Reumatizam 2019;66(2):75–80
https://doi.org/10.33004/reumatizam-66-2-10

CASE REPORT PRIKAZ BOLESNIKA

GIANT CELL ARTERITIS IN A PATIENT WITH BILATERAL PAROTID SWELLING AS THE FIRST SIGN OF THE DISEASE: A CASE REPORT

GIGANTOCELULARNI ARTERITIS: PRIKAZ BOLESNICE S OTEKLINOM PAROTIDNIH ŽLIJEZDA KAO PRVOM MANIFESTACIJOM BOLESTI

Diana Bajo[1], Ana Begović[1], Katarina Borić[1], Mijo Meter[2], Dijana Perković[1]

[1]Division of Clinical Immunology and Rheumatology, Department of Internal Medicine, University of Split, School of Medicine, University Hospital Center Split / Zavod za kliničku imunologiju i reumatologiju, Klinika za unutarnje bolesti, Medicinski fakultet Sveučilišta u Splitu, Klinički bolnički centar Split
[2]Department of Cardiovascular Diseases, University of Split, School of Medicine, University Hospital Center Split / Klinika za bolesti srca i krvnih žila, Medicinski fakultet Sveučilišta u Splitu, Klinički bolnički centar Split

Corresponding author / Adresa autora za dopisivanje:
prim. doc. dr. sc. Dijana Perković, dr. med.
Division of Clinical Immunology and Rheumatology / Zavod za kliničku imunologiju i reumatologiju
Department of Internal Medicine / Klinika za unutarnje bolesti
University of Split, School of Medicine / Medicinski fakultet Sveučilišta u Splitu
University Hospital Centre Split / Klinički bolnički centar Split
Šoltanska 1
21000 Split
E-mail / e-pošta: dijana.perkovic@hotmail.com

Received / Primljeno: July 17, 2019 / 17. srpnja 2019.
Accepted / Prihvaćeno: November 18, 2019 / 18. studenoga 2019.

Abstract

We report a case of a patient with vision loss, whose diagnosis of temporal arteritis was confirmed by temporal artery biopsy. The patient presented with bilateral parotid swelling, *amaurosis fugax,* jaw claudication, and then headache. After the introduction of glucocorticoid therapy, the symptoms of the disease subsided, but there was a permanent loss of vision in one eye. This case indicates that it is important to consider temporal arteritis as a differential diagnosis in patients older than 50 with *amaurosis fugax* or other atypical symptoms that may precede the headache. A multidisciplinary approach involving neurologist, otorhinolaryngologist, infectiologist, ophthalmologist, and rheumatologist is key to early diagnosis and start of appropriate treatment that reduces the risk of permanent vision loss.

Keywords: Giant cell arteritis – complications, diagnosis, drug therapy; Temporal arteries – pathology; Amaurosis fugax – etiology; Optic neuropathy, ischemic – etiology; Parotid diseases – etiology; Temporomandibular joint disorders – etiology; Headache – etiology; Glucocorticoids – therapeutic use

Sažetak

U ovom radu prikazali smo bolesnicu s gubitkom vida u sklopu temporalnog arteritisa potvrđenog biopsijom temporalne arterije. Klinički se bolest prezentirala oteklinom parotidnih žlijezda, *amaurosis fugax* i klaudikacijama čeljusti, a potom i glavoboljom. Nakon uvođenja glukokortikoidne terapije simptomi bolesti su regredirali, no zaostao je gubitak vida na jedno oko. Ovaj slučaj nas upozorava da je važno razmotriti temporalni arteritis kao diferencijalnu dijagnozu u bolesnika starijih od 50 godina s *amaurosis fugax* i ostalim atipičnim simptomima koji se mogu razviti prije pojave glavobolje. Dobra suradnja između neurologa, otorinolaringologa, infektologa, oftalmologa i reumatologa ključna je za rano postavljanje dijagnoze i početak odgovarajućeg liječenja koji smanjuje rizik od trajnog gubitka vida.

