No Increased Frequency of Malignant Neoplasms in Polymyalgia Rheumatica and Temporal Arteritis. A Prospective Longitudinal Study of 398 Cases and Matched Population Controls

GEIRMUND MYKLEBUST, TOM WILSGAARD, BJARNE KOSTER JACOBSEN, and JAN TORE GRAN

ABSTRACT. ***Objective.*** To determine the prevalence and incidence of cancer in patients with polymyalgia rheumatica (PMR) and temporal arteritis (TA) compared to matched population controls.

Methods. In a population based study 1987–97, 398 patients were diagnosed with PMR or TA. Each patient was randomly assigned 4 age and sex matched controls from the same county, totaling 1592 controls. All patients and controls were cross-checked with data files at the Cancer Registry of Norway, for cancers registered up to the end of 1998.

Results. Prior to inclusion, cancer was diagnosed in 32 patients with PMR or TA (8.0%) and 153 controls (9.6%) (OR 0.82, 95% CI 0.55–1.22, p = 0.3). After inclusion, malignant neoplasms were discovered in 34 patients with PMR or TA (9.3%) compared to 143 controls (10.8%) (relative risk 0.86, 95% CI 0.59–1.26, p = 0.4). Thus there was no difference between patients with PMR or TA and their controls regarding prevalence or incidence of cancer. The interval between inclusion and the time of diagnosis of malignant neoplasm did not differ between patients and controls. No significant difference in types or localization of malignant neoplasms was found in patients compared to controls.

Conclusion. No differences were found in frequencies or types of malignant neoplasms between patients with PMR or TA and population controls. Neither PMR nor TA as defined by present diagnostic criteria appears associated with cancer. (J Rheumatol 2002;29:2143–7)

Key Indexing Terms:
POLYMYALGIA RHEUMATICA
TEMPORAL ARTERITIS
CANCER
MALIGNANT NEOPLASM

Polymyalgia rheumatica (PMR) and temporal arteritis (TA) are common disorders in the elderly[1]. In both conditions, the etiology is unknown, but inflammation of medium and large size arteries is characteristic of TA[2], while in PMR muscular and periarticular manifestations predominate. Malignant disorders may be associated with musculoskeletal symptoms or vasculitic syndromes[3]. In some case reports, malignant disease presented as polymyalgia mimicking PMR[4-7] or malignancy was discovered later in the course of PMR[8]. However, no definite association between PMR and malignancy has been established. In biopsy proven TA, an increased risk of malignancy was found in one prospective controlled study[9], but other reports did not show enhanced susceptibility to malignancy in TA[10,11]. Moreover, we recently found no difference in total mortality in patients with TA compared to population controls[12], suggesting no relationship between TA and fatal diseases such as cancer.

In this study we compared the prevalence and incidence of cancer in patients with PMR or TA with that of a matched control population in a prospective longitudinal study.

MATERIALS AND METHODS

In the county of Aust Agder, Norway, 338 patients with PMR or TA were enrolled in the period 1987–97 in a prospective population based study, as described[1,13,14]. In addition, 60 cases were diagnosed at other departments in the same period, but were initially admitted to hospital for reasons not directly related to diagnosis of PMR or TA[12]. Causes of referral included development of osteoporotic fractures due to treatment with corticosteroids and diagnostic evaluation of constitutional manifestations. All patients in the PMR group satisfied the criteria suggested by Bird, *et al*[15] or those of Hamrin[16]. Also, most patients with PMR fulfilled the criteria proposed by Chuang, *et al*[17]. For TA, the American College of Rheumatology criteria[18] were employed, but only those with histologically proven temporal arteritis were included. Patients with malignancy or cancer discovered around the time of the diagnosis of PMR or TA were included if the diagnostic criteria were satisfied. Cases with PMR and a positive temporal biopsy either at the time of diagnosis or later in the course of the disease were denoted PMR/TA. As there were no differences regarding age, sex, and occurrence

From the Department of Rheumatology, Institute of Clinical Medicine, and the Institute of Community Medicine, University of Tromsö, Tromsö; and the Department of Rheumatology, National Hospital, Rikshospitalet, Oslo, Norway.

