British Journal of Rheumatology 1996;35:1161–1168

CLINICAL REVIEW

A PROSPECTIVE STUDY OF 287 PATIENTS WITH POLYMYALGIA RHEUMATICA AND TEMPORAL ARTERITIS: CLINICAL AND LABORATORY MANIFESTATIONS AT ONSET OF DISEASE AND AT THE TIME OF DIAGNOSIS

G. MYKLEBUST and J. T. GRAN

Department of Rheumatology, Central Hospital of Aust Agder, Arendal, Norway

SUMMARY

A prospective study of 287 patients with giant cell arteritis (GCA), including polymyalgia rheumatica (PMR) and temporal arteritis (TA), was conducted during 1987–1994. All patients were evaluated prior to the start of drug treatment. During the same period, 31 patients with GCA, of whom 12 cases had TA, were admitted to other departments in the hospital. At onset of disease, all patients were $\geqslant 50$ yr of age. Peripheral arthritis was found in 24.4% of patients with PMR, while none of the patients with TA exhibited such manifestations. Clinical features at onset of disease differed from those appearing at presentation to the hospital. Thus, the gradual development of a full-blown clinical picture may be responsible for the delay in diagnosis of GCA. The majority of cases (80%) presented with 'pure' PMR without clinical signs or symptoms of concomitant TA. In a random sample of 68 patients with 'pure' PMR, histological examinations of biopsy specimens of the temporal artery revealed inflammatory changes in three patients only (4.4%). Consequently, arterial biopsy in patients with clinical features of PMR only, appears to be unnecessary. Among patients with TA referred to the department of internal medicine, general malaise, loss of weight and sustained fever were prominent manifestations. Such features may thus necessitate a diagnostic arterial biopsy even in the absence of clinical arteritis or myalgia. Both ESR and CRP were within normal levels in 1.2% of the cases. Further clinical and laboratory examinations performed at diagnosis of GCA disclosed only one case of malignancy. Routine chest X-rays did not reveal unexpected pathological findings. Permanent and complete blindness due to arteritis was observed in one patient only. No association between GCA and thyroid dysfunction was detected.

KEY WORDS: Polymyalgia rheumatica, Temporal arteritis, Clinical features, Blindness, Cancer, Thyroid.

POLYMYALGIA rheumatica (PMR) and temporal arteritis (TA) are usually considered as two different clinical expressions of the same disease, often termed giant cell arteritis (GCA). The clinical and laboratory features of the disorder have been the subject of numerous surveys during the last few decades. However, the majority of studies on GCA have been conducted on selected patients referred to hospital, whereas population surveys or observations of unselected cases have been performed more infrequently.

The aim of the present investigation was to study the clinical and laboratory manifestations of GCA prior to the start of drug therapy in an unselected group of patients referred to hospital. Moreover, the prospective design of the study enabled us to evaluate the course, duration and mortality of the disease, and the incidence of malignancy among the patients. We now report on the clinical and laboratory findings at onset and at presentation.

MATERIALS AND METHODS

Geography

The county of Aust Agder, South Norway, is mainly located on the coast, and in the period 1987–1994 the total population averaged 98 000 inhabitants, of whom 29% were $\geqslant 50$ yr old.

Submitted 25 January 1996; revised version accepted 30 April 1996.

Correspondence to: J. T. Gran, Department of Rheumatology, Regional Hospital of Tromsø, 9038 Tromsø, Norway.

Methods

Prior to the start of the study, all physicians in the county of Aust Agder were informed of the study, and asked to refer all patients suspected of having PMR or TA to the department of rheumatology as soon as possible and before initiating drug therapy. The county of Aust Agder has one general hospital, including departments of rheumatology, internal medicine, geriatrics and ophthalmology. There are no private practising rheumatologists in the region.

Patients diagnosed by the department of rheumatology with a disease onset in the period 1987–1994 were included in the study. They were followed up at 3 month intervals during the first year, and yearly thereafter until cessation of therapy and permanent disease remission. All patients were examined by a rheumatologist. In addition, all hospital records with a diagnosis of either PMR or TA were carefully reviewed to estimate the number of patients with GCA who had been treated by the departments of internal medicine, geriatrics and ophthalmology. Ophthalmological examinations were carried out only if patients reported significant visual disturbances.

The ACR criteria [1] for TA were applied, but only cases with histologically proven GCA were included. For PMR, patients meeting either the criteria suggested by Bird *et al.* [2] or those of Hamrin [3] were selected for study. Thus, all patients with PMR included in the

present study had bilateral pain and/or stiffness of the shoulder and/or pelvic girdle which could not be explained by diseases other than GCA.

Careful examination of clinical history at onset of complaints and at presentation were recorded. The onset was defined as acute when the patient could date the initial complaints to the nearest day, as subacute to the nearest week, as gradual to the nearest month and slowly to the nearest year.

