Head-and-neck swelling: an under-recognized feature of giant cell arteritis. A report of 37 patients

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ABSTRACT

Objective. *To describe the frequency of occurrence and characteristics of head-and-neck swelling (HNS) in temporal (giant cell) arteritis (TA).*

Methods. *We analyzed the charts of patients with HNS retrieved from a single department series of 260 consecutive patients with TA and reviewed the published French-English literature. Patients with a swelling limited to the temporal fossa were excluded.*

Results. *A history of HNS was elicited in 17 patients in our series (i.e. 6.5%) and in 20 previously published patients. The swelling was an inaugural feature in most cases and was often transient. Ear-nose-and-throat (ENT) symptoms were observed in 80% of the cases, including jaw claudication or pain upon opening mouth in 22, causing trismus in 10. Two patients had permanent visual impairment and 1 had sudden hearing loss. The temporal artery biopsy yielded giant cell arteritis (GCA) in all the patients but 2. The HNS was often painful and mainly involved mainly the orbital region and face, particularly the lower part of the cheeks and maxillae, less often the neck and, rarely, the forehead and tongue. Concurrent localized limb swellings were also observed in 3 patients. The HNS disappeared in all the patients, either spontaneously or under steroid treatment, and recurred only in 6 patients.*

Conclusion. *HNS is not exceptional in untreated TA and is strongly associated with ENT symptoms and a positive TAB, but not with visual loss or stroke. Such characteristics imply in these cases a prominent, widespread involvement of the external carotid artery system by giant cell arteritis.*

Introduction

Giant cell arteritis (GCA) or temporal arteritis (TA) is a systemic vasculitis of the elderly which affects large and medium-sized arteries, predominantly the branches of external carotid artery. The most common cranial symptoms of TA are headaches, scalp tenderness, visual disturbances and jaw claudication. Other presentations of GCA are much more unusual but are important to recognize so that the potentially catastrophic, but often delayed, ischemic complications of the disease can be avoided. Facial swelling as a prominent feature of TA has seldom been reported (1-4), though mentioned in the past English literature (5, 6). Thus, owing to a relative lack of awareness of this aetiology in the differential diagnosis of facial swelling, delayed or misleading diagnosis, as well as inappropriate therapeutic options, may ensue. In this paper, we report a personal series of 17 patients with head-and-neck swelling (HNS) heralding GCA and survey all similar cases published in the French-English literature, with the aim of reinforcing current knowledge of this feature of TA.

Patients and methods

Patients and data collection

We analyzed retrospectively the clinical, laboratory, and pathologic features of 260 consecutive patients with TA who were referred to the department of Internal Medicine of our hospital between January 1976 and December 2004. Ninety-three percent of the patients were recruited before treatment, the remaining cases being already treated for less than one month at the time of admission. Only cases fulfilling at least three of the American College of Rheumatology criteria for GCA (7) were included in the study. The diagnosis of GCA was pathologically established in 210 patients (8). Pretreatment, clinical, laboratory and pathological data were recorded prospectively at the time of diagnosis by a senior internist using for each patient a specifically designed, comprehensive, questionnaire that includes a precise history and

174 items, then transferred to a computerised database. Special efforts were made in evaluating the delay to diagnosis from the onset of symptoms of vasculitis, the presence of constitutional syndrome (defined by a temperature ≥ 38°C for at least one week, severe asthenia, and/or weight loss > 5%), polymyalgia rheumatica (PMR) (9), abnormal temporal artery on examination (absence of pulses on all or part of its course, nodules, thickening, swelling or tenderness on palpation) and upper limb artery involvement (presence of intermittent arm claudication, absent or decreased radial pulse, Raynaud's phenomenon of recent onset, suggestive findings on selective aortic arch arteriography (10), or at least a murmur heard over subclavian-axillary arteries at admission or within a month). Ear-nose-throat (ENT) symptoms were meticulously recorded and included jaw claudication (11), pain upon opening mouth, trismus, maxillary or dental pain, otalgia, odynophagia, lingual pain or discomfort, thore throat, dysphagia, dry cough, and carotidynia (12). The occult (or silent) form of TA has been defined previously (31).