Supported in part by grants from the Norwegian Women's Public Health Association.

G. Myklebust, MD, Research Fellow, Institute of Clinical Medicine; T. Wilsgaard, MSc, Institute of Community Medicine; B.K. Jacobsen, PhD, Institute of Community Medicine; J.T. Gran, MD, PhD, National Hospital.

Address reprint requests to Dr. G. Myklebust, Department of Rheumatology, Box 14 UNN, 9038 Tromsö, Norway.
E-mail: geirmund.myklebust@unn.no

Submitted January 28, 2002; revision accepted April 17, 2002.

of malignancy between the 338 incident cases of PMR or TA and the 60 patients found by reviewing hospital records, all patients were regarded as one group, a total of 398 patients (Table 1).

From the Central Population Registry in Norway, all 398 patients were each randomly assigned 4 sex matched controls from the same county. The controls were born in the same year and month as the index case and were alive at the time of diagnosis of the index case.

The patients and their controls were cross-checked with the data files at the Cancer Registry of Norway. This registry includes all cases of cancer registered in Norway from 1953. When the study was undertaken, the registration of cancers was complete to the end of 1998. Time of diagnosis and types and sites of cancers were registered for each case identified. Further, the time intervals from diagnosis of malignant neoplasm to inclusion in the study (i.e., the date of diagnosis of PMR or TA), and from inclusion to diagnosis of cancer, were calculated. For subjects (12 patients and 40 controls) with 2 or more cancers, only the characteristics of the first detected neoplasm were used in the analysis. The followup thus started when diagnosis of PMR and/or TA was made and ended when the first cancer was diagnosed, or the patient died, or at end of followup at December 31, 1998. In the calculation of incidence, patients with cancer diagnosed prior to inclusion and their controls as well as all population controls with cancer detected before inclusion were excluded.

Causes of deaths were registered for all cases and their controls in the same period through linkage with Statistics Norway. Approval of the study was obtained from the data protection agency of Norway (Data Inspectorate) and the Board of Health of Norway (Statens Helsetilsyn).

Statistics. Chi-square test and Student t test were used to compare characteristics between patients and controls. When the odds ratios were estimated, a conditional logistic regression model was applied. Stratified Cox proportional hazard regression analyses were used to compare the incidence of cancer between patients and their age and sex matched controls, thereby taking the matching into consideration when computing relative risk (RR) estimates and 95% confidence intervals. The standard multivariate Cox proportional hazard model was used when we compared survival after a cancer diagnosis in patients with PMR or TA with survival after cancer diagnosis in population controls. P values less than 0.05 indicated significant results. All statistical analyses were carried out using the SAS system[19].

RESULTS

Patients. Altogether, 398 patients with PMR and/or TA (273 women, 125 men) were enrolled in the study. Sex and diagnostic subgroups are shown in Table 1. Three women diagnosed at other departments could not be definitely classified, as they all met diagnostic criteria for PMR, but TA was not appropriately evaluated. In one case, biopsy of the temporal artery was performed, but the histological description could not be found. At the end of the study period in 1998, 301 patients (75.6%) were still alive.

Controls. The matched controls numbered 1592 subjects (1092 women, 500 men). At the end of the study period, 1106 of the controls (69.5%) were alive.

Prevalence of cancer. Malignancy was registered in 32 patients (8.0%) and 153 controls (9.6%) before inclusion in the study (Table 2) (OR 0.82, 95% CI 0.55–1.22, p = 0.3). In cases with cancer prior to PMR or TA, the time until inclusion in the study was 8.4 years on average (range 17 weeks to 33.5 years) in patients and 9.4 years (2 weeks to 39.5 years) in controls. The difference was not statistically significant (p = 0.5).

