while only 1% had pulmonary TB [30]. However, no further details of microbiological TB confirmation were provided. In a study of 267 Korean patients with TA, 47 had a diagnosis of TB, with a high prevalence of TB lymphadenitis (12/47, 25.5%)—well above the national Korean rate of 4.4% [29]. In six patients, lymph node biopsy showed caseous granulomas. However, TB was confirmed by polymerase chain reaction (PCR) in one patient only, while the remaining cases were not microbiologically analyzed. Like TA, TB lymphadenitis seems to be more common in young female patients. It presents most commonly as cervical lymphadenitis but various other locations have been described [31]. Of the 57.9% of cases involving TB lymphadenitis in the reviewed reports, hilar (4; 36.4%) and cervical (3; 27.3%) lymphadenitis were the most frequently described locations (Table 1). Microbiological confirmation of TB lymphadenitis was obtained in four (21.1%) of the reviewed cases.

In rare cases TB can mimic the presentation of large vessel vasculitis [32, 33]. In one of the reviewed cases TA lesions could not be differentiated from possible tuberculous arteritis due to anatomical proximity of pulmonary TB lesions to the aortic arch, presenting a possible dissemination route [17]. As both TA and tuberculous arteritis share radiological features, imaging methods do not represent a reliable diagnostic tool for differentiation. In uncertain cases clinical management decisions are challenging as erroneous administration of immunosuppressive therapy could adversely affect the course of TB [17].

An autopsy study in Mexico detected the IS6110 and *HubB* gene sequences of *Mycobacterium tuberculosis* in 70% of aortic tissue samples from TA patients, compared to 82% in patients with tuberculosis and 32% in patients with atherosclerosis [6]. However, the DNA extraction in this study was performed from paraffin-embedded tissue. In fresh arterial samples obtained from 10 TA patients without clinical evidence of active tuberculosis, microscopy, mycobacterial culture, and PCR were all negative for *M. tuberculosis* complex [34].

While there is no conclusive evidence that mycobacteria play a direct role in the pathogenesis of TA, an immunological cross-reaction between mycobacterial and arterial antigens may be possible. Patients with TA tend to display a heightened humoral immune response to *Mycobacterium tuberculosis* antigens, in particular to the 65 kDa heat shock protein [35], which may suggest that, due to the sequence homology between the phylogenetically conserved human and mycobacterial heat shock proteins, a TB infection can cause an autoimmune response directed against the arterial adventitia [35].

This review comprises only a small number of cases spanning a large time period and a wide variety of cultures, both factors which may limit their comparability. Most patients presented with active TB and TA. We found a surprisingly high frequency of TB lymphadenitis.

In the majority of cases antituberculous and immunosuppressive therapy were given to manage both conditions, preventing fatal outcomes within the documented periods of follow-up. However, in our and two other cases, TA had to be treated with additional immunosuppressive therapy after the end of antituberculous therapy, indicating that successful completion of antituberculous therapy does not affect the course of TA.

Author contributions MKJ: writing of manuscript including editing and revision and the literature review at all stages of its production, clinical management of patient. HFG-F: writing of manuscript including editing and revision and the literature review at all stages of its production, clinical management of patient. AK/CK: clinical management of patient, contributions to editing and revision. ECR: manuscript editing and revision at all stages of production, final approval of manuscript, clinical management of patient.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent A written informed consent was signed by the patient in case concerning publication of this case report. A copy of this consent is available for editors of this journal.