Head-and-neck swelling

The finding of HNS, its characteristics and outcome, were recorded in the patient's clinical history, since the questionnaire did not involve such a pre-established item. Cases with a mentioned HNS were extracted from the whole series and the patient's chart was thoroughly reviewed. All patients with an unquestionable history of HNS that had lasted at least one day were included in the study, irrespective of the patient's head and neck appearance at admission. Patients in whom the swelling was confined to the temporal fossa or faced an enlarged salivary gland (14, 15) were excluded.

Literature survey

French or English papers published since 1970 dealing with either GCA, TA or polymyalgia rheumatica (PMR) and facial or neck swelling were reviewed, as well as French congress abstracts. Patient characteristics, clinical presentation, temporal artery biopsy findings and outcome were analyzed. Well-described reports of HNS occurring concurrently with TA were included in the study.

Results

Features of TA in patients with HNS

A history of HNS was elicited in 17 out of 260 patients, i.e. 6.6% of the series, whilst the survey consisted of 20 case reports (1-4, 16-26). The main pre-treatment characteristics of GCA in these 37 patients are summarized in Table I. The age range was 57 to 90 years (mean 75.4 years) and 69% were women. The time to diagnosis from the first symptoms averaged 7 weeks, though the disease onset was acute in 15 out of 25 reported cases and 72% of the patients developed typical cranial arteritis. At least one ENT symptom was recorded in 80% of the cases, with a maximum of 6 symptoms in the same patient. Ear-and-throat symptoms were, apart from HNS, the only presenting manifestations of GCA in 3 patients. Jaw claudication and/or pain upon opening mouth involved 22 patients, with frank trismus in 10 (27%). Six patients had eye or orbital pain and 12 had transient visual ischemic symptoms but only 2 (6%) developed permanent visual loss. Extra-cranial manifestations were recorded in 32% of the patients. The erythrocyte sedimentation rate exceeded 50 mm/h in 87% of the cases and the temporal artery biopsy (TAB) yielded GCA in 33 out of 35 verified cases.

Characteristics of HNS in patients with TA

The main characteristics of TA-associated HNS are shown in Table II. Facial puffiness was frequently an early or even inaugural sign of TA but was often transient and had subsided on the first admission in half of the patients. The swelling lasted less than 1 one week in 60% of the cases with a maximum duration of 2 months. It was bilateral in nearly three-fourths of the cases and involved predominantly the face and cheeks (23 cases), the eyelids and orbital region (15 cases) and the anterior and lateral aspects of the neck (7 cases), less often the tongue (3 cases), forehead and scalp (2 cases), chin and supra-clavicular space (1 case each). Some patients or their relatives recalled a downward displacement of initial swelling from the cheeks to the maxillae and upper parts of the neck. Three patients had segmental arm and/or leg swellings concurrently with HNS; none of these had acquired angioedema. Patients often complained of pain or discomfort in parts of the face or neck precisely where the swelling developed shortly thereafter, but some felt the HNS painful by itself. On physical examination swelling was discretely pinkish in 8 out of 12 patients and generally not pitting or itchy. The HNS outcome was known in 32 patients. Seven noticed a relapsing or waning-and-waxing course. The swelling disappeared spontaneously (n = 13) or quickly upon corticosteroid treatment (n = 19) but recurred in 6 patients, with multiple recurrences on further attempts to taper the prednisone in one (24).

Discussion

In a patient with periorbital, facial and/or neck swelling, there is a lengthy list of diagnostic possibilities to make out, including notably allergic oedema and photosensitive reactions, bacterial, virus or parasitic infections, endocrine disorders, inherited or acquired angioneurotic oedema, acute onset systemic sclerosis or dermatomyositis, superior vena cava syndrome or jugular vein thrombosis, nephrotic syndrome and other causes of hypoproteinemia. Although swelling in the temporal fossa is a well-recognized feature of GCA, more diffuse swelling of the face as a manifestation of GCA has been described in the English literature only in a handful of cases (1-4). Likewise, available the French literature is scanty with several reports published only in an abstract form (16-26). Therefore, in an elderly person presenting with a swollen face, the degree of suspicion concerning the possibility of GCA is currently low, due to lack of awareness, with the risk of blurring more evoking symptoms. This is illustrated by a rather long average delay to diagnosis in these patients and by the fact that some were first seen by laryngologists with a

Table I. Characteristics of temporal arteritis in patients with concurrent head-and-neck swelling.