Ključne riječi: Gigantocelularni arteritis; Amurosis fugax; Oteklina parotidnih žlijezda; Glavobolja

INTRODUCTION

Giant cell arteritis (GCA), or temporal arteritis, is a chronic inflammatory disease affecting the large and medium-sized arteries in persons over the age of 50, with the highest incidence between the ages of 70 and 80 years (1). Most symptoms and signs of GCA are the result of affected cranial branches of arteries originating from the aortic arch, but given the systemic nature of the disease, other blood vessels can be affected too. The diagnosis of GCA should be considered in older patients who complain of new-onset headache, sudden onset of vision disturbances, especially the transient monocular vision loss, jaw claudication, fever of unknown origin, anemia or other systemic symptoms and signs. Elevated erythrocyte sedimentation rate (ESR) is usually present, frequently accompanied by high level of C-reactive protein (CRP) in the serum Current or previous diagnosis of polymyalgia rheumatica (PMR) increases the likelihood of either of these results (2). In approximately 50% of patients, GCA manifests with various visual disturbances. Among the most common ones are the monocular vision loss and anterior ischemic optic neuropathy (AION). It is estimated that binocular blindness will occur in 25–50% of untreated patients with monocular vision loss (3–5).

The main treatment is a high dosage of systemic glucocorticoids, which must be introduced as soon as GCA is diagnosed, especially in patients with recent or imminent vision loss. Treatment should not be delayed while waiting for the results of other diagnostic methods such as temporal artery biopsy, i.e. the results of histological examination of biopsy specimen. For patients who have developed side effects or who depend on high doses of glucocorticoids, treatment can be augmented with methotrexate (MTX) or, as of more recently, interleukin 6 (IL-6) inhibitor tocilizumab (TCZ) (6–9).

In this paper, we present a case of a patient with swollen parotid glands and visual disturbances as part of GCA, with both symptoms manifesting before the onset of headache.

CASE REPORT

The 66-year-old patient was hospitalized at the Department of Ophthalmology due to a monocular vision loss. Two months before admission, the patient experienced parotid gland swelling and thickening of th temporal arteries. A month before admission, she noticed visual disturbances in the left eye on several occasions. The disturbances took the form of transitory "curtain effect" that lasted no longer than 10 minutes, followed by gradual weakening of vision that developed over the course of around 10 days. The patient was examined in another hospital by otorhinolaryn-

UVOD

Gigantocelularni arteritis (GCA) ili arteritis divovskih stanica je kronična upalna bolest koja zahvaća velike i srednje velike arterije u osoba starijih od 50 godina, s najvećom incidencijom u sedmom desetljeću života (1). Većina simptoma i znakova GCA rezultat su zahvaćanja kranijalnih ogranaka arterija koje potječu iz luka aorte, ali budući da je bolest sistemska, mogu biti zahvaćene i ostale krvne žile. Dijagnozu GCA treba razmotriti u starijih bolesnika koji se žale na novonastalu glavobolju, nagli početak poremećaja vida, posebno prolazni monookularni gubitak vida, klaudikaciju čeljusti, neobjašnjivu temperaturu, anemiju ili druge sistemske simptome i znakove. Obično je prisutna ubrzana sedimentacija eritrocita (SE) i / ili visokim C-reaktivnim proteinom (CRP) u serumu. Trenutna ili prethodna dijagnoza reumatske polimialgije (PMR) povećava šansu bilo kojeg od ovih nalaza (2). GCA se u oko 50% bolesnika prezentira različitim poremećajima vida. Među najčešćima je monookularni gubitak vida i prednja ishemijska optička neuropatija [*engl. anterior ischemic optic neuropathy* (AION)]. Procjenjuje se da će se obostrano sljepoća razviti u 25 do 50 % neliječenih bolesnika s jednostranim gubitkom vida (3–5).

Visoke doze sistemskih glukokortikoida okosnica su terapije, a liječenje treba započeti odmah nakon što se postavi dijagnoza GCA, osobito u bolesnika s nedavnim ili prijetećim gubitkom vida. Liječenje ne treba odlagati dok se čeka nalaz drugih dijagnostičkih metoda kao što je biopsija temporalne arterije, odnosno rezultati patohistološke analize bioptata. U bolesnika koji su razvili nuspojave ili su ovisni o visokim dozama glukokortikoida u terapiju se može dodati metotreksat (MTX), a u novije vrijeme inhibitor interleukina – 6 (IL-6), tocilizumab (TCZ) (6–9).