Laboratory examinations

The following blood tests were performed in all patients: complete blood count including white cell count and thrombocytes, haemoglobin, erythrocyte sedimentation rate (ESR) (Westergren), C-reactive protein (CRP), creatinine, creatine kinase, alanine aminotransferase (ALAT), aspartate aminotransferase, gamma glutamyltranspeptidase (GT), alkaline phosphatase (ALP), thyroxin (T4), thyroid-stimulating hormone (TSH), serum electrophoresis, uric acid, rheumatoid factors (Waaler's test) and antinuclear antibodies.

Statistics

Chi-square test, Fisher's exact test and Student's t-test were used for statistical analyses, and a P value of $\leqslant 0.05$ was accepted as significant.

RESULTS

Diagnostic classification

Prospective study. There were 287 patients who were diagnosed and treated by the department of rheumatology (Table I). Of these, 233 patients had pure PMR (group I) (81.2%), 39 patients had TA without PMR (group II) (13.6%), and 15 patients had both PMR and TA (group III) (5.2%). The ratios of females to males were 1.8 in group I, 2.8 in group II and 3.2 in group III ($P > 0.05$).

Retrospective group. Altogether 31 patients were diagnosed by the departments of ophthalmology, geriatrics and internal medicine during the study period (Table I). Of these, 19 patients had pure PMR, 10 patients had TA, and two cases suffered from both PMR and TA. In addition, there were 17 patients in whom drug treatment had been started by the general practitioner prior to referral to hospital. The 48 patients were not further evaluated with respect to clinical and laboratory manifestations.

TABLE I
The number of cases included in the present study (prospective study) and cases diagnosed by others in the same period (retrospective study)

	Number of cases		All cases	
Classification	Females	Males	Number	%
Prospective study				
I PMR	150	83	233	81.2
II TA	29	10	39	13.6
III PMR + TA	12	3	15	5.2
All cases (GCA)	191	96	287	100.0
Retrospective study				
I PMR	28	7	35	72.9
II TA	7	3	10	20.8
III PMR + TA	1	2	3	6.3
All cases				
I PMR	178	90	268	80.0
II TA	36	13	49	14.6
III PMR + TA	13	5	18	5.4
All patients	227	108	335	100.0

Altogether 335 patients with GCA had been seen by the hospital during the study period. PMR exclusively was diagnosed in 268 cases (80.0%), 49 cases had TA only (14.6%), while both PMR and TA were present in 18 cases (5.4%). Of all patients, a biopsy-verified diagnosis of TA was documented in 67 cases (20%).

Demography

There were 191 females and 96 males (ratio 2:0) (Table I). Mean age at onset for all patients was 71.7 yr (range 53–89 yr) in group I, in group II 70.4 yr and in group III 74.4 yr. Males and females had similar age at onset.

Delay of diagnosis

The mean delays in diagnosis were 2.9 months (range 0.25–18 months) in group I, 1.5 months (range 0.25–7 months) in group II and 1.9 months (range 0.5–5 months) in group III. The mean duration of complaints prior to diagnosis for all patients was 2.6 months (females 2.5 months, males 2.9 months). A final diagnosis of GCA was obtained within 6 months in 95% of the cases. The mean diagnostic delay did not change significantly during the period 1987–1993.

Type of onset

An acute or subacute onset of complaints was recorded in 56.3% of the patients with PMR (group I). In TA (group II and those of group III who started with TA), the onset was acute or subacute in 74.5% of cases. The difference between group I and group II/III was statistically significant ($P < 0.05$).

Symptoms at onset and at presentation

The symptoms recorded at onset of disease and at presentation to hospital are given in Tables II (PMR) and III (TA). There were no significant differences between males and females with regard to clinical manifestations.

In PMR, the most frequent complaint was pain and stiffness of the shoulder region, being present in 21.1% at onset of disease, while at presentation 78.0% of patients with PMR reported such symptoms. Pain and stiffness in the proximal parts of the extremities (neck, shoulders, proximal arms, thighs and hips) were present in less than a quarter of patients at onset, but at presentation the majority of patients had such complaints. Night sweats and loss of weight were infrequently (<5%) reported at onset, but 42.2 and 28.9%, respectively, of patients had such manifestations when examined at the time of diagnosis.