Incidence of cancer. A total of 366 patients and 1324 controls were enrolled in the incidence study of malignancy discovered after diagnosis of PMR or TA. The patients were followed for a total of 2596 person-years and their controls for 9391 person-years. As shown in Table 2, malignant neoplasms were diagnosed in 34 patients (9.3%) compared to 143 controls (10.8%) after inclusion. Among women, there were 19 patients (7.5%) and 83 controls (9.1%) with cancer. In men, cancers were discovered in 15 patients (13.4%) and 60 controls (14.5%). The relative risk of cancer in patients was 0.86 (95% CI 0.59–1.26, p = 0.4) compared to controls. Patients with biopsy proven TA with or without PMR had a relative risk of cancer of 1.48 (95% CI 0.65–3.40, p = 0.4) compared to controls. In patients with PMR only, the relative risk of cancer was 0.75 (95% CI 0.47–1.16, p = 0.2). Patients with positive biopsy had a relative risk of cancer of 1.40 (95% CI 0.63–3.12, p = 0.4) compared to patients with PMR only.

Age at inclusion and the time interval from inclusion to diagnosis of malignant neoplasm. In patients whose malignant neoplasms were discovered after inclusion, the mean age at the time of diagnosis of PMR or TA was 73.1 (SD 7.3) years (60–84 yrs) compared to 71.7 (SD 7.2) years (51–91 yrs) in patients without cancer. The difference was not statistically significant (p = 0.3).

The interval from diagnosis of PMR or TA to diagnosis of malignant neoplasm was on average 3.8 (SD 2.7) years (7 weeks–10.5 yrs) compared to 4.3 (SD 3.0) years (2 weeks–12.2 yrs) in controls who contracted cancer. The

Table 1. Number, sex, and diagnosis of patients.

Diagnosis	Woman	Men	Total
PMR	213	102	315
TA	36	18	54
PMR/TA	21	5	26
Not classified	3	0	3
Total	273	125	398

Table 2. Number (%) of cases with malignant neoplasms in patients before and after diagnosis of PMR or TA and in controls.

Diagnosis	Before Inclusion		After Inclusion	
	Patients	Controls	Patients*	Controls*
PMR	25 (7.9)	127 (10.1)	25 (8.6)	117 (11.1)
TA	5 (9.3)	17 (7.9)	5 (10.2)	11 (6.1)
PMR/TA	2 (7.7)	9 (8.7)	3 (12.5)	12 (13.6)
Not classified	0 (0)	0 (0)	1 (33.3)	3 (25.0)
Total	32 (8.0)	153 (9.6)	34 (9.3)	143 (10.8)

*Thirty-two patients with cancer diagnosed before inclusion and their controls (n = 128, including 13 controls with cancer prior to inclusion), as well as remaining controls with cancer detected before inclusion (n = 140) were excluded from calculation of incidence.

differences in intervals between patients and controls were not statistically significant (p = 0.4). The proportion of patients with PMR or TA and population controls who are free of cancer during followup is illustrated in Figure 1.

Types and localization of malignant neoplasms. No significant differences in types and localization of malignant neoplasms were found between patients and controls. Cancers in colon and rectum were discovered in 6 (1.5%) patients (3 cases diagnosed after inclusion) compared to 44 (2.8%) cases among the controls (22 cases diagnosed after inclusion) (p = 0.2). The localization of the malignant neoplasms was similar in subjects diagnosed with cancer before and after diagnosis of PMR or TA (Table 3). A total of 2 cases of hematological malignancy were reported in the patients and 11 in controls. Apart from patients with cancer in the prostate, no clustering of cases near inclusion was found. However, 5 of 16 patients (31%) with prostate cancer were discovered in the period from 6 months before to one year after inclusion, compared to 3 out of 39 cases (8%) in the controls (p = 0.04).

