Comprehensive magnetic resonance imaging evaluation in a cohort of 145 patients with Takayasu's arteritis. Proposal for a standardised and systematic reporting format

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Abstract

Objective

Takayasu's arteritis (TAK) is a rare disease affecting the aorta and its branches. Magnetic resonance imaging (MRI) can assess vascular lesions, disease progression, activity, and the impact on different organs. Comprehensive MRI study including whole-body angiography (WBA) and target-organ assessment is not performed in all patients with suspected TAK. The aim of this study is to evaluate vascular and organic lesions in a cohort of patients with TAK who underwent MRI and to propose a standardised and systematic reporting format of the findings.

Methods

Patients with ≥4 TAK criteria who underwent MRI between 2003-2024, were included. With a standardised format, angiographic analysis of the presence and type of vascular lesion by segment was performed, including cerebral, cardiac, lower limbs, and visceral arteries, as well as the impact on the target organ.

Results

A total of 145 patients with mean age 36±15 years, 128 (88%) women were included. Type V of the angiographic classification was the most frequent. Involvement of cerebral 27 (18%), coronary 12 (8.2%), pulmonary 25 (17%), right renal 50 (34%) and left 42 (39%), lower limb arteries 48 (33%), stroke 27 (19%) was found. Cardiac MRI was done in 108 patients, any cardiac abnormality 70 (65%), myocardial infarction 10 (9.3 %), aortic regurgitation 26 (17%), mitral regurgitation 14 (8.3%) and complete AV-Block 1 (0.7%) was found.

Conclusion

Comprehensive assessment of patients with TAK by MRI with a standardised and systematic reporting format is a tool that may allow better interdisciplinary communication in the study of this vasculitis.

Key words

Takayasu's arteritis, systematic report of images, magnetic resonance comprehensive approach, organ damage

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Introduction

Takayasu's arteritis (TAK) is a rare chronic inflammatory disease, characterised by the development of progressive granulomatous arteritis, which mainly affects the aorta and its branches (1, 2). TAK is more frequent in the Asian continent, although in Latin American countries its diagnosis is increasing (3-5). Annual incidence of TAK in North America is 2.6 cases per million (6). In Mexico TAK is a common vasculitis, although its actual incidence is unknown. Between 80–90% of TAK patients are women, most of them in the second or third decade of life, although it can also affect the paediatric population (3, 7-9).

The aetiology is still unknown, however, in genetically predisposed individuals a chronic inflammatory process is present, probably secondary to antigen deposition in the arterial wall (10, 11). The adventitia shows the earliest histological changes, progressing to panarteritis, with mononuclear infiltration and granulomas, proliferation of the intima, as well as fibrosis and vascularisation of the media with rupture and degeneration of the internal elastic lamina. These alterations lead to the development of different types of arterial lesion (12-14).

Initial symptoms are non-specific and correspond to the inflammatory phase (15), posteriorly symptoms depend on the involved artery and the type of lesion (stenosis, occlusion and/or dilatation).

State of the art of non-invasive imaging techniques, including ultrasound, computed tomography angiography (CTA), FDG-18 PET-CT and magnetic resonance imaging (MRI) (16, 17), are used for integral diagnosis and follow up in TAK patients.

MR characterises the different types of vascular lesion, its progression, as well as the thickening of the vascular wall, allowing to evaluate the activity of the disease, as well as the treatment response (17, 18). In addition, MRI can assess the repercussion of the vascular injury on the different organs (heart, brain, kidney, etc). Cardiac MR (CMR) assesses the cardiac chamber diameters and volumes, wall thickness, segmental systolic thickening, systolic function, and the presence and type of late enhancement (LE).

Some authors have described the findings of MR angiography and its correlation with disease activity (19, 20). However, few studies include information on vascular and non-vascular damage. Current guidelines, evaluated through systematic reviews, provide information on treatment and ideal follow-up imaging studies. However, some patients present with organic damage in the brain, heart, or lower extremities. The arteries supplying these organs are not considered to be fully evaluated from the early stages of the disease. Thus, when an ischaemic or haemorrhagic cerebral event occurs, or cardiac or severe damage to the lower extremities occurs, it is unknown whether the condition of the arterial supplying them has been progressive or is of recent onset. It is highly likely that if a comprehensive magnetic resonance study is available from the onset of the disease when the diagnosis is made, this could allow us to elucidate whether the changes occurred acutely or are chronically progressive over time (21, 22).

This study aimed to describe the findings of vascular and organic lesions using a standardised and systematic reporting format in patients with TAK and to propose the worldwide use of this tool to allow effective communication between all professionals involved in the evaluation, treatment, follow-up and research of this vasculitis.

Methods

This prospective study of a cohort with TAK who underwent MRI between January 2003 and September 2024, at the request of their treating physician.

All patients fulfil ≥ 4 criteria of the American College of Rheumatology (ACR) classification (23). In the case of paediatric patients, PRINTO criteria were used (24). All patients also fulfil absolute requirements of the 2022 ACR/European Alliance of Associations for Rheumatology (EULAR) Classification criteria for TAK (25). Patients were evaluated by a rheumatologist in order to assess these criteria as well as activity according to Kerr and Dabague (26). Cardiovascular risk

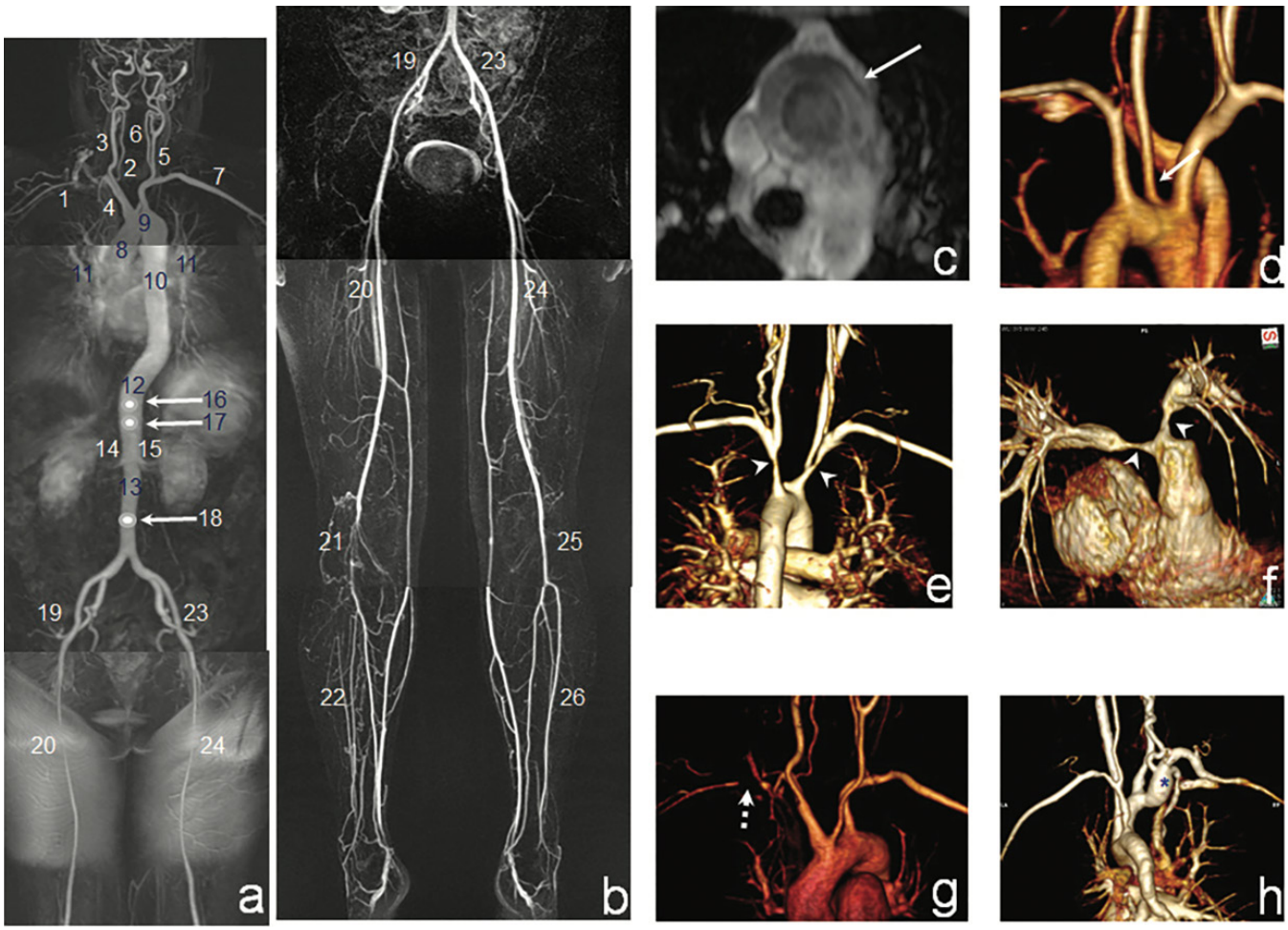

Fig. 1. Localisation and type of lesion. Volumetric reconstructions of angio-resonance, in (a) from the cerebral to the femoral arteries and in (b) from the bifurcation of the aorta to the distal segments of the lower limb's arteries. 1) Right subclavian; 2) Right vertebral; 3) Right common carotid; 4) Brachiocephalic trunk; 5) Left common carotid; 6) Left vertebral; 7) Left subclavian; 8) Ascending aorta; 9) Aortic arch 10) Descending thoracic aorta; 11) Pulmonary artery; 12) Supra renal abdominal aorta; 13) Infra renal abdominal aorta; 14) Right renal artery; 15) Left renal artery; 16) Celiac trunk; 17) Superior mesenteric; 18) Inferior mesenteric; 19) Right iliac artery; 20) Right femoral; 21) Right popliteal; 22) Right tibial/peroneal; 23) Left iliac artery; 24) Left femoral; 25) Left popliteal; 26) Left tibial/peroneal (Table II).
Images c-h are axial (c) and volumetric reconstructions showing the different types of arterial lesions. Wall thickening (c) of the ascending aorta (arrow). d) Non-significant stenosis (<50%) of the left common carotid artery (arrow). In (e) and (f) significant stenosis (≥50%): in e) the arrowheads indicate the significant stenosis of both subclavian arteries and in (f) of both pulmonary branches. In g) occlusion of the right subclavian artery (dashed arrow) and in h) dilatation (*) of the right subclavian artery.