Author (ref)	Age /sex	Onset	Presenting features of TA	ESR (mm/h)	TAB
Cohen (1)	62 / F	acute	glossitis, odynophagia and maxillary pain	73	Pos
Herreman (16)	67 / M	n.s.	cranial	120	Pos
	68 / M	n.s.	cranial and PMR, with JC	70	Pos
Acetta (2)	63 / F	n.s.	cranial, with bilateral episcleritis and conjunctivitis	normal	Pos
Delvigne (17)	71 / M	n.s.	cranial, with diplopia and trismus	50	Pos
Manganelli (4)	71 / F	n.s.	cranial, with trismus and AF	87	Pos
Chevalet (19)	62 / F	acute	cranial and PMR, with trismus and dry cough	140	Pos
Ghanchi (3)	79 / F	acute	cranial and PMR, with PVL	94	Pos
Plantin (18)	84 / F	acute	cranial, with diplopia, trismus, and pain upon swallowing	68	n.d.
	81 / F	n.s.	cranial (after PMR) with trismus, tongue and eye pain	95	n.d.
	68 / F	n.s.	cranial and PMR, with upper limb artery involvement	118	pos
Gaches (21)	75 / M	n.s.	cranial, with JC and bilateral PVL	n.a.	Pos
	77 / M	n.s.	cranial, with dysphagia and transient visual impairement	n.a.	Pos
Salafi (20)	77 / F	n.s.	cranial and PMR, with JC, sore throat, dysphagia and AF	54	Pos
	70 / F	acute	cranial, with trismus	84	Pos
Sarrot-Reynaud (22)	81 / F	progressive	cranial, with trismus, odynophagia and VH	n.a.	Pos
Paris (23)	88 / F	n.s.	cranial, with agueusia	100	Pos
Paulus (24)	85 / F	acute	cranial, with JC	n.a.	Neg
Regouby (25)	70 / M	acute	cranial, with JC	80	Pos
Myara (26)	78 / F	acute	cranial, with diplopia (no headaches)	42	Pos
Current series	79 / F	progressive	cranial, with trismus, sore throat, hoarseness, dry cough otalgia and AF	92	Pos
	86/F	progressive	cranial, with JC, PUOM, lingual pain, dysphagia and AF	130	Pos
	78 / F	progressive	cranial, with JC, maxillary pain, otalgia, eye pain and diplopia	59	Pos
	88 / M	acute	cranial, with eye pain and transient blurred vision	53	Pos
	84 / F	acute	cranial, with JC, dysphagia, sore throat, PUOM and PVL	72	Pos
	72 / F	acute	cranial and PMR, with trismus and eye pain	154	Pos
	78 / M	progressive	occult (silent) form, then eye pain	102	Pos
	76 / M	progressive	cranial, with JC, dry cough and otalgia	42	Pos
	72 / F	progressive	cranial, with JC, PUOM, maxillary pain, dysphagia, otalgia and dry cough	46	Pos
	57 / F	acute	cranial and upper limb artery involvement, with maxillary pain	113	Pos
	79 / M	acute	cranial, with JC, sore throat and dry cough	60	Pos
	78 / F	acute	cranial, with JC, sore throat and dry cough	134	Pos
	81 / F	acute	sore throat and abnormal temporal arteries, without headaches	60	Pos
	76 / F	progressive	trismus and dysphagia, without headaches	90	Pos
	90 / F	acute	cranial	138	Pos
	76 / F	progressive	occult (silent) form	106	Pos
	65 / M	progressive	cranial and PMR, with hoarseness	48	Neg

TA: temporal arteritis; PMR: polymyalgia rheumatica; TAB: temporal artery biopsy; JC: jaw claudication; AF: amaurosis fugax; PVL: permanent visual loss; PUOM: pain upon opening mouth; VH: visual hallucinations; Ns: not specified; Nd: not determined.

presumed diagnosis of sinusitis, since they presented with headaches, facial or periorbital pain and raised ESR. In other patients, the sudden onset of HNS or a concurrent involvement of the tongue, lips or cheek mucosa led to misdiagnose TA as allergic oedema.