Survival in cases with malignant neoplasms diagnosed after inclusion. Among incident cancer cases (34 patients, 143 controls), 7 patients (20.6%) and 51 controls (35.7%) died of cancer during the followup through 1998. After adjusting for age at inclusion and sex, Cox proportional hazards regression analysis may suggest a better survival of cancer in our patients than in population controls (RR 0.46, 95% CI 0.21–1.02, p = 0.05).

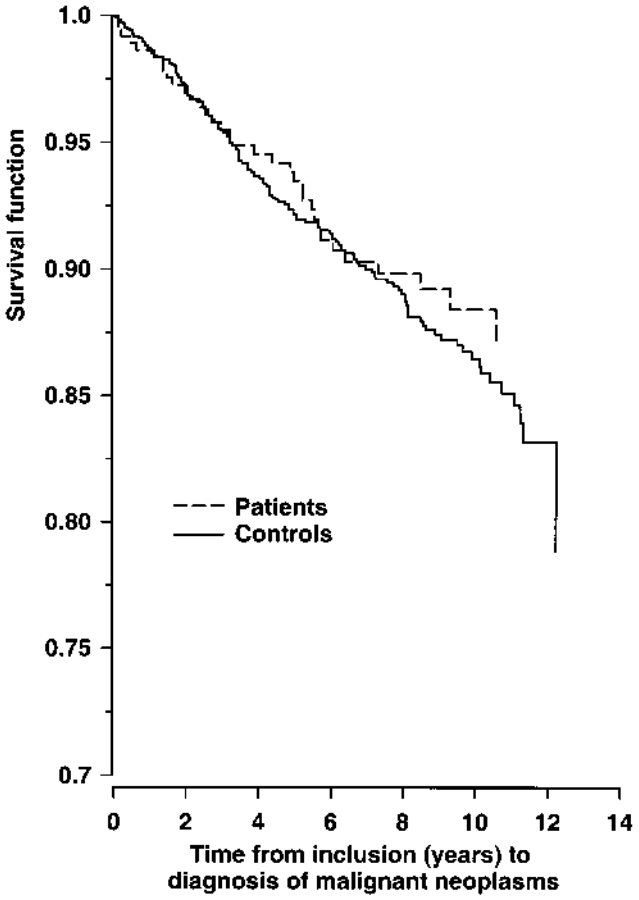

Figure 1. Survival functions for subjects free of cancer comparing patients with PMR or TA and controls.

DISCUSSION

The 2 comprehensive public registries in Norway (the Central Population Registry and the Cancer Registry) provide excellent opportunities to identify cases with malignancy in a cohort of a defined geographic region. Further, the design of this study allowed us to follow a population of patients from a county with a high incidence of PMR or TA[1], and in the observation period no patient or control was lost. Thus, the design of the study and the characteristics of the population evaluated offered unique opportunities to determine the true incidence of malignancy in PMR and TA. On the other hand, patients enrolled and regularly controlled in a hospital based study seem more likely to undergo extensive tests and examinations. Consequently, serious diseases such as cancer are probably discovered more rapidly in such patients. In a prospective study like the present one, it was up to the population controls themselves to establish contact with a general practitioner if symptoms or signs of illness appeared. With regard to referral for more comprehensive examinations and diagnosis of diseases such as cancer, a delay in the final diagnosis is expected and cases with malignant neoplasms might remain undiagnosed. Thus, in this study there existed a risk of overestimating the incidence of cancer in the patient population compared to controls. However, the incidence of cancer in the PMR/TA patients was, if anything, lower than in controls.