factors and symptoms or signs of organ involvement were obtained from medical records. Exclusion criteria were contraindications to MRI (unsafe pacemaker or implantable cardioverter defibrillator, claustrophobia, etc.) or an estimated glomerular filtration rate <30 mL/min/1.73 m^2 BSA.

This study was approved by the medical data authority through the Research and Ethics Committee of the National Institute of Cardiology Ignacio Chavez, with number 13-800.

Study population

This prospective, dynamic cohort study included male and female patients any age, all diagnosed with TAK, regardless of disease duration. The Research Committee approved the study to evaluate outcomes over time. Regardless of the time of diagnosis, patients who experienced acute or recent symptoms of ischaemic or haemorrhagic cerebrovascular disease, as well as angina or acute myocardial infarction, were included. The study was performed based on the ordering physician's recommendation. In some patients, in addition to brain or cardiac imaging, a complete MRI was also considered to assess the patient's overall condition.

The diagnosis of cerebral ischaemic or haemorrhagic events was determined and evaluated by a neurologist and classified by imaging. Typical angina is present if patients report precordial chest pain or discomfort; caused by exertion or emotional stress; and relieved with rest and/or nitro-glycerine. Atypical angina was defined if the patient had chest pain or discomfort lacking one of the characteristics of typical angina (27).

The time of evolution refers to the time elapsed between symptom onset and the MRI. This prospective study included only MRI studies ordered during the study period. These studies could be the first or consecutive. When more than one was ordered, the

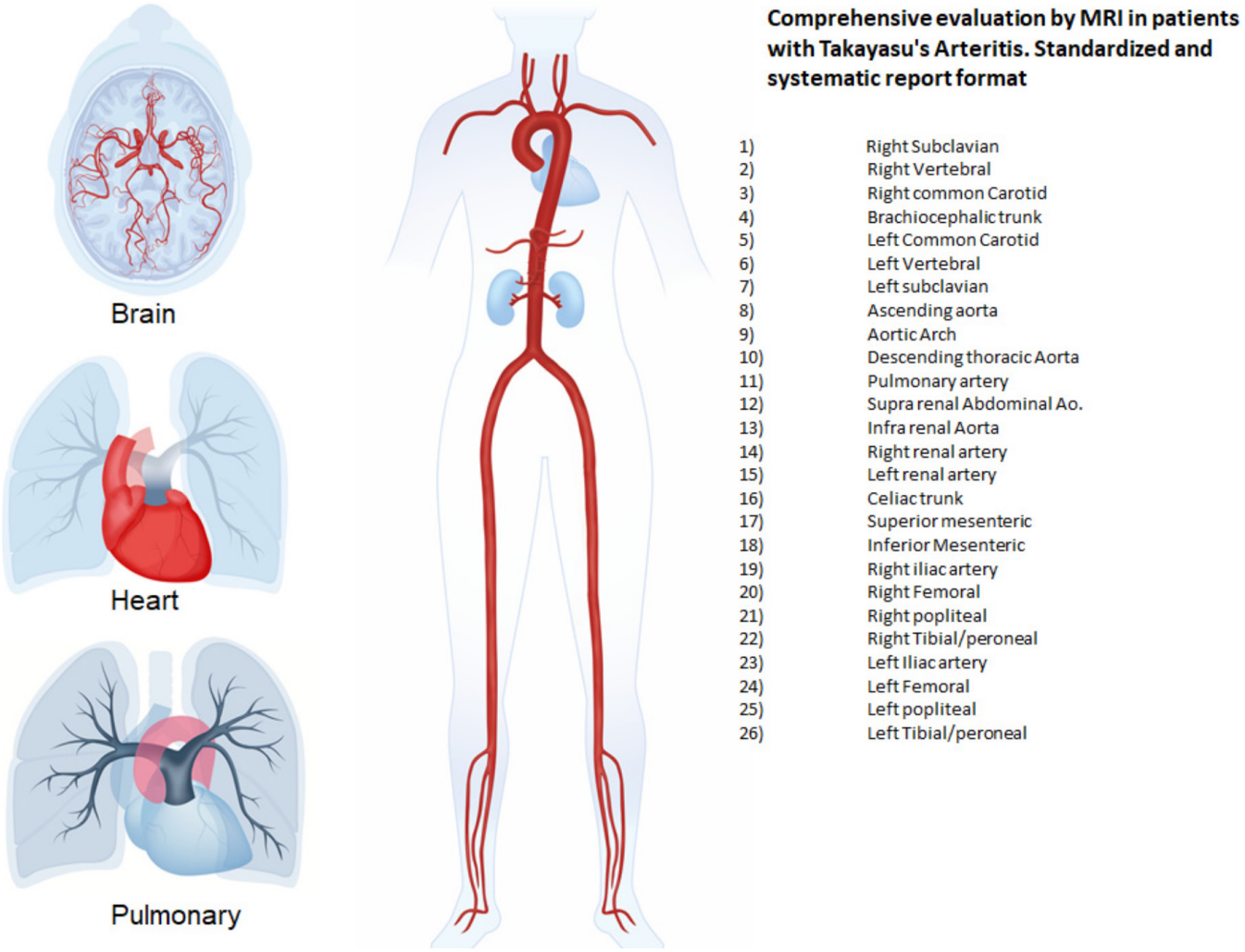

Fig. 2. Comprehensive MRI evaluation in patients with TAK shows a standardised and systematic reporting format that should include the presence and type of arterial lesion in each segment, listed in this chart and identified in Figure 1. Classify them as nonsignificant and significant (<50% and ≥50%), occlusion (total stenosis of 100%) and dilation (when increase ≥50% of normal diameter); as well as wall thickness at different levels of the aorta (in mm). The complete MRI should include the status of the cerebral, coronary, renal, and limb arteries.

one that included the greatest number of regions with arterial damage was considered.

Magnetic resonance

MR studies were performed on 1.5 Tesla MR system (Magnetom Sonata and Magnetom Avanto Siemens Medical Solutions, Erlangen, Germany). In cases where whole body angiography (WBA) was requested, the acquisition included from the cerebral to the tibial arteries. Imaging was performed using phased-array head, neck, chest, abdominal and extremity coils. MR angiography (MRA) was acquired after the administration of gadolinium-based contrast (0.15 mmol/kg, Gadovist, Bayer Schering Pharma) followed by 20 mL of saline chaser, both at a rate of 3 mL/s). Images were acquired in breath-hold in two blocks, 10 seconds for thorax and 12 seconds for abdomen and lower extremities. The parameters were as follow: TR/TE 2.6/0.8, flip angle 25°, FOV 500×320 mm, acquisition matrix 448×140, reconstruction matrix 670, acquisition voxel size 2.2 x 1.1 x 1.6 mm, slice thickness 1.5 mm. In cases of thoracic-abdominal angiography only chest and abdominal coils were used. Axial T1 weighted fat saturated post contrast images were acquired to measure wall thickness.

The CMR protocol included functional study using ECG-triggered breath-hold segmented steady-state free precession (SSFP) cine images in long and short axis views, with the following parameters: TR 62 msec, TE 1.2 msec, flip angle 55°, slice thickness of 8 mm, between 25 and 30 frames per cycle. In order to assess LE, breath-hold ECG-triggered 2D inversion-recovery (I-R) gradient echo sequences (TR 880 mseg/TE 1.1 msec; flip angle 40°) were acquired between 10 and 15 min after the administration of gadolinium in the long and short-axis projections according to the standardised protocols (28, 29).

Brain MRI protocol at our institution consists of T1, T2, FLAIR, T2*, time of flight MR angiography (MRA) to evaluate the intracranial circulation, and diffusion weighted (DWI) sequences (30).

In 68 patients (46.8%) WBA, cardiac and brain MR were done, in 40 (27.6%) thoraco-abdominal MRA and CMR, 26 (17.9%) thoracic and abdominal angi-

ography and 11 (7.6%) brain and thoracic-abdominal angiography.

Image analysis

Evaluation of the aorta and its main branches, including lower limbs arteries was performed. These were reported as 26 different segments (Fig. 1).