References

- WHO (2018) Global Tuberculosis Report
- Brunner J, Feldman BM, Tyrrell PN, Kuemmerle-Deschner JB, Zimmerhackl LB, Gassner I, Benseler SM (2010) Takayasu arteritis in children and adolescents. *Rheumatology* 49:1806–1814. <https://doi.org/10.1093/rheumatology/keq167>
- Li J, Zhu M, Li M, Zheng W, Zhao J, Tian X, Zeng X (2016) Cause of death in Chinese Takayasu arteritis patients. *Medicine* 95:e4069. <https://doi.org/10.1097/MD.0000000000004069>
- Serra R, Butrico L, Fugetto F, Chibireva MD, Malva A, De Caridi G, Massara M, Barbetta A, Cannistrà M, de Franciscis S (2016) Updates in pathophysiology, diagnosis and management of Takayasu arteritis. *Ann Vasc Surg* 35:210–225. <https://doi.org/10.1016/j.avsg.2016.02.011>
- Saruhan-Direskeneli G, Hughes T, Aksu K, Keser G, Coit P, Aydin SZ, Alibaz-Oner F, Kamali S, Inanc M, Carette S, Hoffman GS, Akar S, Onen F, Akkoc N, Khalidi NA, Koening C, Karadag O, Kiraz S, Langford CA, McAlear CA, Ozbalkan Z, Ates A, Karaaslan Y, Maksimowicz-Mckinnon K, Monach PA, Ozer HT, Seyahi E, Fresko I, Cefle A, Seo P, Warrington KJ, Ozturk MA, Ytterberg SR, Cobankara V, Onat AM, Guthridge JM, James JA, Tunc E, Duzgun N, Bicakcigil M, Yentür SP, Merkel PA, Direskeneli H, Sawalha AH (2013) Identification of multiple genetic susceptibility loci in Takayasu arteritis. *Am J Hum Genet* 93:298–305. <https://doi.org/10.1016/j.ajhg.2013.05.026>
- Soto ME, Del Carmen Ávila-Casado M, Huesca-Gómez C, Alarcon GV, Castrejon V, Soto V, Hernandez S, Espinola-Zavaleta N, Vallejo M, Reyes PA, Gamboa R (2012) Detection of IS6110 and HupB gene sequences of *Mycobacterium tuberculosis* and bovis in the aortic tissue of patients with Takayasu's arteritis. *BMC Infect Dis* 12:194. <https://doi.org/10.1186/1471-2334-12-194>
- Barra L, Kanji T, Malette J, Pagnoux C, CanVasc (2018) Imaging modalities for the diagnosis and disease activity assessment of Takayasu's arteritis: a systematic review and meta-analysis. *Autoimmun Rev* 17:175–187. <https://doi.org/10.1016/j.autrev.2017.11.021>
- Arend WP, Michel BA, Bloch DA, Hunder GG, Calabrese LH, Edworthy SM, Fauci AS, Leavitt RY, Lie JT, Lightfoot RW (1990) The American College of Rheumatology 1990 criteria for the classification of Takayasu arteritis. *Arthritis Rheum* 33:1129–1134
- Ozen S, Pistorio A, Iusan SM, Bakkaloglu A, Herlin T, Brik R, Buoncompagni A, Lazar C, Bilge I, Uziel Y, Rigante D, Cantarini L, Hilario MO, Silva CA, Alegria M, Norambuena X, Belot A, Berkun Y, Estrella AI, Olivieri AN, Alpigiani MG, Rumba I, Sztajnbock F, Tambic-Bukovac L, Breda L, Al-Mayouf S, Mihaylova D, Chasnyk V, Sengler C, Klein-Gitelman M, Djeddi D, Nuno L, Pruunsild C, Brunner J, Kondi A, Pagava K, Pederzoli S, Martini A, Ruperto N, Paediatric Rheumatology International Trials Organisation (PRINTO) (2010) EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: final classification criteria. *Ann Rheum Dis* 69:798–806. <https://doi.org/10.1136/ard.2009.116657>
- Hata A, Noda M, Moriwaki R, Numano F (1996) Angiographic findings of Takayasu arteritis: new classification. *Int J Cardiol* 54(Suppl):S155–S163
- Almohammadi AA, Consunji-Araneta R (2007) Two cases of Takayasu's arteritis and tuberculosis. *Chest* 132:675A. https://doi.org/10.1378/chest.132.4_MeetingAbstracts.675A
- Pantell RH, Goodman BW (1981) Takayasu's arteritis: the relationship with tuberculosis. *Pediatrics* 67:84–88
- Modi G, Modi M (2000) Cold agglutinins and cryoglobulins in a patient with acute aortoarteritis (Takayasu's disease) and tuberculous lymphadenitis. *Rheumatology* 39:337–338
- Yang M-C, Yang C-C, Chen C-A, Wang J-K (2013) Takayasu arteritis presenting with acute heart failure. *J Am Coll Cardiol* 61:1302. <https://doi.org/10.1016/j.jacc.2012.09.070>
- Al-Aghbari K, Al-Motarreb A, Askar F (2010) Takayasu's arteritis associated with tuberculosis in a young Yemeni woman. *Heart Views* 11:117–120. <https://doi.org/10.4103/1995-705X.76804>
- Bajjal R, Chogle A, Kumar P, Shah N, Kulkarni S, Doshi S, Gupta D, Amarapurkar D (2015) A case of tuberculous colitis with associated Takayasu's arteritis. *J Assoc Physicians India* 63:62–65
- Moura C, Aquino MA, Rocha Filho J, Santiago M (2015) Takayasu's or tuberculous arteritis? *BMJ Case Rep*. <https://doi.org/10.1136/bcr-2014-208717>
- Khemiri M, Douira W, Barsaoui S (2016) Co-occurrence of Takayasu's arteritis and tuberculosis: Report of a Tunisian pediatric case. *Ann Pediatr Cardiol* 9:75–78. <https://doi.org/10.4103/0974-2069.171398>
- Walters HM, Aguiar CL, MacDermott EJ, Adams A, Barinstein L, Dayton JD, Salvatore C, Thimmappa N, Lehman TJ (2013) Takayasu arteritis presenting in the context of active tuberculosis. *J Clin Rheumatol* 19:344–347. <https://doi.org/10.1097/RHU.0b013e31829ce750>
- Duzova A, Türkmen O, Cinar A, Cekirge S, Saatci U, Ozen S (2000) Takayasu's arteritis and tuberculosis: a case report. *Clin Rheumatol* 19:486–489
- Kontogiannis V, Dalziel KL, Powell RJ (2000) Papulonecrotic tuberculide and stenosis of the abdominal aorta. *Rheumatology* 39:205–208
- Zaki S, Shanbag P, Chavan V (2011) Unusual presentation of Takayasu's arteritis as posterior reversible encephalopathy syndrome. *Ann Indian Acad Neurol* 14:214. <https://doi.org/10.4103/0972-2327.85900>
- Mukherjee D, Niyogi P (2013) Active tuberculosis with Takayasu arteritis. *Indian Pediatr* 50:349–350
- Wensing G, Kirch W, Ohnhauss EE (1987) Patientin mit Takayasuarteritis und protein C- sowie AT III-Mangel. *Klin Wochenschr* 65:752–756. <https://doi.org/10.1007/BF01736813>
- Sivashanmugam S, Gopalan V, Radhakrishnan AR, Sukumar P, Baskar N, Radhakrishnan N, Prasad PV, Ramamurthy A (1981) Tuberculous aorto-arteritis. A case report. *Indian Heart J* 33:73–77
- Ahasan HAMN, Alam B, Chowdhury MH, Mohammed FR, Nur Z (2009) Takayasu's arteritis in association with tuberculosis in a young woman. *Pak J Med Sci* 25:1009–1011
- Reshkova V, Kalinova D, Rashkov R (2016) Takayasu's arteritis associated with tuberculosis infections. *J Neurol Neurosci* 7:114. <https://doi.org/10.21767/2171-6625.1000114>
- Minozzi S, Bonovas S, Lytras T, Pecoraro V, González-Lorenzo M, Bastiampillai AJ, Gabrielli EM, Lonati AC, Moja L, Cinquini M, Marino V, Matucci A, Milano GM, Tocci G, Scarpa R, Goletti D, Cantini F (2016) Risk of infections using anti-TNF agents in rheumatoid arthritis, psoriatic arthritis, and ankylosing spondylitis: a systematic review and meta-analysis. *Expert Opin Drug Saf* 15:11–34. <https://doi.org/10.1080/14740338.2016.1240783>
- Lim AY, Lee GY, Jang SY, Gwag HB, Choi SH, Jeon E-S, Cha H-S, Sung K, Kim Y-W, Kim SM, Choe YH, Koh W-J, Kim D-K