U ovom radu prikazujemo bolesnicu s oteklinom parotidnih žlijezda i smetnjama vida u sklopu GCA, koji su nastali prije pojavljivanja glavobolje.

PRIKAZ BOLESNIKA

Šezdesetšestogodišnja bolesnica hospitalizirana je na Klinici za oftalmologiju zbog jednostranog gubitka vida. Dva mjeseca pred prijam pojavila joj se oteklina parotidnih žlijezda uz zadebljanja u predjelu temporalnih arterija. Mjesec dana pred prijam je u više navrata primijetila poremećaje vida lijevog oka u obliku kratkotrajnih „efekata zavjese" u trajanju do desetak minuta, a potom i postupno slabljenje vida koje se razvijalo kroz desetak dana. Pregledana je u drugoj ustanovi od strane specijalista otorinolaringologije, neurologije i oftalmologije. Tada joj je učinjena kompjutorizirana tomografija (CT) glave i otkrivena upala lijevog maksilarnog sinusa, zbog čega joj je ordiniran cefuroksim tijekom 10 dana, ali bez učinka. Nekoliko dana pred

gology, neurology and ophthalmology specialists. She underwent the computed tomography (CT) of the head at the same time, which revealed the inflammation of the left maxillary sinus. She was prescribed a 10-day course of cefuroxime, to no effect. Several days before admission to our hospital, she was subfebrile (axillary temperature up to 37.6°C) and began to experience pain in the jaw and temporal regions. After completely losing vision in her left eye, she was hospitalized at the Department of Ophtalmology, where she was diagnosed with anterior ischemic optic neuropathy (AION) of the left eye. During her hospital stay, we noticed that her right-eye vision was weakening too, so 3 days after hospitalization she was examined by a rheumatology specialist and transferred to the Department of Rheumatology. The patient received intravenous pulse glucocorticoid therapy on the same day (Solu-Medrol, 500 mg/day for 3 days). The treatment was continued with the dosage of 1 mg/kg, with gradual tappering. Her right-eye vision normalized 2 days after the start of the treatment, but left-eye vision loss persisted throughout her hospital stay. The patient had been treated for arterial hypertension and bronchial asthma, and she suffered two cerebrovascular insults (CVI) in 2013 due to the left carotid artery stenosis, for which she underwent a thromboendarterectomy. Upon admission, the patient was afebrile, her vital signs were normal, and she had several crusts that remained after herpes zoster infection in the parieto-occipital region Laboratory test results showed increased ESR (80 mm/h), elevated CRP (41.1 mg/L), leukocytosis (11.5×10^9/L) with neutrophilia (92.7%) and mild hyperglycemia (7.0 mmol/L). Other hematological and biochemistry parameters (erythrocytes, thrombocytes, transaminases, creatinine, electrolytes, C3 and C4 complement) were within normal reference ranges. Color Doppler ultrasound (CDUS) of the temporal arteries showed hypoechoic *halo* of both temporal arteries. Temporal artery biopsy was performed, and the histological examination determined that the media was thickened, with inflammatory lymphocyte and histiocyte infiltration and presence of multinuclear giant cells (CD68-positive). Magnetic resonance imaging of the brain showed a left supratentorial parietooccipital area of malacia/atrophy of the parenchyma, resembling the sequelae of the chronic vascular lesion, and a smaller chronic lacunar vascular lesion in the left frontal region subcortically. Results of microbiological tests (urine culture, hemoculture), tumor markers (carcinoembryonic antigen [CEA], CA 19-9, CA 125, CA 15-3, alpha fetoprotein [AFP]) and immunological parameters (antinuclear antibodies [ANA], rheumatoid factor [RF], antineutrophil cytoplasmic antibodies [ANCA]) all came back normal. Eight days after the start of the glucocorticoid therapy, follow-up testing