The appearance of HNS in TA was varied and was in no way pathognomonic. The most common picture was a bilateral (or less often unilateral) painful, slightly pink, non-pitting swelling of the cheeks and maxillae, but other areas could be affected, including the eyelids, orbital region, neck, forehead, cheek mucosa and tongue. Thus, these patients can be first seen by an ear, nose and throat specialist or an ophthalmologist or be sent to the emergency room, before the possibility that a vasculitic process is taken into consideration.

Facial or neck swelling appears to be an underreported early feature of GCA, since it was observed in a sizeable proportion of our patients, nearly 7%. Furthermore, this figure is a minimal estimate since no specifically designed item was devoted to HNS in our prospective questionnaire. Moreover, in many cases the diagnosis of HNS was only based on the patient's history. In a prospective study of 345 patients with TA, the frequency of facial oedema was 12% (27). The discrepancy existing between the frequency of HNS in our study and the aforementioned study, and the rarity ascribed to this finding in the literature, is best explained by the use of questionnaires and prospective study designs, which allow the recognition of discrete and

Table II. Characteristics of head-and-neck swelling in patients with concurrent temporal arteritis.

Author (ref)	Location	Characteristics	Outcome
Cohen (1)	neck, face, cheek, periorbital (bilat)	n.s.	settled within 2 days with CS
Herreman (16)	periorbitar	n.s.	n.s.
	eyelid, lips, limbs	pinkish, migratory, discretely tender	n.s.
Acetta (2)	forehead, face, maxillae (bilat)	painful, fluctuating	n.s.
Delvigne (17)	periorbitar (bilat)	thickened, tender maxillary arteries	settled without CS, relapsed and subsided with CS
Manganelli (4)	lower part of the face (bilat)	n.s.	settled within 3 days with CS (80 mg/d)
Chevalet (19)	face, cheek	skin tenderness facing swelling	settled without treatment
Ghanchi (3)	face, periorbital	"heavy" feeling after swelling had settled	settled without treatment
Plantin (18)	face, periorbitar (bilat)	pinkish, more prominent in the morning	settled with CS (15 mg)
	periorbitar (bilat)	pinkish, tender	settled within a few days with CS (25 mg)
	lower part of the face, scalp	n.s.	settled after a short CS cure, multiple relapses then subsided with continuous CS (20 mg/d)
Gaches (21)	cheek, eyelid (bilat)	n.s.	settled rapidly with CS
	cheek (bilat)	relapsing course	settled rapidly with CS
Salafi (20)	face, supraclavicular space (bilat)	n.s.	settled rapidly with CS but relapsed
	lower part of the face	pinkish	settled rapidly
Sarrot-Reynaud (22)	neck, face, cheek (bilat)	"inflammatory" appearance	settled within 10 days with CS (1 mg/kg)
Paris (23)	cheek, periorbital, neck	pinkish, sensible	settled rapidly with CS
Paulus (24)	periorbital, malar region	pinkish, pitting, painless	settled within 4 days with CS (1 mg/kg), numerous relapses
Regouby (25)	lower part of the cheek (bilat)	pinkish, pitting, tender, enhancing overnight	settled rapidly with CS (60 mg/d) but relapsed
Myara (26)	face, eyelids (bilat)	white, pitting, painless	settled within a few days with CS (0.7 mg/kg/d)
Current series	face, maxillae, chin, neck (bilat)	painless, "non inflammatory", lasted 1 month	settled without treatment
	face, neck, supraclavicular, tongue	associated with segmental limb swellings, lasted 2 weeks	settled without treatment
	periorbital, eyelids (bilat)	preceded by orbital and facial pain	settled quickly without treatment
	periorbital, eyelids (bilat)	preceded by eye and orbital pain	settled quickly without treatment
	face, temporal fossa (bilat)	short-lived (3 days)	settled without treatment
	neck (anterior-lateral aspects) (bilat)	n.s.	settled rapidly with intravenous CS
	periorbital, eyelid	waning and waxing course	settled within 1 month without treatment
	face, cheek and neck (bilat)	n.s.	settled rapidly with CS (20 mg/d)
	face, cheek	n.s.	settled rapidly with CS, relapsed (6th week), subsided definitively (6th mo)
	neck (anterior-lateral aspects) (bilat)	waning and waxing course	settled within 2 weeks without treatment
	periorbital, eyelids (bilat), root of nose	short-lived (5 days)	settled quickly with CS (0,7 mg/kg)
	face, cheek	short-lived (7 days)	settled without treatment
	face	painless, short-lived (a few days)	settled without treatment
	eyelids, neck (submaxillary), tongue	painless, short-lived	settled quickly with CS (20 mg/d)
	face, cheek, lateral aspect of the neck (bilat)	"non inflammatory", painless	settled quickly with CS (25 mg/d)
	face, cheek (bilat)	short-lived (two episodes)	settled without treatment
	cheek, maxillae, neck (lateral aspect)	"non inflammatory", painless	settled without treatment

Ns: not specified; CS: corticosteroids.

short-lived forms of HNS.