- (2016) Comparison of clinical characteristics in patients with Takayasu arteritis with and without concomitant tuberculosis. *Heart Vessels* 31:1277–1284. <https://doi.org/10.1007/s00380-015-0731-8>
30. Lupi-Herrera E, Sánchez-Torres G, Marcushamer J, Mispireta J, Horwitz S, Vela JE (1977) Takayasu's arteritis. Clinical study of 107 cases. *Am Heart J* 93:94–103
 31. Handa U, Mundi I, Mohan S (2012) Nodal tuberculosis revisited: a review. *J Infect Dev Ctries* 6:6–12
 32. Jain A, Misra DP, Ramesh A, Basu D, Jain VK, Negi VS (2017) Tuberculosis mimicking primary systemic vasculitis: not to be missed! *Trop Doct* 47:158–164. <https://doi.org/10.1177/0049475516687432>
 33. Gornik HL, Creager MA (2008) Aortitis. *Circulation* 117:3039–3051. <https://doi.org/10.1161/CIRCULATIONAHA.107.760686>
 34. Arnaud L, Cambau E, Brocheriou I, Koskas F, Kieffer E, Piette J-C, Amoura Z (2009) Absence of *Mycobacterium tuberculosis* in arterial lesions from patients with Takayasu's arteritis. *J Rheumatol* 36:1682–1685. <https://doi.org/10.3899/jrheum.080953>
 35. Aggarwal A, Chag M, Sinha N, Naik S (1996) Takayasu's arteritis: role of *Mycobacterium tuberculosis* and its 65 kDa heat shock protein. *Int J Cardiol* 55:49–55