In TA, headache was present initially in 54.1% of the patients, while at presentation 84.6% of them

TABLE II
Initial complaints among patients with polymyalgia rheumatica

Localization	Initially (n = 190) (%)	At presentation (n = 232) (%)
Shoulders	21.1	78.0
Neck	18.4	55.2
Thighs	16.3	65.5
Proximal arms	14.2	56.5
Lumbar spine	11.6	21.1
Peripheral joints (except shoulders and hips)	11.1	31.9
Malaise	9.5	27.6
Unilateral shoulder	8.4	<5
Hips	8.4	37.1
Whole body	7.4	11.2
Flu	6.8	<5
Buttocks	<5	14.7
Popliteal	<5	25.4
Night sweats	<5	42.2
Weight loss	<5	28.9
Night pain	NR*	21.6

In <5% of patients, the initial complaints were: fever, unilateral buttock, unilateral proximal arms, distal arms, inguinal regions, unilateral thigh and distal legs. Morning stiffness of affected regions was present in 77.6% at presentation.
Jaw claudication was reported by 6.1% of cases at diagnosis.
*Not recorded.

complained of headache (Table III). Twenty-four per cent of the patients started with classical temporal headache. At presentation, occipital headache (16 cases) was almost as frequent as headache localized to the temporal regions (19 cases).

Table IV shows the various causes for referral to hospital among the patients diagnosed by the departments of geriatrics, internal medicine and ophthalmology. The major cause for referral was general malaise, being responsible for 32.3% of the hospital admissions. Other frequent causes for referral to hospital were sustained fever (19.4%), weight loss (9.7%), and development of osteoporotic fractures (9.7%) and diabetes induced by the administration of corticosteroids (9.7%).

Peripheral arthritis

Among patients in group I, 24.4% had clinically detectable inflammation of the peripheral joints. Of these, 58.4% were localized to the knees, 35.8% to the wrists, 22.6% to the MCPs, 15.1% to the PIPs, 9.4% to the ankles, 7.5% to the sternoclavicular joints and 1.9% to the hips. Polyarthritis was found in 3.8%, whereas 1.9% had tenosynovitis of the flexor tendons of the hands. Popliteal cysts were found in 13.2% of the patients.

Among patients with TA, no patient had peripheral arthritis. The difference between PMR and TA regarding the frequency of peripheral arthritis was statistically significant ($P < 0.05$).

Eye complications and malignancy

One patient presented with bilateral complete and permanent loss of vision. No other patient had clinically significant lasting impairment of vision, although another seven patients with TA reported some loss of vision when examined at diagnosis. Review of the hospital records of patients admitted to the department of ophthalmology did not disclose a single case of TA or PMR with visual disturbances who were not registered in the prospective study.

One patient had concomitant lung cancer. No other neoplastic disease was found at presentation. In none of the patients seen by the departments of internal medicine and geriatrics was malignancy discovered at onset of GCA.

Biopsies of the temporal arteries

All patients classified as TA had transmural cellular infiltrates in the temporal artery. Giant cells were seen in 70.9%, fibrinoid necrosis in 36.4% and thrombolization in 16.4%. Bilateral biopsies were obtained when clinical evidences of arteritis was present on both

TABLE III
Symptoms at onset and at diagnosis of 37 patients with temporal arteritis

Complaint	At onset (n = 37) No.	%	At diagnosis (n = 39) No.	%
Headache	20	54.1	33	84.6
Temporal	9	24.3	15	38.5
Occipital	7	18.9	7	17.9
Frontal	2	5.4	1	2.6
Frontal/occipital	2	5.4	5	12.8
Temporal/occipital	0	0.0	4	10.5
Generalized	0	0.0	1	2.6
Fatigue	11	29.7	16	41.0
Fever	10	29.7	17	43.4
Jaw claudication	1	2.7	11	28.2
Night sweats	1	2.7	10	25.6
Weight loss	0	0.0	16	41.0
Loss of appetite	0	0.0	5	12.8
Taste changes	0	0.0	4	10.2
Visual impairment	1	2.7	7	17.9

TABLE IV
Cause of referral to departments of internal medicine, geriatrics and ophthalmology (%) among patients diagnosed as having polymyalgia rheumatica and temporal arteritis

	Polymyalgia rheumatica (n = 19)	Temporal arteritis (n = 12)	All cases (n = 31)
Malaise	15.8	58.3	32.3
Fever	10.5	33.3	19.4
Weight loss	2.6	16.7	9.7
Diabetes	15.8	0.0	9.7
PMR	15.8	0.0	9.7
Osteoporosis	10.5	0.0	6.5
Headache	0.0	16.7	6.5
Gastrointestinal bleeding	5.2	0.0	3.2
Transient stroke (TIA)	5.2	0.0	3.2
Anaemia	5.2	0.0	3.2
Stroke	5.2	0.0	3.2
Cholecystitis	5.2	0.0	3.2
Pneumonia	5.2	0.0	3.2
Pleuritis	0.0	8.3	3.2
Lymphadenopathy	0.0	8.3	3.2

sides. Bilateral arteritis was found in 11 cases in group II and in seven cases in group III.

Biopsies of the temporal artery were performed in a random sample of 68 patients with PMR. None of these patients had clinical features of arteritis, but three cases (4.4%) had histological changes compatible with a diagnosis of arteritis. The three patients were included in group III.