According to the angiography results, each segment was classified as with or without lesion. Vascular lesion included, wall thickening (wall thickness >3 mm) (20) non-significant and significant stenosis (<50% and ≥50%), occlusion and dilatation (increase ≥ 50% of normal diameter) (Fig. 1). When more than one type of lesion was found in a segment, the one with the greatest repercussion was recorded (occlusion on significant stenosis).

According to magnetic resonance angiography findings, patients were categorised using the Hata angiographic classification (31). Coronary involvement was recorded when it had been documented by invasive angiography or computed tomography, or when magnetic resonance imaging showed LE with an ischaemic pattern.

Cardiac functional parameters and mass were calculated using a specialised software (Leonardo and Argus, Siemens Medical Systems, Erlangen, Germany). The analysis of the left ventricle (LV) was done using the American Heart Association's segmentation (32). The presence of any myocardial LE was visually determined and the predominant pattern was recorded as follows: 1) subendocardial, 2) transmural (≥50% wall thickness, involving the endocardium) 3) mid wall <50% wall thickness, 4) mid wall ≥50% wall thickness or 5) subepicardial (33).

MR images were evaluated by consensus by a cardiologist and a radiologist with 9 years of experience in cardiovascular imaging, unaware of patient clinical information. All CMR measurements were done according to the current guidelines. (34, 35).

If a stroke was detected, the report included whether it was acute or chronic, as well as the affected vascular territory (36).

Figure 2 shows a proposal for the comprehensive evaluation of MRI in patients with TAK through a standardised and systematic report format.

Statistical analysis

Continuous variables were expressed as mean ± SD or as median and interquartile range (according to their distribution) and categorical variables as percentages for descriptive variables. This study does not require comparative tests or optimal scaling studies such as logistic or multivariate regression. Statistical analysis was performed using SPSS version 22.0 (SPSS, Inc., Chicago, IL).

Results

A total of 145 patients were included, 128 (88%) were women, the mean age was 36±15 years. Demographic and clinical data are shown in Table I. Hypertension was the most frequent comorbidity while claudication and headache were the most frequent symptoms.

Whole-body angiography (WBA) included all blood vessels from the brain to the extremities, and cardiac and brain magnetic resonance imaging (MRI) was performed in 68 patients (46.8%). Thoracoabdominal angiography and cardiac MRI were performed in 40 patients (27.6%).

Therefore, we were able to study 108 patients with CMR, as the two previous studies included evaluation of this region. Thoracic and abdominal angiography was performed in 26 patients (17.9%), and cerebral and thoracoabdominal angiography in 11 (7.6%). Thus, we were able to evaluate the brain in 79 patients: the 68 who underwent WBA and the 11 patients who underwent thoracoabdominal angiography and brain MRI.

The percentage of patients with arterial or target organ abnormalities not included in the guide was high: in the brain 27 of 79 (34%), in the heart 34 of 108 (31%) and in lower limb arteries 48 of 68 (70%).

According to the angiographic classification, type V was the most frequent (67% isolated or combined). Coronary and pulmonary involvement was documented in 12 (8%) and 25 (17%) of the patients, respectively.

Table II shows presence and type of arterial lesion. The arteries most frequently involved were the subclavian, with occlusion as the most common type of lesion in these vessels. Globally, non-significant stenosis and occlusion were the most frequent types of lesions. Dilation affected 97 (2.6%) segments, mainly the brachiocephalic trunk and ascending aorta. Wall thickening was found mainly in the ascending aorta, aortic arch and descending thoracic aorta.

Forty-eight patients out of 68 patients (70%) in whom lower limb angiography was performed had at least one affected lower limb artery, with a median age of 40 years (13-64) and a time of evolution of the disease of 14 years (2-46) (Table III). Of the 48 patients, 37 had single vessel involvement: iliac, femoral or tibial (unilateral or bilateral). In 11 patients, more than one site was affected (Fig. 3e). Lesions that affected the distal arteries, without affecting the iliac were observed in 9 patients.

Twenty-seven out of 79 (34%) in whom brain MRI was performed had

Table I. Demographic and clinical characteristics n=145.

Age (years)	36 ± 15
Female gender (%)	128 (88)
Hypertension (%)	77 (53)
Renal failure (%)	18 (12)
Stroke (%)	27 (19)
Myocardial infarction (%)	10 (7)
Symptoms	
Headache (%)	67 (46)
Angiodynia (%)	35 (24)
Cataract (%)	7 (5)
Amaurosis (%)	24 (17)
Dizziness (%)	45 (31)
Claudication (%)	66 (46)
Upper limbs (%)	46 (32)
Lower limbs (%)	53 (37)
Angiographic classification (Hata)[27]	
I	15 (10)
I+P	2 (1)
I+P+C	1 (0.7)
IIa+P	4 (2.8)
IIa+C	1 (0.7)
IIb	8 (5.5)
IIb+P	3 (2)
III	1 (0.7)
IV	11 (8)
V	75 (52)
V+P	13 (9)
V+C	9 (6.2)
V+P+C	1 (0.7)

C: coronary involvement (12 patients); P: pulmonary involvement (25 patients).

Table II. Frequencies and type of arterial lesion in the aorta and its main branches.

Arterial lesion	Without lesion	With lesion	Non-significant stenosis	Significant stenosis	Occlusion	Dilatation	Wall thickening
1) Right subclavian	70 (48)	75 (52)	15 (10)	15 (10)	34 (23)	10 (7)	1 (0.7)
2) Right vertebral	129 (89)	16 (11)	5 (3.4)	3 (2)	6 (4)	2 (1.4)	-
3) Right common carotid	82 (57)	63 (43)	11 (8)	19 (13)	20 (14)	8 (5)	5 (3)
4) Brachiocephalic trunk	74 (51)	71 (49)	14 (9.5)	19 (13)	16 (11)	20 (14)	2 (1.4)
5) Left common carotid	75 (52)	70 (48)	23 (16)	21 (14.5)	19 (13)	2 (1.4)	5 (3.4)
6) Left vertebral	124 (86)	21 (14)	9 (6.2)	3 (2)	8 (5.5)	1 (0.7)	-
7) Left subclavian	46 (32)	99 (68)	21 (14)	21 (14)	52 (36)	4 (3)	1 (0.7)
8) Ascending aorta	105 (72)	40 (28)	5 (3.4)	1 (0.7)	-	15 (10)	19 (13)
9) Aortic arch	111 (77)	34 (23)	7 (4.8)	-	-	6 (4)	21 (15)
10) Descending thoracic aorta	93 (64)	52 (36)	19 (13)	5 (3.4)	3 (2)	7 (5)	18 (12)
11) Pulmonary artery	120 (83)	25 (17)	11 (7.6)	10 (6.9)	1 (0.7)	3 (2.1)	-
12) Supra renal abdominal Ao.	95 (65)	50 (35)	23 (16)	5 (3.4)	2 (1.4)	7 (4.8)	13 (9)
13) Infra renal aorta	83 (57)	62 (43)	31 (21)	11 (7.6)	6 (4.1)	4 (2.8)	10 (6.9)
14) Right renal artery	95 (66)	50 (34)	17 (12)	27 (19)	6 (4)	-	-
15) Left renal artery	103 (71)	42 (39)	9 (6)	22 (15)	10 (7)	1 (0.7)	-
16) Celiac trunk	115 (79)	30 (21)	13 (9)	8 (5.5)	5 (3.4)	4 (3)	-
17) Superior mesenteric	119 (82)	26 (18)	4 (3)	12 (8)	10 (7)	-	-
18) Inferior mesenteric	144 (99)	1 (1)	-	-	-	1 (0.7)	-
19) Right iliac artery	111 (77)	34 (23)	15 (10.3)	8 (5.5)	11 (8)	-	-
20) Right femoral	132 (91)	13 (9)	2 (1)	6 (4)	5 (3)	-	-
21) Right popliteal	141 (97)	4 (3)	-	2 (1.4)	2 (1.4)	-	-
22) Right tibial/peroneal	141 (97)	4 (3)	1 (0.7)	1 (0.7)	2 (1.4)	-	-
23) Left iliac artery	115 (79)	30 (21)	11 (7.6)	7 (4.8)	11 (7.6)	1 (0.7)	-
24) Left femoral	135 (93)	10 (7)	1 (0.7)	4 (2.8)	4 (2.8)	1 (0.7)	-
25) Left popliteal	143 (99)	2 (1)	-	1 (0.7)	1 (0.7)	-	-
26) Left tibial/peroneal	141 (97)	4 (3)	1 (0.7)	2 (1.4)	1 (0.7)	-	-
Total	2842 (75)	928 (25)	268 (7.1)	233 (6.1)	235 (6.2)	97 (2.6)	95 (2.5)

Table III. Frequency of arterial injury in lower limbs with lesion in one or more vessels.