prijam u našu ustanovu je bila subfebrilna (Tax do 37,6 °C) te je počela osjećati bolove u čeljusti i u temporalnim regijama. Zbog potpunog gubitka vida na lijevo oko hospitalizirana je na Klinici za očne bolesti gdje je utvrđen AION lijevog oka. Tijekom hospitalizacije je primijetila slabljenje vida i na desnom oku, te je tri dana nakon hospitalizacije konzilijarno pregledana od strane reumatologa i premještena u Zavod za reumatologiju. Isti dan je primijenjena intravenska pulsna terapija glukokortikoidom (Solu-Medrol, 500 mg/dan kroz tri dana), a potom je nastavljeno liječenje u dozi od 1 mg/kgTT, uz postupno snižavanje doze. Dva dana nakon početka terapije vid na desno oko se normalizirao, no na lijevom oku je zaostao gubitak vida do kraja hospitalizacije. Bolesnica se inače liječila zbog arterijske hipertenzije i bronhijalne astme, a u dva navrata je 2013. godine preboljela cerebrovaskularni inzult (CVI), zbog stenoze lijeve karotidne arterije, te joj je učinjena trombendarterektomija. Prilikom prijma bila je afebrilna, s urednim vitalnim parametrima, a parijetookcipitalno je bilo vidljivo nekoliko krustoznih efloroscencija u sklopu herpes zoster infekcije, koja je bila u fazi sanacije. U laboratorijskim nalazima zabilježena je ubrzana SE (80 mm/h), povišena vrijednost CRP (41,1 mg/L), leukocitoza ($11,5 \times 10^9$/L) s neutrofilijom (92,7%) i blaga hiperglikemija (7,0 mmol /L). Ostali hematološki i biokemijski nalazi (eritrociti, trombociti, transaminaze, kreatinin, elektroliti, C3 i C4 komplement) bili su u granicama referentnih vrijednosti. Kolor dopler ultrazvuk (CDUS) temporalnih arterija je pokazao hipoehogeni *halo* obje temporalne arterije. Učinjena je i biopsija temporalne arterije, a patohistološkom analizom je utvrđeno zadebljanje medije s upalnim infiltratom limfocita i histiocita uz multinuklearne divovske stanice (CD68 pozitivne). Magnetska rezonancija (MR) mozga je pokazala lijevo supratentorijalno parijetookcipitalno zonu malacije / atrofije parenhima u smislu sekvele kronične vaskularne lezije, te manju kroničnu lakunarnu vaskularnu leziju lijevo frontalno subkortikalno. Nalazi mikrobioloških pretraga (urinokulture, hemokulture), tumorskih biljega [karcinoembrionalni antigen (CEA), CA 19-9, CA 125, CA 15-3, alfa-fetoprotein (AFP)] i imunoloških parametara [antinuklearna protutijela (ANA), reumatoidni faktor (RF), anti-neutrofilna citoplazmatska protutijela (ANCA)] bili su uredni. Osam dana od početka terapije glukokortikoidima ponovljene laboratorijske pretrage su pokazale normalizaciju upalnih parametara (CRP 2,6 mg/L), a bolesnica je otpuštena na kućnu njegu.

RASPRAVA

Prema Američkom reumatološkom društvu [*engl. American College of Rheumatology* (ACR)] za klasifikaciju GCA potrebni su dob od 50 godina ili više, novo-

showed normalization of inflammation parameters (CRP 2.6 mg/L), and the patient was discharged to home care.

DISCUSSION

According to the American College of Rheumatology (ACR), classification criteria for GCA includes age of 50 years or older, new-onset headache, temporal artery tenderness or decreased temporal artery pulsation, ESR of at least 50 mm/h, and positive artery biopsy results characterized by mononuclear infiltration or granulomatous inflammation (10). Definitive diagnosis is based on histological analysis of temporal artery or diagnostic imaging. Histological and immunohistochemical analyses show inflammation of the arterial wall dominated by CD4+ T lymphocytes and macrophages that frequently show granulomatous organization, forming giant cells. Vascular remodeling caused by inflammation leads to the intimal hyperplasia and occlusion of the lumen, which leads to ischemic complications. Histological specimen to prove GCA is most commonly obtained by the temporal artery biopsy (11). However, in the hands of experienced ultrasound specialists, CDUS can replace biopsy (12).

Our patient met all the criteria for the GCA diagnosis, but interestingly, the headache only began after the first manifestation of visual disturbances. Ordinarily, the onset of headache precedes visual disturbances and is present in approximately 90% of GCA patients. Available literature describes cases with atypical onset of the disease, such as occipital headache, limited range of jaw motion or orofacial pain (13–15). Such atypical onset of the disease, as was the case with our patient, hinders the timely diagnosis and beginning of treatment, which can have far-reaching consequences. The swelling of the parotid glands is an extremely rare symptom of this disease, caused by the vasculitis of the posterior auricular artery (16, 17). The patient in question experienced the swelling 2 months before hospitalization, and it subsided spontaneously over the course of approximately one month.