We found no increased frequency of cancer in patients with PMR or TA compared to population controls. This is in agreement with the low number of cancers discovered in earlier followup studies[4,7,9,10,17,20-26]. However, in a prospective controlled study by Haga and coworkers[9], a 2.35-fold higher risk of cancer was found in patients with a positive temporal artery biopsy compared to controls. In these patients, the authors also found a hazard rate of cancer 4.40 times higher than the rest of the patients studied. This is in contrast to our findings, where the relative risk of cancer in biospy proven TA or PMR/TA was 1.48 (95% CI 0.65–3.40) compared to their controls. Moreover, in patients with positive biopsy we found a relative risk of cancer of 1.40 (95% CI 0.63–3.12) compared to patients with negative biopsy or where biopsy was not taken (i.e., PMR). The difference in the risk of cancer within patients might be explained by a combination of a higher frequency of cancer in patients with biopsy proven TA and a smaller number of cancers in patients with PMR in the study of Haga and coworkers[9]. In that study, the patient population was based on medical records from 2 hospitals, in contrast to our study based on

Table 3. Sites of malignant neoplasms. Number (%) and sex of patients and controls before and after inclusion (diagnosis of PMR or TA).

Sites	No of Cases in Patients and Controls	Before Inclusion				After Inclusion			
		Women		Men		Women		Men	
		Patient	Control	Patient	Control	Patient	Control	Patient	Control
Gastrointestinal region	72 (19.9)	2 (10.5)	20 (17.9)	3 (23.1)	10 (24.4)	3 (15.8)	20 (24.1)	0 (0.0)	14 (23.3)
Lung and airways	26 (7.2)	0 (0.0)	1 (0.9)	0	5 (12.2)	2 (10.5)	8 (9.6)	1 (6.7)	9 (15.0)
Breast and female genital	97 (26.8)	13 (68.4)	56 (50.0)	0	0	3 (15.8)	25 (30.1)	0	0
Male genital	50 (13.8)	—	—	6 (46.2)	15 (36.6)	—	—	9 (60.0)	20 (33.3)
Skin (including non-melanoma)	56 (15.5)	3 (15.8)	17 (15.2)	2 (15.4)	4 (9.8)	5 (26.3)	14 (16.9)	1 (6.7)	10 (16.7)
Other	61 (16.9)	1 (5.3)	18 (16.1)	2 (15.4)	7 (17.1)	6 (21.1)	16 (19.3)	4 (26.7)	7 (11.7)
No. of cases with cancer	362 (100.0)	19 (100.0)	112 (100.0)	13 (100.0)	41 (100.0)	19 (100.0)	83 (100.0)	15 (100.0)	60 (100.0)

cases selected from general practitioners. In general, subjects with symptoms of serious diseases have a greater chance of being referred to hospital and undergo comprehensive examinations such as biopsy of the temporal artery than cases with milder disease. Thus, important differences in study design might explain the contradictory results in the 2 surveys. In Southern Europe, where the incidence of PMR and TA is much lower, cases of cancer presenting with PMR-like manifestations have been described[7]. However, in that part of Europe the mortality of isolated PMR was similar to the control population[27]. This was also the case when mortality in patients with biopsy proven giant cell arteritis, with or without associated PMR, was assessed[28].

In TA, associations with leukemia[29-31], lymphoma[32,33], Waldenstroms's macroglobulinemia[34,35], and lung cancer[36] have been suggested. Furthermore, symptoms resembling PMR have been described in metastatic cancers[6], myeloma[7], lymphoma that later transformed into subsequent acute lymphoblastic leukemia[37], and in renal cell carcinoma[5] and thyroid carcinoma[38] before surgical removal of the tumor. In PMR, followup studies[4,7,9,20,23-26] have described a wide range of malignant neoplasms mainly developing long before or after the diagnosis of PMR[4,9]. Thus, it is unlikely to consider PMR as a paraneoplastic syndrome, but evidently many types of cancer may present with polymyalgic symptoms.

We found no differences in types and sites of malignant neoplasm between patients and controls. However, among men there was a clustering of patients with cancer in the prostate close to the time of inclusion. Among these 5 patients, only one had symptoms from the urinary system at the time of inclusion resulting in further examination. In the other cases, referral to a department of urology was done by the general practitioners. Thus, the simultaneous manifestation of cancer in the prostate seemed to be coincidental, and is conceivably not explained by improved medical surveillance.