			Angiographic classification (Hata)														
Type of injury in lower limbs	Age	Evolution years	I	I+P	1+P+C	IIa+P	IIa+C	IIb	IIb +P	III	IV	IV+P	V	V+P	V+C	V+P+C	Total
Iliac (I)	40 (13-59)	13 (2-46)	--	--	--	--	--	1	1		4	1	16	5	2	--	30
Femoral (F)	21 (15-37)	12 (10-22)	1	--	--	--	--	--	--	--	1	--	2	--	--	--	4
Tibial (T)	40 (35-50)	19 (18-23)	--	1	--	--	--	--	--	--	--	--	1	--	--	1	3
One vessel																	37
I+F+Poplitea (P)	64	10	--	--	--	--	--	--	--	--	--	--	1	--	--	--	1
I+F	45 (18-61)	16 (7-30)	1	--	--	--	--	--	--	--	--	--	3	2	1	--	7
F+P+T	41 (41-42)	3 (3-3)	1	--	--	--	--	--	--	--	--	--	1	--	--	--	2
I+F+T	42	24	--	--	--	--	--	--	--	--	--	--	--	--	1	--	1
More than one vessel																	11
Total	40 (13-64)	14 (2-46)	3	1	--	--	--	1	1	--	5	1	24	7	4	1	48

stroke (Table IV), 17 of the 66 women (25.7%) and 10 of the 13 men (76.9 %), the median age and time of evolution was 31 years (ICR 24-44) and 15 months (ICR 6-26 months) respectively. Stroke as the first manifestation of TAK occurred in 8 (29.6%) patients. Nineteen (70%) of the patients with stroke had a type V lesion of the angiographic classification; 3 type I, 2 type IIa and 2 IIb; only 1 patient had type IV lesion with involvement of the right middle cerebral artery. In 5 of the 27 patients (19%) direct involvement of the cerebral arteries was documented (Fig. 3 a-c).

In 70 out of the 108 (65%) patients in whom CMR was performed at least one cardiac abnormality was found (Table V, Fig. 3f, 4d, 4g).

Thirty-four patients had alteration in cardiac geometry, twenty-three patients had LV systolic dysfunction of which 6 had myocardial infarction, and 8 ventricular dilatation.

Thirty-eight patients had valvular lesion, most of them (30/38) affecting only one valve. Regurgitation was the most common valvular lesion, involving the aortic valve in 24 patients (22.2%) (Fig. 4d), two more patients had double lesion. Isolated mitral regurgitation was documented in 12 pa-

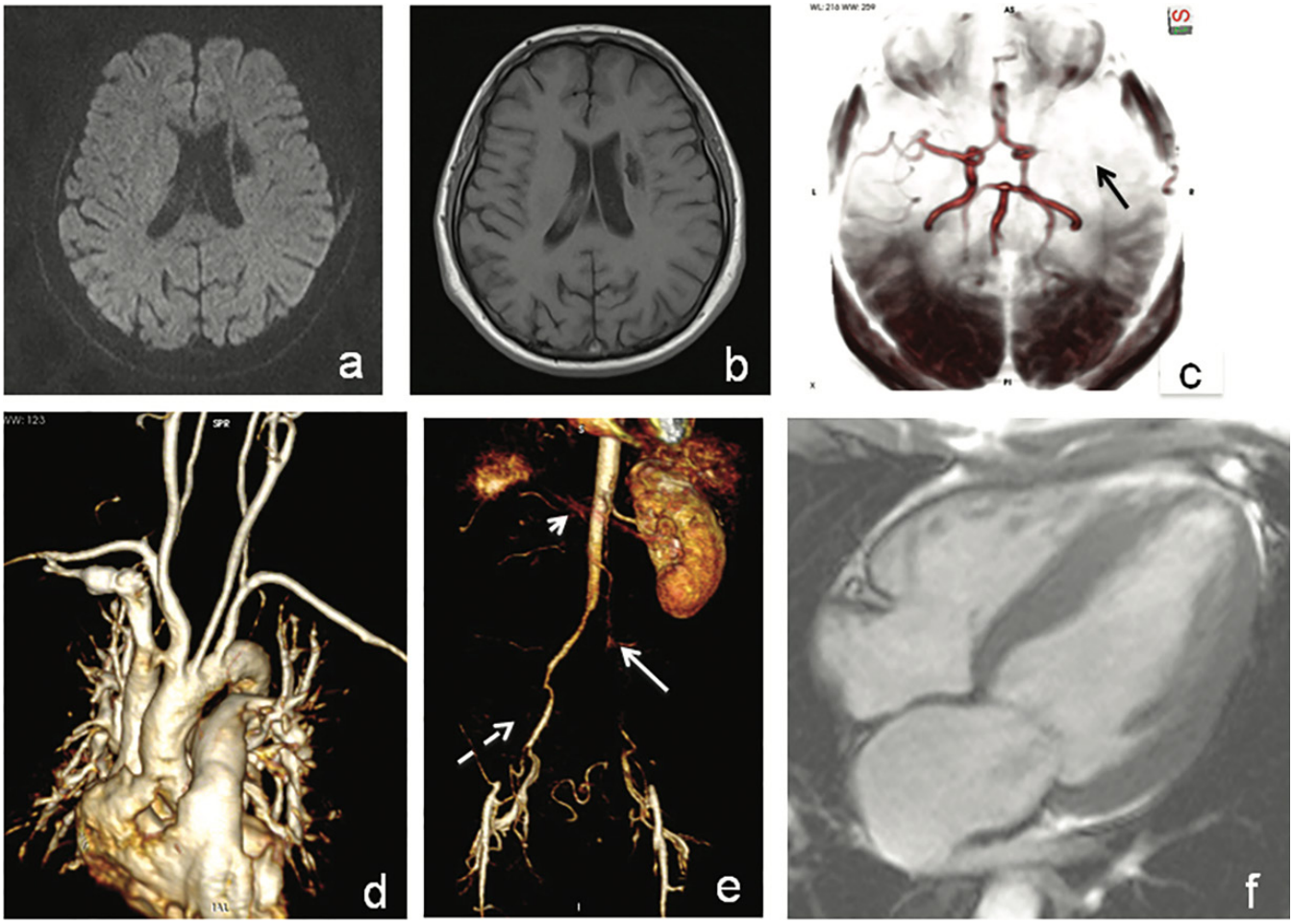

Fig. 3. 29-year-old male with type IV TAK of the angiographic classification. DWI sequence (**a**) and T1 (**b**), show an area of encephalomalacia adjacent to the left lateral ventricle corresponding to a chronic ischaemic vascular event. The cerebral MRA(**c**) shows lack of signal from the left middle cerebral artery (black arrow). Angiographic reconstructions (**d**, **e**) show normal diameter and morphology of the thoracic-abdominal aorta and supra-aortic vessels. The right renal artery (arrowhead) is not observed with corresponding renal atrophy. Occlusion of the left common iliac artery and right external iliac artery (white dashed arrow and arrow). True FISP sequence in 4 chambers in systole (**f**) showing concentric hypertrophy and systolic dysfunction of the left ventricle. LVEF 36% and mitral regurgitation.

tients (11%) and tricuspid regurgitation in 9 patients.

Aortic dilatation was more frequent in patients who presented moderate or severe aortic regurgitation than in those who did not, this showed significant statistical differences 9/10 (90%) versus 7/94 (7 %) (p=0.0001). Thirteen of the 16 (81%) patients with moderate or severe aortic regurgitation had wall thickening, versus 47/94 (62%) those without regurgitation (p=0.16).

Pericardial effusion was documented in 8 patients and one patient had complete A-V block as a first manifestation (37).

Inversion recovery sequence was performed in 83 patients, LE was demonstrated in 46 (55%); 9 patients with transmural LE (ischaemic pattern) and mid-wall LE in 37 (80%) (Table V, Fig. 4g).

According to Kerr criteria activity was found in 76% of the patients and by MRI (wall thickening >3 mm) in 59%, with concordance between them of 50% (p=0.001).

Discussion

Comprehensive imaging evaluation is essential in patients with TAK since this pathology can affect not only vessels but also several organs. Therefore, it is essential to systematise the acquisition and analysis of images since the diagnosis.

Angiography

This prospective study included 145 Mexican patients with TAK; according to the angiographic classification, we found that most of patients were type V and the supra-aortic vessels, mainly the subclavian arteries, were the most affected, which is similar to that found in the Chinese population (38).

The prevalence of brachiocephalic trunk affection ranges from 8.8 to 46.8% (20, 39-44). In our study 49% of patients had any type of the lesion of this vessel with dilatation in 14%. Superior mesenteric artery (SMA) involvement was 18%. Patients with TAK may complain of non-specific abdominal pain, which is a challenge for the physician, the differential diagnosis should include the possibility of an occlusive mesenteric event where imaging findings would allow prompt diagnosis and improve the prognosis (44-46). In the Mirouse study (47), mesenteric ischaemia was the cause of 4 of the 16 deaths (25%).

In our series the involvement in renal

Table IV. Clinical and arterial characteristics of patients with stroke.