If GCA is suspected and visual disturbances have already manifested, the planned biopsy should not delay the start of the glucocorticoid treatment. According to some studies, temporal artery biopsy performed 1–4 weeks after the start of the glucocorticoid treatment reveals signs of inflammation typical for GCA, providing useful information for diagnosis even during that time (6). Negative biopsy results do not rule out the GCA diagnosis and can be expected in 10–15% of GCA patients (8, 12, 18). Positron emission tomography (PET), CT, CT angiography (CTA), and magnetic resonance angiography (MRA) lack the resolution for proper visualization of temporal artery (19). That may explain why our patient was not diagnosed with GCA after undergoing CT.

nastala glavobolja, osjetljivost temporalne arterije ili oslabljen puls temporalne arterije, brzina SE od najmanje 50 mm/h i pozitivna biopsija arterije obilježena mononuklearnom infiltracijom ili granulomatoznom upalom (10). Konačna dijagnoza se temelji na patohistološkoj analizi temporalne arterije ili slikovnom prikazu. Patohistološki i imunohistokemijski se nalazi upala stijenke arterije s prevladavanjem CD4+ T limfocita i makrofaga koji se često granulomatozno organiziraju te formiraju divovske stanice. Vaskularno remodeliranje uzrokovano upalom dovodi do hiperplazije intime i okluzije lumena što je izvor ishemijskih komplikacija bolesti. Histopatološki uzorak za dokazivanje GCA najčešće se dobiva biopsijom temporalne arterije (11). No, CDUS u rukama iskusnih ultrasoničara može zamijeniti biopsiju (12).

U naše bolesnice su zadovoljeni svi kriteriji za dijagnozu GCA, a zanimljivo je da je glavobolja počela nakon pojave prvih vidnih poremećaja. Naime, glavobolja se uglavnom javlja prije vidnih poremećaja i može se naći u oko 90% bolesnika s GCA. U dostupnoj literaturi su opisani prikazi bolesnika s atipičnim početkom bolesti, poput okcipitalne glavobolje, ograničenog otvaranja vilice ili orofacijalnih bolova (13–15). Ovakav atipičan početak bolesti, kao što je slučaj i u naše bolesnice, otežava pravovremeno postavljanje dijagnoze i početak liječenja što može imati dalekosežne posljedice. Oteklina parotidnih žlijezda je izrazito rijedak simptom bolesti koji nastaje kao posljedica vaskulitisa stražnje aurikularne arterije (16, 17). Takva oteklina se u prikazane bolesnice pojavila dva mjeseca prije hospitalizacije, a spontano se povukla kroz oko jedan mjesec.

Kod opravdane sumnje na GCA i pojave vidnih poremećaja planirana biopsija ne smije odlagati početak glukokortikoidne terapije. Prema nekim studijama, biopsija temporalne arterije uzeta 1–4 tjedna nakon početka liječenja glukokortikoidima otkriva upalne promjene tipične za GCA te i tada daje korisne informacije za dijagnozu (6). Negativni rezultati biopsije ne isključuju dijagnozu GCA, a očekuju se u 10–15% bolesnika s GCA (8, 12, 18). Pozitronska emisijska tomografija (PET), kmpjuterizirana tomografija (skr. CT), CT s angiografijom (CTA) i magnetna rezonancija s angiografijom (MRA) nemaju dovoljnu rezoluciju da omoguće vizualizaciju temporalne arterije (19). To može objasniti zašto našoj bolesnici nije dijagnosticiran GCA nakon što je bio učinjen CT.