Laboratory examinations

The mean values of ESR and CRP are shown in Table V. In the total group, one patient had an ESR of $\leqslant 10$ (0.4%) and five patients had an ESR of $\leqslant 20$ (1.9%). A CRP value of < 10 was recorded in 10 out of 221 patients (4.5%). Both ESR and CRP were increased in 93.4% of cases, ESR normal and CRP elevated in 1.6%, CRP normal and ESR increased in 3.7%, and normal ESR and normal CRP were found in 1.2% of the patients.

Leucocytosis was observed in 32.9% and thrombocytosis in 37.4%. There were no statistically significant differences between groups I, II and III with regard to the frequency of leucocytosis and thrombocytosis.

Elevated serum levels of the liver enzymes ALAT, GT and ALP were found in 13.8, 29.2 and 8.5%, respectively. The number of patients with elevated levels of ALP was significantly ($P < 0.02$) higher among cases in groups II and III (18.2%) compared to those in group I (6.0%).

Routine chest X-rays were performed in 160 patients. Normal findings or abnormalities that were previously recognized were observed in 156 patients (97.5%). New pathological findings were demonstrated in four patients. Two patients had pathological amounts of pleural fluids which disappeared during treatment with corticosteroids. One patient had diffuse pulmonary infiltrates which after initiation of corticosteroid treatment were no longer seen on subsequent chest X-rays. The last patient presented with cough, dyspnoea and haemoptysis, and chest X-rays suggested lung cancer. Subsequent histological examination of the lung tumour disclosed malignancy.

TABLE V

Laboratory findings among patients with polymyalgia rheumatica (group I), temporal arteritis (group II), and both polymyalgia rheumatica and temporal arteritis (group III)

Laboratory variable	Group I	Group II	Group III	All cases
ESR				
Mean values	72.0	92.1	91.4	75.5
% elevated	97.7	100.0	100.0	98.1
CRP				
Mean values	68.7	104.6	100.3	75.6
% elevated	94.8	97.1	100.0	95.5
ESR and CRP				
% with both normal	1.5	0.0	0.0	1.2
White blood cells				
% with elevations	35.5	25.0	30.8	32.9
Thrombocytes				
% with elevations	35.8	39.4	60.0	37.4
ALP				
% with elevations	6.0	15.6	25.0	8.5

Determinations of serum TSH and T4 were carried out in 142 cases. Three patients had hypothyroidism that was already under treatment, while unrecognized hypothyroidism was disclosed in two patients (2.6%). Hyperthyroidism was not observed.

DISCUSSION

The sex ratio and mean age at onset of our patients were similar to those of previous reports on GCA [1, 4–13]. Although patients with disease onset prior to age 50 have been reported [6, 12, 14–17], all of the present patients were aged $\geqslant 50$ yr at onset of GCA. Our findings thus support those [1, 5] suggesting that an age at onset of $\geqslant 50$ yr should be regarded as a useful diagnostic criterion for GCA. Why GCA manifests in old age and shows a preference for females is at present unknown.

There is considerable controversy regarding the prevalence of TA in PMR [17–22]. In the USA, between 0 and 22% of PMR patients have histological evidence of concomitant arteritis [23]. In Scandinavian reports, however, the chance of finding TA in PMR without clinical evidence of arteritis appears higher, of the order of 40–45% [22, 23]. It was, therefore, rather surprising that biopsies of the 68 patients with pure PMR disclosed TA in only three patients (4.4%). Moreover, only 4% of our total patient group could be classified as having both PMR and TA. Thus, only 19.5% of our patients had TA, a figure contrasting with most previous reports [8, 9, 15, 24–30] which conclude with a frequency of TA in GCA of 29.5–56.1%. However, the vast majority of previous reports have been based on patients attending referral centres, and are consequently not directly comparable to the present population-based survey. The rather pronounced variance in the reports may also have been caused by differences in study design and use of inclusion criteria, as well as variation in the employment of diagnostic criteria for TA. Our findings correspond to those of Cimmono *et al.* [7], who found TA in 17.2% of all patients with GCA.

It can also be argued that if routine biopsies of the temporal arteries had been performed for all our cases, the number of cases of TA would have been greater. However, all patients with either symptoms or signs of TA were examined histologically, and the low prevalence of arteritis in pure PMR cases indicates that rather few cases of TA have been missed. In a recent study of similar design, Noltorp and Svensson [31] failed to disclose a single case of arteritis among 17 patients with pure PMR. Furthermore, the low number of cases with TA diagnosed by the departments of internal medicine and ophthalmology strongly supports the conclusion that the majority of cases with GCA do not present evidence of concomitant arteritis.