	Age years	G	Time months	Hata	Arterial lesion					Stroke location
					Occlusion	Significative stenosis	Non significative stenosis	Wall thickening	Dilatation	
1	16	M	2	IIb	Left vertebral, middle cerebral artery		Left, common carotid (CC), descending thoracic aorta (DTA)			Left basal nuclei and caudate nucleus.
2	17	F	5	V*	Right, CC Right and Left Subclavian (SC)	Left CC, right renal, Celiac trunk				Lacunar infarction in right centrum semiovale
3	19	F	14	V	Left SC	Right SC	Right CC, DTA Right and left renal			Right parietal lobe.
4	21	M	6	IIb	Supra diaphragmatic Ao		Left subclavian artery		Basilar artery aneurysm.	Right putamen.
5	22	F	10	V +P	Left CC	Left Pulmonary, Left renal	Right CC, SC, celiac trunk	DTA		Left insular cortex.
6	23	M	5	I	Right and Left CC	Left middle cerebral artery	Right SC			Left parieto-temporal
7	24	F	26	V+C+LL	Right and left SC	Right and left Vertebral, Right CC, iliac, femoral, Left Tibial	DTA, right renal		Brachiocephalic trunk (BCT)	Left occipital lobe.
8	25	F	8	V		Right and left CC, left Vertebral, left SC, suprarenal aorta.				Left middle cerebral artery territory.
9	26	M	7	V	Left SC	Left renal, celiac trunk	Right renal	Ascending Aorta	Aortic root	Left parietal lobe and external capsule
10	28	M	15	I + LL	Right and left CC, right SC, right femoral.		Left SC, vertebral and femoral			Right frontal and parietal infarct.
11	29	M	2	IV (iliac)	Right renal, right and left iliac, right mid. cerebral.					Right frontal and left parietal lobes.
12	30	F	72	V	Right CC, left SC, Celiac trunk, left renal				Abdominal Aorta	Right basal nuclei.
13	30	M	30	V	Supra-renal aorta		Right renal		BCT, right SC	Right internal capsule.
14	31	F	10	V	Left SC	Left CC	Right SC and carotid, abdominal Ao			Left temporal, parietal, and frontal lobes.
15	31	M	15	V+P+LL		Right and left CC, right SC, desc. Thoracic Ao, right iliac, left pulmonary	Left SC, Abdominal Aorta			Right frontal and parietal lobes.
16	33	F	156	V	Right CC		Aortic Arch, Infra-renal Ao.			Right frontal, parietal and temporal lobes.
17	34	F	16	IIa +P	Right and left CC, right and left SC	Right and left pulmonary				Left frontal lobe.
18	34	F	24	V	.	Left renal			Aortic root, Ascending aorta, aortic Arch, DTA, supra-renal Aorta, BCT and left SC	Right parietal lobe.
19	40	F	27	V + LL	Right SC Right tibial and popliteal	Infra-renal Ao. and left renal artery	Right femoral, Celiac Trunk, superior Mesenteric, right renal		Inferior mesenteric, right and left pulmonary	Bilateral parietal lobes.
20	41	F	18	V	Left CC	BCT, right CC, right renal	Infra-renal Aorta		Right SC	Right frontal and parietal, Left parietal lobes.

	Age years	G	Time months	Hata	Arterial lesion					Stroke location
					Occlusion	Significative stenosis	Non significative stenosis	Wall thickening	Dilatation	
21	44	F	15	IIa +P	Left CC and right pulmonary		Left SC	Right CC	Abdominal aorta, BCT	Left temporal lobe.
22	50	F	25	I	Right CC, right and left SC					Right frontal, parietal and temporal lobes.
23	52	M	40	V	BCT, right and left CC, right and left SC, Infra-renal aorta	Descending thoracic Ao	Left vertebral			Cerebellum.
24	53	F	1	V+ LL	Left SC, left renal, right and left iliac			Ascending Aorta		Right and left parietal lobes.
25	59	F	5	V+P+LL			Right SC, desc T Ao, Ab. Ao left renal, left pulmonary left iliac.			Multiple lacunar infarcts.
26	60	M	20	V+C+LL	Middle cerebral, Infra-renal Aorta, left renal, right, superior mesenteric, right and left iliac, left femoral					Right frontal lobe.
27	66	F	51	V	Right and left CC left SC	Right renal		Infra-renal Aorta	BCT and right SC	Right occipital lobe.

Ao: aorta; BCT: brachiocephalic trunk; CC: common carotid; C-T: celiac-trunk; DTA: descending thoracic aorta; F: female; G: gender; L: lower limb; M: male; SC: subclavian.

arteries was observed in 34% and 39% (right and left respectively), with significant stenosis as the most frequent type of lesion. According to the study by Chen *et al*., the involvement of the renal arteries confers higher risk of severe cardiac and renal dysfunction compared to those without (48).

Pulmonary artery affection sometimes is the initial and the only manifestation in TAK patients, which makes the early diagnosis difficult (49, 50). In this series the involvement of the pulmonary arteries was documented in 17% of the patients. In the literature, the results are highly variable, which could be explained by the imaging methods used (CT angiography or MRA), as well as if the study is only requested in patients with pulmonary symptoms (51).

Claudication is one of the classification criteria for TAK and traditionally it is attributed to aortic and/or iliac affection, the peripheral vessels are not routinely assessed, despite the fact that catastrophic cases have been documented (52). In our study affection of lower limb arteries, was found in 48 out of 145 patients (33%), with a predominance in the female gender and median age of 40 years, claudication was found in 49% of them. The majority were type V of the angiographic classification; however, lower limb affection was also found in cases with type I and II, due to the systemic nature of this disease. The most frequently affected vessels were the iliac arteries, with a higher percentage than that reported in the literature (53). In 9 patients, peripheral lesions without iliac involvement were found, so the conventional MRA field of view that includes the supra-aortic vessels to the common iliac might not detect them.

Our findings in relation to lower limb lesions show that they are not necessarily exclusive of older patients or the time of evolution. In patients with TAK and claudication, it is important to determine if this is due to direct stenosis and/or occlusion of the peripheral vessels to define the treatment and improve the patient's symptoms and to avoid fatal outcomes.

Evaluation of wall thickness as a surrogate of activity has already been proposed, in this study we found a good concordance between the presence of wall thickening and the NIH activity criteria. Wall thickness at different levels of the aorta should be reported (in mm) which will allow the comparison in follow up studies and detect activity. Thickening of the arterial wall as the only finding that should alert in the diagnosis of TAK in the early stages even without occlusive involvement of the lumen of the vessel.

Brain

In our series stroke was found in 27 (19%) of the patients, which is higher than previously reported (54), with an increased proportion in men and also being the initial symptom in 30% of the cases. Most of the patients were type V of the angiographic classification, however one patient had type IV with right middle cerebral artery lesion. Direct involvement of the cerebral arteries was documented in 18% of the cases which is lower than that reported by Hwang *et al*. (36) who found intracranial artery stenosis in 11 (52%) of 21 patients with stroke suggesting that haemodynamic compromise in large-artery stenosis and thromboembolic mechanisms play significant roles in ischaemic stroke associated with TAK.

Table V. Cardiac abnormalities.

	n=108	Age (years)	Female	Angiographic classification [27]					
				I (n=11)	IIa (n=5)	IIb (n=11)	III (n=1)	IV (n=5)	V (n=75)
Cardiac abnormality	70 (65%)	33 (23.7-46.2)	60 (85.7)	3 (4.3)	4 (5.7)	8 (11.4)	1 (1.4)	3 (4.3)	51 (72.9)
Alteration in cardiac geometry					n=34/108				
Concentric remodelling	4 (3.7)	53 (31.5-61)	4 (100)	1 (25)	--	1 (25)	--	--	2 (50)
Concentric hypertrophy	7 (6.5)	21 (13-36)	4 (57.1%)	--	1 (14.3)	1 (14.3)	--	2 (28.6)	3 (42.9)
Eccentric hypertrophy	3 (2.8)	38 (23-44)	3 (100)	--	1 (33.3)	1 (33.3)	--	--	1 (33.3)
LV dilatation	12 (11.1)	25.5 (18.2-30)	10 (83.3)	--	--	1 (8.3)	--	--	11 (91.7)
RV dilatation	7 (6.5)	34 (29-44)	7 (100)	1 (14.3)	--	4 (57.1)	--	--	2 (28.6)
Biventricular hypertrophy	1 (0.9)	62	1	--	--	--	--	--	1
Systolic disfunction									
LV Mild (47-56)	6 (5.6)	25.5 (18-36.5)	5 (83)	--	--	--	--	--	6 (100)
LV Moderate (37-46)	7 (6.5)	30 (25-56)	4 (57.1)	--	--	--	--	1 (14.3)	6 (85.7)
LV Severe (<36)	10	28 (11-38)	7 (70)	--	--	1 (10)	--	1 (10)	8 (80)
Right Ventricle	6	33.5 (24.7-47)	6 (100)	--	--	4 (66.7)	--	--	2 (33.3)
Biventricular	3	19 (8-55)	3 (100)	--	--	--	--	--	3 (100)
Myocardial infarction	10 (9.3)	34.5 (23.5-49)	7 (70%)	--	--	--	--	--	10 (100)
Aortic regurgitation					**n= 26 (24)**				
Mild	8 (7.4)	37 (18-78)	7 (87.5%)	--	1 (12.5)	1 (12.5)	--	--	6 (75)
Moderate	7 (6.5)	27 (18-55)	7 (100)	1 (14.3)	1 (14.3)	--	--	--	5 (71.4)
Severe	9 (8.3)	28 (9-38)	7 (77.8)	--	--	--	--	--	9 (100)
Double lesion	2 (1.8)		2 (100)	--	--	--	--	--	-100
Mitral regurgitation					**n=14(12.9)**				
Mild	4 (3.7)	52 (8-61)	4 (100)	--	--	--	--	--	4 (100)
Moderate	4 (3.7)	33 (26-38)	2 (50)	--	1 (25)	--	--	1 (25)	2 (50)
Severe Mitral	4 (3.7)	33 (24-41)	4 (100)	--	--	--	--	--	4 (100)
Double lesion	2 (1.8)		2 (100)	--	--	--	--	--	2 (100)
Tricuspid regurgitation					**n=9 (8.3)**				
Mild	5 (4.6)	55 (22-63)	5 (100)	--	1 (20)	1 (20)	--	--	3 (60)
Moderate	3 (2.7)	43 (26-78)	3 (100)	--	--	1 (33.3)	--	--	2 (66.7)
Severe	1 (0.9)	44	1 (100)	1 (100)	--	--	--	--	--
Pericardial effusion									
Mild	7 (6.5)	34 (13-59)	7	--	1 (14.3)	2 (28.6)	--	--	4 (57.1)
Severe	1 (0.9)	43	1	--	--	1 (100)	--	--	0
A-V block	1 (0.9)	38	1 (100)	--	1	--	--	--	--
Late enhancement									
Transmural	9 (8.3)		6 (66.7)	--	--	--	--	1 (11.1)	8 (88.9)
Mild wall < 50%	31 (28.7)		30 (96.8)	1 (3.2)	4 (12.9)	4 (12.9)	--	1 (3.2)	21 (67.7)
Mid wall > 50%	6 (5.5)		5 (83.3)	--	--	2 (33.3)	--	--	4 (66.7)