Najozbiljnija komplikacija GCA je trajni gubitk vida koji je obično bezbolan i iznenadan, a može biti djelomičan ili potpun te jednostran ili obostran. Trajni gubitak vida u GCA rezultat je AION-a, okluzije središnje ili grane retinalne arterije [*engl. central retinal artery occlusion/branch retinal artery occlusion* (CRAO/BRAO)], stražnje ishemijske optičke neuropatije [*engl.*

The most serious GCA complication is permanent vision loss, which is usually painless and sudden. It can be partial or complete, monocular or binocular. Permanent vision loss in GCA is the result of AION, central retinal artery occlusion or branch retinal artery occlusion (CRAO/BRAO), posterior ischemic optic neuropathy (PION) or, rarely, cerebral ischemia. Fundoscopy is indicated in patients with subjective change in visual acuity (3,4,18). The first ophthalmological examination of our patient did not reveal anything unusual, while the follow-up examination found the AION of the left eye, which caused the permanent vision loss. The reported recurrence of CVI could also be a consequence of GCA, as CVI is described as part of GCA clinical presentation. However, it does not correspond with the GCA clinical presentation in our patient because it occurred several years earlier.

Given the fact that patients with undiagnosed GCA often "wander" between neurologists, ophthalmologists, infectiologists and rheumatologists, glucocorticoid therapy is usually not introduced in a timely manner in clinical practice (20, 21). It can lead to the permanent vision loss and other serious complications of this disease, as was the case with our patient.

CONCLUSION

In this paper we described the case of a patient with temporal arteritis who suffered irreversible vision loss in one eye as the result of delayed introduction of appropriate treatment. This case should alert us to the importance of taking temporal arteritis into consideration as a differential diagnosis in patients older than 50 with *amaurosis fugax*, even if typical symptoms are missing at the onset of disease. Additionally, bilateral parotid swelling, although exceedingly rare, can be one of the first manifestations of the disease. Good collaboration between ophthalmology and rheumatology specialists, as well as other specialists, is key to early diagnosis and start of appropriate treatment to reduce the risk of permanent vision loss and other effects of the disease.

Conflict of interest statement: Authors declare no conflict of interest.

posterior ischemic optic neuropathy (PION)] ili rijetko, cerebralne ishemije. Fundoskopija je indicirana u bolesnika sa subjektivnom promjenom oštrine vida (3,4,18). Prvi oftalmološki pregled naše bolesnice je bio uredan, dok je pri ponovljenom pregledu utvrđen AION lijevog oka, koji je doveo do trajnog gubitka vida. Podatak o recidivu CVI-a bi također mogao biti posljedica GCA. Naime, CVI se opisuje kao dio kliničke slike GCA, iako vremenski odmak od više godina se u ovom slučaju ne bi uklopio u kliničku sliku GCA.

Obzirom da bolesnici s neprepoznatim GCA često „lutaju" između neurologa, oftalmologa, infektologa i reumatologa, početak terapije glukokortikoidima u kliničkoj praksi nije pravovremen (20, 21), što može dovesti do trajnog gubitka vida i ostalih ozbiljnih komplikacija ove bolesti kao što je bio slučaj i u naše bolesnice.

ZAKLJUČAK

U ovom radu prikazali smo bolesnicu s temporalnim arteritisom u koje je zaostao gubitak vida na jedno oko zbog zakašnjele primjene odgovarajuće terapije. Ovaj slučaj upozorava nas da je važno razmotriti temporalni arteritis kao diferencijalnu dijagnozu u bolesnika starijih od 50 godina s *amaurosis fugax* čak i ako izostanu tipični simptomi u početku bolesti. Osim toga, oteklina u području parotidnih žlijezda, iako vrlo rijetka, može biti jedna od prvih manifestacija bolesti. Dobra suradnja specijalista oftalmologije i reumatologije, ali i drugih specijalista od ključne je važnosti za rano postavljanje dijagnoze i početak odgovarajućeg liječenja kako bi se smanjio rizik od trajnog gubitka vida i ostalih posljedica bolesti.

Izjava o sukobu interesa: Autori izjavljuju da nisu u sukobu interesa.