According to the results of the present study and those of Mertens *et al.* [32], there is no need for a biopsy of the temporal artery in patients with a clinical diagnosis of PMR without clinical evidence of arteritis. It should be noted, however, that the present report

deals exclusively with clinical manifestations occurring during the very early stages of GCA. It is conceivable that some patients with PMR may develop arteritis when seen at subsequent follow-up examinations. Finally, indications for performing diagnostic biopsy of the temporal artery may differ among patients admitted to departments of rheumatology and departments of internal medicine. As clearly shown by the present study, general symptoms such as sustained fever, malaise and weight loss were major causes for referral of patients with TA to the department of internal medicine (Table IV). Thus, a diagnostic biopsy of the temporal artery may be necessary in such patients provided infectious and malignant diseases have been appropriately excluded.

Another possible result of the design of this study was the rather short mean interval between the onset of complaints and diagnosis (2.8 months for PMR and 1.5 months for TA), which contrasted with earlier reports of a mean diagnostic delay of >6 months [11, 12, 33]. We believe that the rather short duration of symptoms prior to diagnosis in our study enabled us to study the clinical manifestations of the cases in detail.

More than half of the patients with pure PMR had a rather abrupt onset of disease, and almost three-quarters of the patients with TA started rather suddenly. Thus, in agreement with some previous observations [7, 12], GCA appears as a disease which clinically manifests rather abruptly. A slow development of GCA (over months) was observed in only 13.2% of the present cases.

However, although the start of PMR appears rather sudden, the development of typical symptoms may take some time. In PMR, there was a marked difference between the clinical manifestations at onset and those at diagnosis (Table II). The disease often started unilaterally, with general symptoms only, or with symptoms localized to either the upper or lower muscle groups, and then gradually evolved into a clinical picture characterized by symmetrical proximal muscle stiffness affecting all four extremities. The rather slow evolution of well-known clinical features of PMR may thus be responsible for the frequently recorded diagnostic delay in this disease.

In TA, headache was present at onset in 54.1% of patients, whereas at diagnosis 84.6% of the patients complained of significant headache. It is also of diagnostic importance that the headache in patients with TA was rather frequently localized to the frontal and occipital regions, and not consistently to the temporal parts of the head. Such findings have also been observed by previous authors [16, 34–36].

The frequency and distribution of peripheral joint inflammation in PMR equalled that of most previous observations [7, 37–40]. However, the particular absence of peripheral arthritis in patients with TA has been infrequently noted by previous workers [31]. Why PMR and not TA is associated with peripheral arthritis is not easily explained. One putative explanation is that PMR and TA are two clinical expressions of the same disease, PMR being dominated by muscle and joint manifestations, while TA represents the more systemic form, presenting most often with arteritis. However, overlapping forms and intergroup transitions are not infrequently observed. Further studies of genetic markers, for example HLA antigens [41, 42], may reveal factors responsible for the various clinical expressions.

The most serious manifestation of TA is probably unilateral and bilateral blindness. The reported frequencies of loss of vision vary considerably, ranging from 0 to 50% [9, 11, 13, 14, 16, 17, 21, 28, 29, 35–37, 43–47], depending to some degree on the design of the study. Retrospective studies based on patients attending hospital clinics, departments of ophthalmology in particular, generally reveal higher incidence rates of ocular manifestations than prospective population-based surveys (Table VI). Although seven of our patients had some visual impairment at presentation, only one patient (2.7%) with complete and permanent loss of vision was observed in the present study. This patient presented with sudden onset of bilateral amaurosis fugax and was shown to have TA. We cannot completely explain the rareness of eye complications among our patients. A putative explanation could have been that some cases with loss of vision had been referred to the department of ophthalmology [48]. However, reviewing all records with a diagnosis of TA disclosed only two patients with loss of vision diagnosed by ophthalmologists during the period of the study. The two patients were, however, registered by us and included in the prospective survey. Another possible explanation is that the lack of regular eye examinations of our patients is responsible for the low frequency of visual impairment. Some cases may have developed less severe eye complications without being appropriately diagnosed. Thirdly, initiation of treatment with oral corticosteroids was started rather rapidly in all patients, hence the short interval from the onset of complaints to the start of therapy may have inhibited the development of ocular complications. There is, however, no general agreement as to whether or not treatment with steroids prevents or lowers the incidence of eye manifestations [49, 50]. Thus, according to the results of the present study, the development of important loss of vision among patients with GCA is rather infrequent, and may have been previously overestimated by the selection of hospitalized patients exclusively.