Patients with HNS in the setting of TA shared some important characteristics. Though the swelling was often an early or even the first manifestation of the disease, it remained very rarely the sole finding for prolonged periods (2). Moreover, less than 10% of the patients presented without headaches or jaw claudication and in none of them was HNS the only cranial symptom of GCA. The most striking finding in our study was that 80% of the patients had ENT symptoms and nearly 30% developed trismus. Patients with GCA complain of varied ear-jaw-mouth-throat symptoms in up to 57% of the cases (13). These symptoms are probably caused by an inflammatory involvement of branches of the external carotid artery such as facial, internal maxillary and ascending pharyngeal arteries, although this has been rarely documented (28, 32). Trismus has been only occasionally reported in TA (29, 33-35), where it may be considered as the greatest degree of jaw claudication (35). Although such an association of HNS with trismus may be overemphazized due to publication biases, this is unlikely in our unselected series, where 3 out of 4 patients who presented with frank trismus, also had HNS. The cause of HNS in TA is unclear, but in most cases its almost simultaneous appearance with facial or periorbital pain, jaw claudication or trismus as well as other ENT symptoms and a positive TAB result, and its prompt regression with corticosteroid treatment suggest that it may be due to a widespread inflammatory involvement of the external carotid tree. Carotid tenderness, which points to an inflammatory involvement of the external carotid artery itself in patients with TA (36), might also rely on HNS. Unfortunately, this feature was not included as a specific item in our questionnaire and examination of the external carotid artery was generally left aside in Döppler studies. Alternatively, abnormal release of vascular endothelial growth factor, a protein that both induces hyperpermeability and stimulates endothelial cell growth (37), could play a role in the development of local or distant swellings in some patients with GCA (38, 39).

Contrary to previous reports (3, 40), our study showed no association of facial swelling with sight-threatening complications, although one-half of the patients reported either transient ischemic visual symptoms or eye pain. Friedman and Friedman described the sensation of puffiness of the face without altered facial appearance as a predictor of visual disturbance in TA (40). Similarly, Ghanchi *et al.* described a patient in whom such a sensation preceded objective facial swelling and visual impairement (3). Visual loss in GCA could be associated, therefore, with subjective rather than true facial puffiness. Likewise, no patients with HNS developed stroke, a manifestation that has been strongly associated with permanent visual loss (41). Why patients with TA and HSN seldom develop irreversible visual loss is unclear. However, most patients described in this study presented with an acute disease onset and an otherwise typical picture of cranial arteritis, which may have prompted caregivers to act preventively before the development of irreversible complications. Finally, variations in the clinical presentation of GCA were found to be correlated with cytokine mRNA expression, the risk of visual ichemic complications being higher in patients whose temporal artery biopsy express high amounts of interferon γ-mRNA (42, 43). One can therefore speculate about the possibility of different cytokine profiles in GCA patients with cranial arteritis and HNS and without.

HNS associated with TA ran a benign course but recurred in some patients in attempts to taper corticosteroids, pointing to a poorly controlled underlying process. In these patients the recurring facial swelling could not be attributed solely to a corticosteroid side-effect because it responded promptly to further increases in the prednisone dose. Similarly, sinusitis with facial swelling (44) was reasonably ruled out, since none of the patients were receiving antibiotics during the swelling relapse or subsequently.

In conclusion, HNS appears to be an under-recognized, early feature of TA. Objective puffiness of the face and/or neck is strongly associated with ENT symptoms, particularly jaw claudication or trismus and a positive TAB, but not visual loss or stroke. Such characteristics imply in these cases a widespread involvement of the external carotid artery system by GCA, with relative sparing of the internal carotid artery system. This case series of patients with HNS underlines the preferential attack of the external carotid tree by GCA for reasons that remain to be elucidated.

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