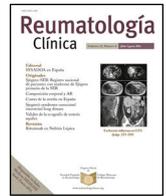




Sociedad Española
de Reumatología -
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de Reumatología



Letter to the Editor

Monckeberg's medial sclerosis in temporal artery mimicking giant cell arteritis



Esclerosis medial de Monckeberg de la arteria temporal simulando arteritis de células gigantes

Dear Editor:

Monckeberg's medial sclerosis (MMS) is a non-inflammatory degenerative condition affecting primarily the arteries of extremities and visceral organs. MMS in temporal artery is rare and may present some clinical similarities with giant cell arteritis (GCA). We aimed to draw attention to this pathology by sharing our case with MMS in temporal artery (TA) mimicking GCA.

A 72-year-old female patient was referred to our clinic due to severe headache without any pathological finding in cranial computerized tomography and magnetic resonance imaging. She had a two-week history of headache with severe tenderness on right temporal area. Clinical and laboratory evaluations were unremarkable except the tenderness on right temporal area, high erythrocyte sedimentation rate (ESR 37 mm/h) and mild anemia. There was no "halo sign" in color Doppler ultrasonography (USG). Oral methylprednisolone was started and temporal artery biopsy (TAB) was performed to confirm GCA. Her complaints were responded to glucocorticoid and resolved in three weeks. In the histopathological evaluation TAB, there were MMS characteristics with degeneration and calcification in the internal elastic lamina in tunica media and obliteration in lumen of medium-large-sized vessel (Fig. 1). GCA was excluded and medication was gradually stopped.

MMS is a non-inflammatory degenerative condition affecting primarily the tunica media of arteries resulting in their calcification.¹ As a mimicker of GCA, it may be considered as a non-giant cell temporal arteritis pathology. It is frequently, but not exclusively, associated with aging, type 2 diabetes mellitus, chronic kidney diseases, hormonal disorders and vitamin deficiencies and it may lead to hemodynamic changes in the microcirculation and with its progress, it may cause decreased organ perfusion.² It occurs predominantly in femoral, tibial, radial, coronary, cerebral and visceral vessels with generally no symptoms. However, acute vascular complications including ischemic changes have also been reported.³

MMS in TA has only rarely been reported previously and due to shared features with GCA, it may become a diagnostic challenge for the clinician.^{1–3} Symptoms indistinguishable from GCA such as headache, facial pain, tenderness in the temporal area, temporal artery hardening and acute vision loss have been reported in patients with MMS in TA.^{1,2} Also the pain due to MMS in TA was reported to be managed with glucocorticoids.¹ Although high ESR is expected in GCA, normal values do not exclude the diagnosis. The "halo sign" in Doppler USG is important in GCA however, it is not seen in all patients. Several imaging findings in plain radiographs,

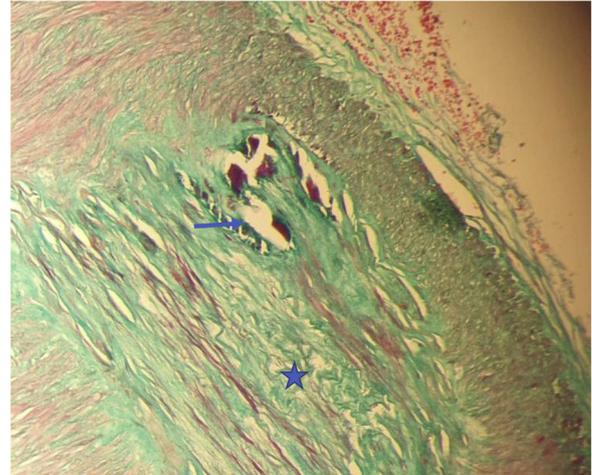


Fig. 1. Masson Trichrome, 200×. The histopathological evaluation of temporal artery biopsy specimen; Monckeberg's medial sclerosis characteristics with degeneration (star) and calcification (arrow) in the internal elastic lamina in tunica media and obliteration in lumen of medium-large-sized vessel.

CT and USG have also been reported for MMS in the head and neck region.¹

TAB remains the gold standard for the diagnosis of GCA. However, besides the possibility of false-negative biopsy result due to the skip lesions in GCA, other vasculopathy possibilities (atherosclerosis in the majority and MMS in the minority of the cases) should also be kept in mind. Histopathologically, besides an intact intima, degenerations and calcification in the internal elastic lamina in tunica media and obliteration in lumen of medium-large-sized vessel can be seen in MMS. However, endothelial structural disorganization, repair and thickening of the intima, as well as deformity of the arterial wall can be identified in atherosclerosis. Indeed, in a large series in suspected GCA cases, MMS was described in 6% of the TAB specimens.⁴

In conclusion, MMS in TA can mimic GCA due to the similarities of clinical features. It may become a diagnostic challenge for the clinician and an increased awareness of the disease may contribute to diagnosis.