REFERENCES / LITERATURA

1. Dejaco C, Brouwer E, Mason JC, Buttgereit F, Matteson EL, Dasgupta B. Giant cell arteritis and polymyalgia rheumatica: current challenges and opportunities. Nat Rev Rheumatol. 2017;13(10):578.
2. Nordborg E, Nordborg C. Giant cell arteritis: strategies in diagnosis and treatment. Curr Opin Rheumatol 2004;16:25–30.
3. Miller NR. Visual manifestations of temporal arteritis. Rheum Dis Clin North Am. 2001;27(4):781.
4. Hayreh SS, Podhajsky PA, Zimmerman B. Ocular manifestations of giant cell arteritis. Am J Ophthalmol. 1998;125(4):509.
5. Barbazan C, Gonzalez-Gay MA, Aspe B, Fucionos L, Mantecon-Apariocio JM. Visual recovery after retinal stroke due to giant cell arteritis. Clin Exp Rheumatol. 2000;18:425–6.
6. Proven A, Gabriel SE, Orces C, O'Fallon WM, Hunder GG. Glucocorticoid therapy in giant cell arteritis: duration and adverse outcomes. Arthritis Rheum. 2003;49(5):703.
7. Villiger PM, Adler S, Kuchen S, i sur. Tocilizumab for induction and maintenance of remission in giant cell arteritis: A phase 2, randomised, double-blind, placebo-controlled trial. Lancet. 2016;387(10031):1921. Epub 2016 Mar 4.
8. Buttgereit F, Dejaco C, Matteson EL, Dasgupta B. Polymyalgia Rheumatica and Giant Cell Arteritis: A Systematic Review. JAMA. 2016;315(22):2442–58.
9. Hellmich B, Agueda A, Monti S, i sur. 2018 Update of the EULAR recommendations for the management of large vessel vasculitis. Ann Rheum Dis. 2020 Jan;79(1):19–30.
10. Hunder GG, Bloch DA, Michel BA, i sur. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. Arthritis Rheum 1990;33:1122–8.
11. Cid MC, Campo E, Ercilla G, i sur. Immunohistochemical analysis of lymphoid and macrophage cell subsets and their immunologic activation markers in temporal arteritis. Influence of corticosteroid treatment. Arthritis Rheum. 1989;32(7):884.
12. Luqmani R, Lee E, Singh S, i sur. The role of ultrasound compared to biopsy of temporal arteries in the diagnosis and treatment of giant cell arteritis (TABUL): a diagnostic accuracy and cost-effectiveness study. Health Technol Assess 2016;20:1–238.
13. Rana AQ, Saeed U, Khan OA, Qureshi AR, Paul D. Giant cell arteritis or tension-type headache?: A differential diagnostic dilemma. J Neurosci Rural Pract. 2014;5(4):409–11.
14. Reiter S, Winocur E, Goldsmith C, Emodi-Perlman A, Gorsky M. Giant cell arteritis misdiagnosed as temporomandibular disorder: A case report and review of the literature. J Orofac Pain. 2009;23:360–5.
15. Kraemer M, Metz A, Herold M, Venker C, Berlit P. Reduction in jaw opening: A neglected symptom of giant cell arteritis. Rheumatol Int. 2011;31:1521–3.
16. Amor-Dorado JC, Llorca J, Garcia-Porrua C, Costa C, Perez-Fernandez N, Gonzalez-Gay MA. Audiovestibular manifestations in giant cell arteritis: a prospective study. Medicine (Baltimore). 2003;82(1):13–26.
17. Czihal M, Tatò F, Hoffmann U, Kuhlencordt PJ. Juvenile temporal arteritis. Clin Exp Rheumatol. 2013;31(1 Suppl 75):S89.
18. Hayreh SS, Biousse V. Treatment of acute visual loss in giant cell arteritis: should we prescribe high-dose intravenous steroids or just oral steroids? J Neuroophthalmol. 2012;32(3):278.
19. Duftner C, Dejaco C, Sepriano A, i sur. Imaging in diagnosis, monitoring and outcome prediction of large vessel vasculitis: a systematic literature review and meta-analysis informing the EULAR recommendations. RMD open. 2018;4(1):e000612. doi: 10.1136/rmdopen-2017-000612. eCollection 2018.
20. Laskou F, Coath F, Mackie SL, Banerjee S, Aung T, Dasgupta B. A probability score to aid the diagnosis of suspected giant cell arteritis. Clin Exp Rheumatol 2019; 37 (Suppl. 117):S104–S108.
21. Lyons HS, Quick V, Sinclair AJ, Nagaraju S, Mollan SP**.** A new era for giant cell arteritis. Eye. https://doi.org/10.1038/s41433-019-0608-7.