Heart

Cardiovascular compromise was frequent, at least one cardiac abnormality was found in 65% of the patients in whom CMR was performed. Previous echocardiographic studies found that between 8.6 and 64.7% of TAK patients have cardiac involvement (55, 56).

Alterations in LV geometry were frequent and could be explained by systolic overload caused by stenosis in the aorta and its branches, valvular, and/or coronary involvement (56).

RV dilatation was found in 7 patients, secondary to left ventricular disease and/or direct involvement of the pulmonary arteries. Twenty percent of the patients had LV disfunction which is higher than that reported by Yang *et al*. (57) who in a retrospective echocardiographic study found left ventricular ejection fraction (LVEF) <50% in 11.6% of 493 patients.

The prevalence of valve damage ranges between 30 and 64% (55, 56). We found that 38% of patients had some type of valvular injury, aortic regurgitation was the most common, which was similar to other series (56). Moderate- severe aortic regurgitation was found in 16 out of 108 patients (15%) in our cohort.

Pericardial effusion was documented in 8 patients (7%) which was less than that reported by Li *et al*. (56). Exudative pericarditis is an extremely rare manifestation, but it has been reported as the first clinical manifestation of TAK (58).

LE was frequent (46 out of 108 patients), with non-ischaemic pattern (mid wall involved <50% of wall thickness) in 31 patients and 9 with ischaemic pattern. One of the explanations of the prevalence of mid wall LE could be the systolic overload due to aortic and/or its main branches involvement. The most frequent type of LE from our series differs to that reported in the literature (59).

Complete A-V block as the initial manifestation of TAK is rare and only isolated cases have been reported. De-

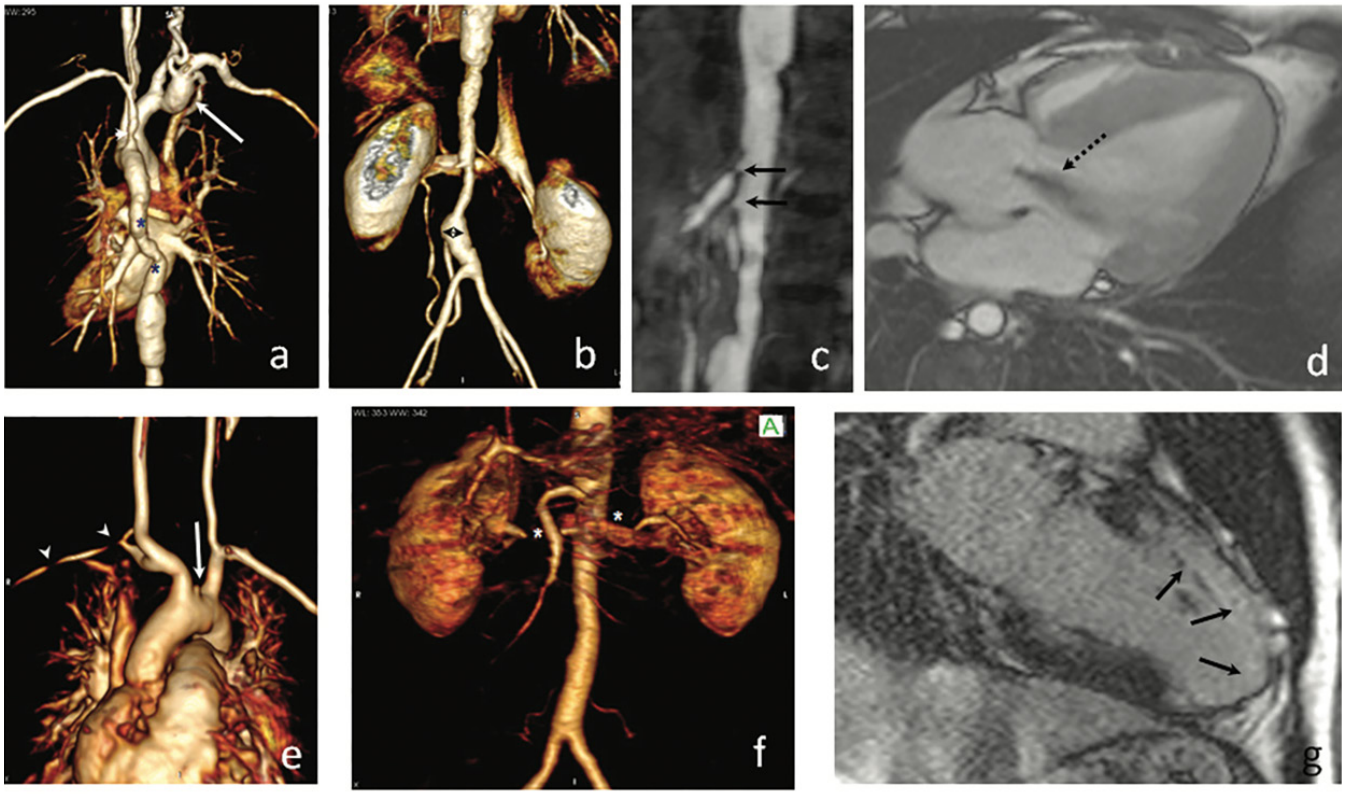

Fig. 4. In a-d a 47-year-old female with Numano type V TAK. MRA (a, b and c) shows dilation of the right subclavian (arrow), non-significant stenosis in the proximal segment of the left subclavian (arrowhead), as well as irregularity of the wall and decreased calibre of the aorta supradiaphragmatic and infrarenal (*), with dilatation of the distal aorta (◆). Significant stenosis of the (c) celiac trunk and superior mesenteric artery (black arrows) TRUE FISP sequence in 3 chambers (d) show severe aortic regurgitation (dashed arrow). In e-g) 26-year-old male with Takayasu arteritis type V. In (e-f) angiographic reconstructions show occlusion of the left common carotid artery (arrow), significant middle segment stenosis of the right subclavian artery (arrowhead). Both renal arteries with lack of signal (*) due to presence of stents with distal flow. In the inversion recovery sequences in 2 chambers (g) there is anterior transmural LGE in the middle and apical third and apex (black arrows). Dilated left ventricle, with mild systolic dysfunction (LVEF 49%).

generative cause of a complete A-V block in young people is rare and TAK should be considered in the differential diagnosis. In our series one patient had complete A-V block as a first manifestation (37).

TAK is considered the great mimicker, due to the insidious symptoms and variable progression, and frequently its diagnosis is delayed with catastrophic outcomes in some young patients.

In this series of TAK patients undergoing MRI the damage is extensive, and this is what we documented since we found that the injuries involve vessels that are not included in the current classification, such as the brain, heart (myocardium, valves, and conduction systems), visceral arteries and lower limb arteries. These findings are relevant based on the fact that it has been found that in patients with TAK, manifestations of organic damage may be the initial symptom, such as the presence of stroke (60), AMI (61), heart failure (62) and necrosis in the lower limbs (63), without it being known whether the affectation of intracerebral, coronary or extremity vessels began at the same time as the aorta and its main branches or even they were the first affected site.

We propose that in patients with TAK a complete MRI study be performed at the first evaluation, with a systematic report that includes the location and type of vascular lesion, wall thickness, as well as cerebral, cardiac, and visceral findings.

This initial study will allow the most exact comparison with the follow-up studies that are carried out, which should be requested according to the symptoms that the patients present during the evolution of the disease.

This is one of the first prospective studies on this rare disease. Imaging studies performed on them using complete magnetic resonance imaging showed that in some patients during their evolution of the disease they had arterial involvement in organs that were not initially evaluated, in which it is not possible to define whether the arterial lesion was of recent onset or this had already progressed chronically. This suggests that the arterial damage in these patients could have arterial damage in other sites in addition to that specifically suspected by the treating physician at the time of requesting an imaging study. Therefore, our proposal is that reports of arterial damage in TAK by MRI should be complete and also use a systematised format for reporting the damage, which could support the physician in timely therapeutic decisions in this vasculitis and improve quality of patient care. On the other hand, it could lead to benefit in research among centres that treat TAK.