Much attention has been paid to the possible association between GCA and concurrent malignant disease [10, 30, 32, 51]. Among our patients, only one case of neoplastic disorder was found during the initial stage of GCA. Of particular interest is the absence of malignancy among our patients with TA since this type of GCA has been previously associated with neoplastic disease [30]. Earlier reports have, however, been mostly based on patients attending tertiary referral centres which are often subjected to a significant risk of selection bias. Our cases were derived from a

TABLE VI
Incidence of ocular manifestations in temporal arteritis

Author	Year	Cases (no.)	Positive biopsy (%)	Loss of vision (%)	Visual disturbances (%)	Reference
Hospital-based studies (departments of medicine and rheumatology)						
Wadman	1972	53	90	19	26	47
Fauchald	1972	61	100	0	7	17
Sørensen	1977	46	100	28	–	21
Malmvall	1978	42	100	0	19	60
Fainaru	1979	47	100	30	47	36
Dare	1980	25	100	16	–	61
Desmet	1990	34	100	6	29	35
Gonzales-Gay	1992	57	100	16	30	62
Aiello*	1993	245	83	14	–	50
Hospital-based studies (eye departments)						
Jonasson	1979	136	100	65	–	16
Graham	1981	90	100	48	–	45
Population-based studies						
Huston	1978	42	90	20	40	46
Bengtsson	1981	74	100	12	16	29
Smith	1983	26	81	21	29	44
Boesen	1987	15	100	0	0	9
Machado	1988	94	94	13	30	43
Franzen	1992	15	100	0	0	63
Baldursson	1994	133	91	–	14	13
Present	1996	54	100	2	15	–

*All departments included.

prospective study based on cases visiting general practitioners, a study design which may have reduced the over-representation of cases suffering from both GCA and malignant disease. We therefore tentatively conclude that the prevalence of malignancy in the early stages of GCA does not exceed that of the general population. Further follow-up of the present cases will reveal possible associations with malignancy during the subsequent disease course.

The single case with lung cancer was diagnosed with the help of routine chest X-rays. However, pulmonary symptoms were prominent, including recent development of cough, haemoptysis and dyspnoea. None of the other patients had findings on routine chest X-rays which necessitated further clinical and laboratory examinations. Thus, we suggest that routine chest X-rays are unnecessary in the initial stages of GCA unless clinical manifestations of pulmonary disease are presented.

Routine laboratory examinations of the present patients revealed findings similar to most previous reports [9, 35, 52, 53]. However, we cannot support the impression gained from single case reports [34, 54–56] that GCA not infrequently presents with normal levels of ESR and CRP. Only 1.2% of our patients had both normal ESR and CRP at diagnosis. Moreover, the present study failed to support previous suggestions of an association between GCA and thyroid disease [57]. This lack of association has also been noted by previous workers [58, 59].

We finally conclude that, based on an unselected group of patients, GCA appears as a disease which is infrequently accompanied by loss of vision and co-existent malignant disease, and that the overwhelming majority of patients present with musculoskeletal manifestations without clinical and histological evidence of TA. These findings will be further evaluated as our patients all participate in an ongoing prospective follow-up survey.

References

1. Hunder GG, Bloch DA, Michel BA *et al.* The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum* 1990; **33**:1122–8.
2. Bird HA, Esselinckx W, Dixon ASTJ, Mowat AG, Wood PHN. An evaluation of criteria for polymyalgia rheumatica. *Ann Rheum Dis* 1979;**38**:434–9.
3. Hamrin B. Polymyalgia arteritica. *Acta Med Scand* 1972;**533**(suppl.):1–13.
4. Rajala SA, Ahvenainen JE, Mattila KJ, Saarni MI. Incidence and survival rate in cases of biopsy-proven temporal arteritis. *Scand J Rheumatol* 1993;**22**:289–91.
5. Catoggio LJ, Soriano ER, Imamura PM. Treatment of polymyalgia rheumatica: lower initial dose. *Br J Rheumatol* 1991;**30**:393–4.
6. Nordborg E, Bengtsson BÅ. Epidemiology of biopsy-proven giant cell arteritis (GCA). *J Int Med* 1990; **227**:233–6.
7. Cimmino MA, Caporali R, Montecucco CM, Rovida S, Baratelli E, Broggini M. A seasonal pattern in the onset of polymyalgia rheumatica. *Ann Rheum Dis* 1990;**49**: 521–3.
8. Delecoeuillerie G, Joly P, de Lara AC, Paolaggi JB. Polymyalgia rheumatica and giant cell arteritis: a retrospective analysis of prognostic features and different corticosteroid regimens (11 year survey of 210 patients). *Ann Rheum Dis* 1988;**47**:733–9.
9. Boesen P, Sørensen SF. Giant cell arteritis, temporal arteritis, and polymyalgia rheumatica in a Danish county. *Arthritis Rheum* 1987;**30**:294–9.
10. von Knorring J. Treatment and prognosis in polymyalgia