Conflict of interest

The authors declare that they have no conflict of interest.

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Osteoarticular tuberculosis mortality in Spain between 1997 and 2018



Mortalidad de la tuberculosis osteoarticular en España entre 1997 y 2018

Dear Editor,

Tuberculosis (TB) is a worldwide public health problem. Although the osteoarticular form represents from 3% to 5% of its manifestations, few data on the mortality caused by this type are available¹. To expand the recently published study on its incidence and evolution over time², we analysed mortality due to osteoarticular TB in Spain from a hospital perspective in the years from 1997 to 2018.

The mortality and lethality of osteoarticular TB (OA TB) over 22 years were estimated in an observational retrospective study based on the data gathered in the minimum basic set of hospital discharge data for patients with a main or secondary diagnosis (according to CIE-9 and CIE-10) of OA TB in Spain from 1997 to 2018. 336 deaths occurred in 5710 patients.

The average annual mortality of patients with OA TB was 0.35 cases per million inhabitants (CI 95% 0.31–0.38). The World Health Organization has detected a falling tendency in the estimated rates of mortality due to TB in all regions since 2000³. The study found

a significant difference between the annual average mortality per million inhabitants in the first period (1997–2007) with 0.12 deaths p.m., and the second period (2008–2018) with 0.07 deaths p.m. ($P < .001$).

It is striking that there was no mortality among the 31 babies and 133 children aged from 1 to 14 years with OA TB, when the average rate of lethality due to TB in children worldwide is about 24%, and that TB is one of the 10 main causes of death among children in the whole world⁴. Nevertheless, this may be explained by the fact that more than 96% of all deaths due to TB occur in children who received no treatment against TB⁵.

On the other hand, the incidence and mortality were higher among those aged above 75 years (0.25 deaths per million). The overall fatality rate was 5.9% (CI 95% 5.3–6.53). Mortality among the patients with OA TB as their main diagnosis at admission was 3.5 (CI 95% 2.85–4.20). A meta-analysis estimated that the combined percentage of patients with TB who died during treatment of the same was 18.8% in the patients infected with HIV and 3.5% in those not infected with HIV⁶.

The fatality rates for coinfection of OA TB with miliary TB or CNS TB were, respectively, 9.5% and 9.3%, while in two works in Spain that used methodology similar to ours, the fatality rates for miliary TB and CNS TB were 14% and 15.5%, respectively^{7,8}. The fatality rate of the osteoarticular form is lower than that of the other forms of TB, and this may be due to the fact that

Table 1
Bivariate and multivariate analysis of risk factors and comorbidities for death.

Bivariate and multivariate analysis of the risk factors associated with mortality						
Variables	Death in hospital with osteoarticular TB (n = 336)		Bivariate analysis		Multivariate analysis	
	Yes	No	P value	OR (CI 95%)	P value	OR (CI 95%)
	N (%)	N (%)				
Neurological deficit						
Yes	23 (11.4%)	179 (88.6%)	.001	2.133 (1.361–3.341) Control group	.001	2.403 (1.446–3.994) Control group
No	313 (5.7%)	5195 (94.3%)				
Cerebrovascular disease						
Yes	28 (24.1%)	88 (75.9%)	<.001	5.461 (3.515–8.483) Control group	<.001	3.385 (2.044–5.608) Control group
No	308 (5.5%)	5286 (94.5%)				
Diabetes mellitus						
Yes	57 (9.2%)	564 (90.8%)	<.001	1.742 (1.293–2.347) Control group	.529	0.896 (0.637–1.261) Control group
No	279 (5.5%)	4.810 (94.1%)				
Malign neoplasia						
Yes	38 (24.8%)	115 (75.2%)	<.001	5.831 (3.969–8.569) Control group	<.001	6.229 (4.025–9.639) Control group
No	298 (5.4%)	5.259 (94.6%)				
Chronic liver disease						
Yes	22 (17.7%)	102 (82.3%)	<.001	3.621 (2.253–5.821) Control group	<.001	3.503 (1.923–6.381) Control group
No	314 (5.6%)	5.272 (94.4%)				