Limitations

Although the number of patients included may seem a limitation, TAK

is a rare disease and few centres have larger sample sizes. Our findings could be strengthened by increasing the number of patients with a multicentre study.

Conclusion

The comprehensive evaluation of patients with TAK by MRI, using a standardised and systematic reporting format that describes the findings of vascular and organ lesions, is a tool that could facilitate better interdisciplinary communication in the study of this vasculitis. The usefulness of systematic reporting could have a global benefit that should be defined by its use through reproducibility by other groups.

The findings show that in patients with TAK, performing a complete imaging study from the moment of diagnosis will allow for timely follow-up during the course of the disease and establish disease surveillance with effective and timely therapy. The use of the format will establish comparisons between patients and between studies of other populations, with a precision that should be evaluated in future multicentre studies.

References

1. NUMANO F, OKAWARA M, INOMATA H, KOBAYASHI Y: Takayasu's arteritis. *Lancet* 2000; 356(9234): 1023-5. https://doi.org/10.1016/S0140-6736(00)02701-x
2. JOHNSTON SL, LOCK RJ, GOMPELS MM: Takayasu arteritis: A review. *J Clin Pathol* 2002; 55(7): 481-6. https://doi.org/10.1136/jcp.55.7.481
3. SOTO ME, ESPINOLA N, FLORES-SUAREZ LF, REYES PA: Takayasu arteritis: clinical features in 110 Mexican Mestizo patients and cardiovascular impact on survival and prognosis. *Clin Exp Rheumatol* 2008; 26 (Suppl. 49): S9-15.
4. GAMARRA AI, CORAL P, QUINTANA G *et al*.: History of primary vasculitis in Latin America. *Med Sci Monit* 2010; 16(3) RA58-72.
5. DE MATOS SOEIRO A, FERES DE ALMEIDA MC, ANDREUCCI TORRES T *et al*.: Clinical characteristics and long-term outcome of patients with acute coronary syndromes and Takayasu arteritis. *Rev Port Cardiol* 2013; 32(4): 297-302. https://doi.org/10.1016/j.repc.2012.06.020
6. HALL S, BARR W, LIE JT, STANSON AW, KAZMIER FJ, HUNDER GG: Takayasu arteritis: a study of 32 north American patients. *Medicine* 1985; 64(2): 89-99. https://doi.org/10.1097/00005792-198503000-00002
7. ONEN F, AKKOC N: Epidemiology of Takayasu arteritis. *Press Med* 2017; 46(7-8P2): e197-e203. https://doi.org/10.1016/j.lpm.2017.05.034
8. AESCHLIMANN FA, TWILT M, YEUNG RSM: Childhood-onset Takayasu Arteritis. *Eur J Rheumatol* 2020; 7(1): 58-66. https://doi.org/10.5152/eurjrheum.2019.19195
9. KOTHARI S: Takayasu's arteritis in children - a review. *Images Paediatr Cardiol* 2001; 3(4): 4-23.
10. DI SANTO M, STELMASZEWSKI E V, VILLA A: Takayasu arteritis in paediatrics. *Cardiol Young* 2018; 28(3): 354-61. https://doi.org/10.1017/S1047951117001998
11. ARNAUD L, HAROCHE J, MATHIAN A, GOROCHOV G, AMOURA Z: Pathogenesis of Takayasu's arteritis: a 2011 update. *Autoimmun Rev* 2011; 11(1): 61-67. https://doi.org/10.1016/j.autrev.2011.08.001
12. INDER S: Immunophenotypic analysis of the aortic wall in Takayasu's arteritis: involvement of lymphocytes, dendritic cells and granulocytes in immuno-inflammatory reactions. *Cardiovasc Surg* 2000; 8(2): 141-48. https://doi.org/10.1016/s0967-2109(99)00100-3
13. DEIPOLYI AR, CZAPLICKI CD, OKLU R: Inflammatory and infectious aortic diseases. *Cardiovasc Diagn Ther* 2018; 8 (Suppl. 1): S61-S70. https://doi.org/10.21037/cdt.2017.09.03
14. KERR GS, HALLAHAN CW, GIORDANO J *et al*.: Takayasu arteritis. *Ann Intern Med* 1994; 120(11): 919-29. https://doi.org/10.7326/0003-4819-120-11-199406010-00004
15. GULATI A, BAGGA A: Large vessel vasculitis. *Pediatr Nephrol* 2010; 25(6): 1037-48. https://doi.org/10.1007/s00467-009-1312-9
16. PARK JH, CHUNG JW, IM JG, KIM SK, PARK YB, HAN MC: Takayasu arteritis: evaluation of mural changes in the aorta and pulmonary artery with CT angiography. *Radiology* 1995; 196(1): 89-93. https://doi.org/10.1148/radiology.196.1.7784596
17. TANIGAWA K, EGUCHI K, KITAMURA Y *et al*.: Magnetic resonance imaging detection of aortic and pulmonary artery wall thickening in the acute stage of Takayasu arteritis. Improvement of clinical and radiologic findings after steroid therapy. *Arthritis Rheum* 1992; 35(4): 476-80. https://doi.org/10.1002/art.1780350419
18. TOMBETTI E, MASON JC: Application of imaging techniques for Takayasu arteritis. *Presse Med*. 46(7-8 Pt 2): e215-e223. https://doi.org/10.1016/j.lpm.2017.03.022
19. SUN Y, MA L, JI Z *et al*.: Value of whole-body contrast-enhanced magnetic resonance angiography with vessel wall imaging in quantitative assessment of disease activity and follow-up examination in Takayasu's arteritis. *Clin Rheumatol* 2016; 35(3): 685-93. https://doi.org/10.1007/s10067-015-2885-2
20. LEE GY, JANG SY, KO SM *et al*.: Cardiovascular manifestations of Takayasu arteritis and their relationship to the disease activity: analysis of 204 Korean patients at a single center. *Int J Cardiol* 2012; 159(1): 14-20. https://doi.org/10.1016/j.ijcard.2011.01.094
21. MORETTI M, TREPPO E, MONTI S *et al*.: Systemic vasculitis: one year in review 2023. *Clin Exp Rheumatol* 2023; 41(4): 765-73. https://doi.org/10.55563/clinexprheumatol/zf4daj
22. TREPPO E, MONTI S, DELVINO P *et al*.: Systemic vasculitis: one year in review 2024. *Clin Exp Rheumatol* 2024; 42(4): 771-81. https://doi.org/10.55563/clinexprheumatol/gkve60
23. FRIES JF, HUNDER GG, BLOCH DA *et al*.: The American College of Rheumatology 1990 criteria for the classification of vasculitis: Summary. *Arthritis Rheum* 1990; 33(8): 1135-36. https://doi.org/10.1002/art.1780330812
24. OZEN S, PISTORIO A, IUSAN SM *et al*.: EULAR/PRINTO/PRES criteria for Henoch-Schonlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: Final classification criteria. *Ann Rheum Dis* 2010; 69(5): 798-806. https://doi.org/10.1136/ard.2009.116657
25. GRAYSON PC, PONTE C, SUPPIAH R *et al*.: 2022 American College of Rheumatology/EULAR classification criteria for Takayasu arteritis. *Ann Rheum Dis* 2022; 81(12): 1654-60. https://doi.org/10.1136/ard-2022-223482
26. DABAGUE J, REYES PA: Takayasu arteritis in Mexico: a 38-year clinical perspective through literature review. *Int J Cardiol* 1996; 54 Suppl: S103-9. https://doi.org/10.1016/s0167-5273(96)88779-1
27. GIBBONS RJ, BALADY GJ, BRICKER JT *et al*.: ACC/AHA 2002 guideline update for exercise testing: summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). *J Am Coll Cardiol* 2002; 40(8): 1531-40. https://doi.org/10.1016/s0735-1097(02)02164-2
28. KRAMER CM, BARKHAUSEN J, FLAMM SD *et al*.: Standardized cardiovascular magnetic resonance (CMR) protocols 2013 update. *J Cardiovasc Magn Reson* 2013; 15: 91. https://doi.org/10.1186/1532-429x-15-91
29. KRAMER CM, BARKHAUSEN J, BUCCIARELLI-DUCCI C, FLAMM SD, KIM RJ, NAGEL E: Standardized cardiovascular magnetic resonance imaging (CMR) protocols: 2020 update. *J Cardiovasc Magn Reson* 2020; 22(1): 17. https://doi.org/10.1186/s12968-020-00607-1
30. SRINIVASAN A, GOYAL M, AL AZRI F, LUM C: State-of-the-art imaging of acute stroke. *Radiographics* 2006; 26 Suppl 1: S75-95. https://doi.org/10.1148/rg.26si065501
31. HATA A, NODA M, MORIWAKI R, NUMANO F: Angiographic findings of Takayasu arteritis: New classification. *Int J Cardiol* 1996; 54(S2): S155-S163. https://doi.org/10.1016/S0167-5273(96)02813-6
32. CERQUEIRA MD, WEISSMAN NJ, DILSIZIAN V *et al*.: Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation* 2002; 105(4): 539-52. https://doi.org/10.1161/hc0402.102975
33. CUMMINGS KW, BHALLA S, JAVIDAN-NEJAD C, BIERHALS AJ, GUTIERREZ FR, WOODARD PK: A pattern-based approach to assessment of delayed enhancement in nonischemic cardiomyopathy at MR imaging. *Radiographics*