rheumatica and temporal arteritis. *Acta Med Scand* 1979;**205**:429–35.
11. Coomes EN, Ellis RM, Kay AG. A prospective study of 102 patients with the polymyalgia rheumatica syndrome. *Rheum Rehabil* 1976;**15**:270–6.
12. Mowat AG, Hazleman BL. Polymyalgia rheumatica—a clinical study with particular reference to arterial disease. *J Rheumatol* 1974;**1**:190–202.
13. Baldursson O, Steinsson K, Bjørnsson J, Lie JT. Giant cell arteritis in Iceland. *Arthritis Rheum* 1994;**37**:1007–12.
14. Østergård J, Andersen K, Kæmpecellearteritt. *Ugeskr Læger* 1985;**147**:1678–82.
15. Behn AR, Perera T, Myles AB. Polymyalgia rheumatica and corticosteroids: how much for how long? *Ann Rheum Dis* 1983;**42**:374–8.
16. Jonasson F, Cullen JF, Elton RA. Temporal arteritis. *Scott Med J* 1979;**24**:111–7.
17. Fauchald P, Rygvold O, Øystese B. Temporal arteritis and polymyalgia rheumatica. *Ann Intern Med* 1972; **77**:845–52.
18. Fernandez-Herlihy L. Temporal arteritis: clinical aids to diagnosis. *J Rheumatol* 1988;**15**:1797–801.
19. Robb-Nicholson C, Chang RW, Anderson S *et al.* Diagnostic value of the history and examination in giant cell arteritis: a clinical pathological study of 81 temporal artery biopsies. *J Rheumatol* 1988;**15**:1793–6.
20. Healey LA. The relationship of polymyalgia rheumatica to giant cell arteritis—single or separate syndromes? *J Rheumatol* 1986;**13**:1190.
21. Sørensen PS, Lorenzen I. Giant cell arteritis, temporal arteritis and polymyalgia rheumatica. *Acta Med Scand* 1977;**201**:207–13.
22. Lantin JP, Duc J. Giant cell arteritis: a diagnostic and therapeutic update. *EULAR Bull* 1991;**4**:124–6.
23. Healey LA. On the epidemiology of polymyalgia rheumatica and temporal arteritis. *J Rheumatol* 1993; **20**:1639–40.
24. Myles AB, Perera T, Ridley MG. Prevention of blindness in giant cell arteritis by corticosteroid treatment. *Br J Rheumatol* 1992;**31**:103–5.
25. Bowness P, Shotliff K, Middlemiss A, Myles AB. Prevalence of hypothyroidism in patients with polymyalgia rheumatica and giant cell arteritis. *Br J Rheumatol* 1991;**30**:349–51.
26. Lundberg I, Hedfors E. Restricted dose and duration of corticosteroid treatment in patients with polymyalgia rheumatica and temporal arteritis. *J Rheumatol* 1990; **17**:1340–5.
27. Kyle V, Tudor J, Wraight EP, Gresham GA, Hazelman BL. Rarity of synovitis in polymyalgia rheumatica. *Ann Rheum Dis* 1990;**49**:155–7.
28. Jones JG, Hazleman BL. Prognosis and management of polymyalgia rheumatica. *Ann Rheum Dis* 1981;**40**:1–5.
29. Bengtsson BÅ, Malmvall BE. The epidemiology of giant cell arteritis including temporal arteritis and polymyalgia rheumatica. *Arthritis Rheum* 1981;**24**:899–904.
30. Haga HJ, Eide GE, Brun J, Johansen A, Langmark F. Cancer in association with polymyalgia rheumatica and temporal arteritis. *J Rheumatol* 1993;**20**:1335–9.
31. Noltorp S, Svensson B. High incidence of polymyalgia rheumatica and giant cell arteritis in a Swedish community. *Clin Exp Rheumatol* 1991;**9**:351–5.
32. Mertens JCC, Willemsen G, van Saase JLCM, Bolk JH, Dijkmans BAC. Polymyalgia rheumatica and temporal arteritis: a retrospective study of 111 patients. *Clin Rheumatol* 1995;**14**:650–5.
33. Dixon AStJ, Beardwell C, Kay A, Wanka J, Wong YT. Polymyalgia rheumatica and temporal arteritis. *Ann Rheum Dis* 1966;**25**:203–8.
34. Jundt JW, Mock D. Temporal arteritis with normal erythrocyte sedimentation rates presenting as occipital neuralgia. *Arthritis Rheum* 1991;**34**:217–9.
35. Desmet GD, Knockaert DC, Bobbaers HJ. Temporal arteritis: the silent presentation and delay in diagnosis. *J Int Med* 1990;**227**:237–40.
36. Fainaru M, Friedman G, Friedman B. Temporal arteritis in Israel. A review of 47 patients. *J Rheumatol* 1979; **6**:330–5.
37. Ayoub WT, Franklin CM, Torretti D. Polymyalgia rheumatica. *Am J Med* 1985;**79**:309–15.
38. Salvarani C, Rossi F, Macchioni P *et al.* Synovitis in polymyalgia rheumatica: an immunogenetic study. *Br J Rheumatol* 1992;**31**:720.
39. Healey LA. Long-term follow-up of polymyalgia rheumatica: evidence for synovitis. *Semin Arthritis Rheum* 1984;**13**:322–8.
40. Salvarani C, Gabriel SE, O'Fallon M, Hunder GG. Epidemiology of polymyalgia rheumatica in Olmsted county, Minnesota, 1970–1991. *Arthritis Rheum* 1995; **38**:369–73.
41. Armstrong RD, Behn A, Myles A, Panayi PS, Welsh KI. Histocompatibility antigens in polymyalgia rheumatica and giant cell arteritis. *J Rheumatol* 1983;**10**:659–61.
42. Richardson JE, Gladman DD, Fam A, Keystone EC. HLA DR4 in giant cell arteritis: association with the polymyalgia rheumatica syndrome. *Arthritis Rheum* 1987;**30**:1293–7.
43. Machado EBV, Michet CJ, Ballard DJ *et al.* Trends in incidence and clinical presentation of temporal arteritis in Olmsted County, Minnesota, 1950–1985. *Arthritis Rheum* 1988;**31**:745–9.
44. Smith CA, Fidler WJ, Pinals RS. The epidemiology of giant cell arteritis. *Arthritis Rheum* 1983;**26**:1214–9.
45. Graham E, Holland A, Avery A, Russell RWR. Prognosis in giant cell arteritis. *Br Med J* 1981;**282**: 269–71.
46. Huston KA, Hunder GG, Lie JT, Kennedy RH, Elveback LR. Temporal arteritis. *Ann Intern Med* 1978;**88**:162–7.
47. Wadman B, Werner I. Observations on temporal arteritis. *Acta Med Scand* 1972;**192**:377–83.
48. Laidlaw DAH, Bloom PA, Williams C, Easty DL. Steroid treatment in giant cell arteritis. *Br J Rheumatol* 1992;**31**:431–2.
49. Myles AB. Steroid treatment in giant cell arteritis. *Br J Rheumatol* 1992;**31**:787.
50. Aiello PD, Trautmann JC, McPhee TJ, Kunselman AR, Hunder GG. Visual prognosis in giant cell arteritis. *Ophthalmology* 1993;**100**:550–5.
51. Kohli M, Bennett RM. An association of polymyalgia rheumatica with myelodysplastic syndromes. *J Rheumatol* 1994;**21**:1357–9.
52. Kyle V, Wraight EP, Hazleman BL. Liver scan abnormalities in polymyalgia rheumatica/giant cell arteritis. *Clin Rheumatol* 1991;**10**:294–7.
53. Kyle V, Cawston TE, Hazleman BL. Erythrocyte sedimentation rate and C reactive protein in the assessment of polymyalgia rheumatica/giant cell arteritis on presentation and during follow up. *Ann Rheum Dis* 1989;**48**:667–71.
54. Klemsdal TO. Arteritis temporalis uten forhøyet senkningsreaksjon. *Tidsskr Nor Lægeforen* 1991;**111**: 1848–9.
55. Grodum E, Petersen HA. Temporal arteritis with normal