2009; 29(1): 89-103. https://doi.org/10.1148/rg.291085052
34. SCHULZ-MENGER J, BLUEMKE DA, BREMERICH J *et al*.: Standardized image interpretation and post processing in cardiovascular magnetic resonance: Society for Cardiovascular Magnetic Resonance (SCMR) board of Trustees task force on standardized post processing. *J Cardiovasc Magn Reson* 2013; 15: 35. https://doi.org/10.1186/1532-429X-15-35
35. SCHULZ-MENGER J, BLUEMKE DA, BREMERICH J *et al*.: Standardized image interpretation and post-processing in cardiovascular magnetic resonance - 2020 update: Society for Cardiovascular Magnetic Resonance (SCMR): Board of Trustees Task Force on Standardized Post-Processing. *J Cardiovasc Magn Reson* 2020; 22(1): 19. https://doi.org/10.1186/s12968-020-00610-6
36. HWANG J, KIM SJ, BANG OY *et al*.: Ischemic stroke in Takayasu's arteritis: lesion patterns and possible mechanisms. *J Clin Neurol* 2012; 8(2): 109-15. https://doi.org/10.3988/jcn.2012.8.2.109
37. MELÉNDEZ-RAMÍREZ G, VERA-URQUIZA R, SOTO ME: Interventricular septum involvement with complete atrioventricular block as first manifestation in Takayasu arteritis. *J Nucl Cardiol* 2019; 26(1): 324-27. https://doi.org/10.1007/s12350-017-1165-8
38. LI J, SUN F, CHEN Z *et al*.: The clinical characteristics of Chinese Takayasu's arteritis patients: a retrospective study of 411 patients over 24 years. *Arthritis Res Ther* 2017; 19(1): 107. https://doi.org/10.1186/s13075-017-1307-z
39. GUDBRANDSSON B, MOLBERG Ø, GAREN T, PALM Ø: Prevalence, incidence and disease characteristics of Takayasu Arteritis differ by ethnic background: data from a large, population-based cohort resident in Southern Norway. *Arthritis Care Res* (Hoboken) 2017; 69(2): 278-85. https://doi.org/10.1002/acr.22931
40. SCHMIDT J, KERMANI TA, BACANI AK *et al*.: Diagnostic features, treatment, and outcomes of Takayasu arteritis in a US cohort of 126 patients. *Mayo Clin Proc* 2013; 88(8): 822-30. https://doi.org/10.1016/j.mayocp.2013.04.025
41. SUGIYAMA K, WATANUKI H, FUTAMURA Y, OKADA M, MAKINO S, MATSUYAMA K: Surgical treatment for brachiocephalic artery aneurysm with Takayasu arteritis using isolated cerebral perfusion: a case study. *J Cardiothorac Surg* 2021;1 6(1): 37. https://doi.org/10.1186/s13019-021-01413-1
42. WU X, DUAN HY, GU YQ *et al*.: Surgical treatment of brachiocephalic vessel involvement in Takayasu's arteritis. *Chin Med J* (Engl) 2010; 123(9): 1122-26.
43. ANGILETTA D, MARINAZZO D, GUIDO G, FULLONE M, PULLI R, REGINA G: Eight-year follow-up of endovascular repair of a brachiocephalic trunk aneurysm due to Takayasu's arteritis. *J Vasc Surg* 2012; 56(2): 504-7. https://doi.org/10.1016/j.jvs.2012.02.031
44. BAKHRITDINOV FS, SUYUMOV AS, SHARAPOV NU: [Outcomes of surgical management of patients with nonspecific aortoarteritis with isolated lesion of brachiocephalic arteries]. *Angiol Sosud Khir* 2016; 22(4): 144-50.
45. CHANDRASHEKHARA SH, GULATI GS, SHARMA S, JAGIA P, KUMAR S, CHOUDHARY S: Comparison between time-resolved magnetic resonance angiography and diagnostic digital subtraction angiography in the vascular assessment of nonspecific aorto-arteritis patients: a prospective study. *Vasc Endovascular Surg* 2021; 55(6): 586-92. https://doi.org/10.1177/15385744211010593
46. MATSUMOTO T, ISHIZUKA M, ISO Y, KITA J, KUBOTA K: Mini-Laparotomy for Superior Mesenteric Artery Aneurysm Due to Takayasu's Arteritis. *Int Surg* 2015; 100(4): 765-69. https://doi.org/10.9738/INTSURG-D-14-00127.1
47. MIROUSE A, BIARD L, COMARMOND C *et al*.: Overall survival and mortality risk factors in Takayasu' s arteritis: A multicenter study of 318 patients. *J Autoimmun* 2019; 96: 35-39. https://doi.org/10.1016/j.jaut.2018.08.001
48. CHEN Z, LI J, YANG Y *et al*.: The renal artery is involved in Chinese Takayasu's arteritis patients. *Kidney Int* 2018; 93(1): 245-51. https://doi.org/10.1016/j.kint.2017.06.027
49. SHLOMAI A, HERSHKO AY, GABBAY E, BEN-CHETRIT E: Clinical and radiographic features mimicking pulmonary embolism as the first manifestation of Takayasu's arteritis. *Clin Rheumatol* 2004; 23(5): 470-72. https://doi.org/10.1007/s10067-004-0929-0
50. NAKABAYASHI K, KURATA N, NANGI N, MIYAKE H, NAGASAWA T: Pulmonary artery involvement as first manifestation in three cases of Takayasu arteritis. *Int J Cardiol* 1996; 54 Suppl: S177-83. https://doi.org/10.1016/s0167-5273(96)88787-0
51. HE Y, LV N, DANG A, CHENG N: Pulmonary Artery Involvement in Patients with Takayasu Arteritis. *J Rheumatol* 2020; 47(2): 264-72. https://doi.org/10.3899/jrheum.190045
52. FIELDER JF: A 23-year-old woman admitted to Kijabe Mission Hospital with bilateral lower extremity gangrene. *MedGenMed* 2004; 6(1): 56.
53. DONG H, CHEN Y, XIONG HL, CHE WQ, ZOU YB, JIANG XJ: Endovascular treatment of iliac artery stenosis caused by Takayasu arteritis: a 10-year experience. *J Endovasc Ther* 2019; 26(6): 810-15. https://doi.org/10.1177/1526602819874474
54. DUARTE MM, GERALDES R, SOUSA R, ALARCÃO J, COSTA J: Stroke and transient ischemic attack in Takayasu's arteritis: a systematic review and meta-analysis. *J Stroke Cerebrovasc Dis* 2016; 25(4): 781-91. https://doi.org/10.1016/j.jstrokecerebrovasdis.2015.12.005
55. REN Y, DU J, GUO X *et al*.: Cardiac valvular involvement of Takayasu arteritis. *Clin Rheumatol* 2021; 40(2): 653-60. https://doi.org/10.1007/s10067-020-05290-2
56. LI J, LI H, SUN F *et al*.: Clinical characteristics of heart involvement in Chinese patients with Takayasu arteritis. *J Rheumatol* 2017; 44(12): 1867-74. https://doi.org/10.3899/jrheum.161514
57. YANG L, ZHANG H, JIANG X *et al*.: Clinical manifestations and long-term outcome for patients with Takayasu arteritis in China. *J Rheumatol* 2014; 41(12): 2439-46. https://doi.org/10.3899/jrheum.140664
58. FATEH-MOGHADAM S, HUEHNS S, SCHMIDT WA, DIETZ R, BOCKSCH W: Pericardial effusion as primary manifestation of Takayasu arteritis. *Int J Cardiol* 2010; 145(1): e33-35. https://doi.org/10.1016/j.ijcard.2008.12.133
59. COMARMOND C, CLUZEL P, TOLEDANO D *et al*.: Findings of cardiac magnetic resonance imaging in asymptomatic myocardial ischemic disease in Takayasu arteritis. *Am J Cardiol* 2014; 113(5): 881-87. https://doi.org/10.1016/j.amjcard.2013.11.045
60. DAVARI P, SUTTON P, JONES KS: Stroke as the initial presentation of Takayasu's arteritis: A case report. *Radiol Case Rep* 2020; 15(5): 556-59. https://doi.org/10.1016/j.radcr.2019.12.019
61. ZHANG T, PENG B, TU X, ZHANG S, ZHONG S, CAO W: Acute myocardial infarction as the first manifestation of Takayasu arteritis: A case report. *Medicine* (Baltimore) 2019; 98(15): e15143. https://doi.org/10.1097/md.0000000000015143
62. CASTRO DE OLIVEIRA FIGUEIRÔA MDL, MOURA COSTA MC, ROCHA LOBO P, SOUZA PEDREIRA AL, BARRETO SANTIAGO M: Acute heart failure due to myocarditis in Takayasu's arteritis. *Reumatismo* 2024; 76(4). https://doi.org/10.4081/reumatismo.2024.1681
63. MISRA DP, CHOWDHURY AC, LAL H, MOHINDRA N, AGARWAL V: Gangrene in Takayasu's arteritis: a report of two cases and review of literature. *Rheumatol Int* 2016; 36(3): 449-53. https://doi.org/10.1007/s00296-015-3392-0