erythrocyte sedimentation rate. *J Int Med* 1990;**227**: 279–80.
56. O'Duffy JD, Hunder GG, Wahner HW. A follow-up study of polymyalgia rheumatica: evidence of chronic axial synovitis. *J Rheumatol* 1980;**7**:685–93.
57. Wiseman P, Stewart K, Rai GS. Hypothyroidism in polymyalgia rheumatica and giant cell arteritis. *Br Med J* 1989;**298**:647–8.
58. Juchet H, Labarthe MP, Ollier S, Vilain C, Arlet P. Prevalence of hypothyroidism in patients with giant cell arteritis or polymyalgia rheumatica: A controlled study in one hundred and four cases. *Rheumatisme* (Engl edn) 1993;**60**:406–11.
59. Barrier J, Magali A, Brisseau JM. Autoimmune thyroid disease, thyroid antibodies and giant cell arteritis: the supposed correlation appears fortuitous. *J Rheumatol* 1992;**19**:1733–4.
60. Malmvall BE, Bengtsson BÅ. Giant cell arteritis. *Scand J Rheumatol* 1978;**7**:154–8.
61. Dare B, Byrne E. Giant cell arteritis. *Med J Aust* 1980;**1**:372–3.
62. Gonzales-Gay MA, Alonso MD, Aguero JJ, Bal M, Fernandez-Camplor B, Sanchez-Andrade A. Temporal arteritis in a north-western area of Spain: Study of 57 biopsy proven patients. *J Rheumatol* 1992;**19**: 277–80.
63. Franzen P, Sutinen S, von Knorring J. Giant cell arteritis and polymyalgia rheumatica in a region of Finland: an epidemiologic, clinical and pathologic study, 1984–1988. *J Rheumatol* 1992;**19**:273–80.