Finally, no tendency to increased frequency of cancer throughout the observation period appeared in patients compared to controls (Figure 1). The interval from diagnosis of PMR or TA to diagnosis of cancer was on average 3.8 years, and this may speak against previous suggestions of these diseases representing paraneoplastic disorders[6]. As PMR and TA are relatively low prevalence diseases, and due to the paucity of cancer in our patients, we have limited statistical power to detect statistically significant differences in cancer prevalence and incidence between our patients and the controls. It may, however, be argued that the 95% confidence intervals do indicate that a clinically significant increased cancer risk is rather unlikely.

No differences in frequencies and types of malignant neoplasms between patients with PMR or TA and population controls were found in this study. Thus, neither PMR nor TA as defined by present diagnostic criteria appears to be associated with cancer in general. However, cancer by itself may present with polymyalgic manifestations and the older ages of patients with PMR and TA should always alert the clinician to the possible coexistence of malignancy. It is the experience of many rheumatologists that patients with biopsy negative TA and cases with inadequate responses to corticosteroid are those at highest risk of having a concomitant malignant disease.

ACKNOWLEDGMENT

The authors thank Aage Johansen, Cancer Registry of Norway, and Magne Magnussen, IBM, for their contributions in the data acquisition phase of this project.

REFERENCES

1. Gran JT, Myklebust G. The incidence of polymyalgia rheumatica and temporal arteritis in the county of Aust Agder, South Norway: A prospective study 1987-94. J Rheumatol 1997;24:1739-43.
2. Hunder GG. Giant cell arteritis and polymyalgia rheumatica. Med Clin North Am 1997;81:195-219.
3. Naschitz JE, Rosner I, Rozenbaum M, Elias N, Yeshurun D. Cancer-associated rheumatic disorders: Clues to occult neoplasia. Semin Arthritis Rheum 1995;24:231-41.
4. Mertens JC, Willemsen G, Van Saase JL, Bolk JH, Dijkmans BA. Polymyalgia rheumatica and temporal arteritis. A retrospective study of 111 patients. Clin Rheumatol 1995;14:650-5.
5. Sidhom OA, Basalaev M, Sigal LH. Renal cell carcinoma presenting as polymyalgia rheumatica. Resolution after nephrectomy. Arch Intern Med 1993;153:2043-5.
6. Naschitz JE, Slobodin G, Yeshurun D, Rozenbaum M, Rosner I. A polymyalgia rheumatica-like syndrome as presentation of metastatic cancer. J Clin Rheumatol 1996;2:305-8.

7. Gonzalez-Gay MA, Garcia-Porrua C, Salvarani C, Olivieri I, Hunder GG. The spectrum of conditions mimicking polymyalgia rheumatica in Northwestern Spain. J Rheumatol 2000;27:2179-84.
8. Bahlas S, Ramos-Remus C, Davis P. Clinical outcome of 149 patients with polymyalgia rheumatica and giant cell arteritis. J Rheumatol 1998;25:99-104.
9. 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.
10. Huston KA, Hunder CG, Lie JT, Kennedy RH, Elveback LR. Temporal arteritis: A 25-year epidemiological, clinical and pathological study. Ann Intern Med 1978;88:162-7.
11. Boesen P, Freiesleben F, Sorensen S. Giant cell arteritis, temporal arteritis, and polymyalgia rheumatica in a Danish county. Arthritis Rheum 1987;30:294-9.
12. Gran JT, Myklebust G, Wilsgaard T, Jacobsen BK. Survival in polymyalgia rheumatica and temporal arteritis: a study of 398 cases and matched population controls. Rheumatology 2001;40:1238-42.
13. Myklebust G, Gran JT. 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. Br J Rheumatol 1996;35:1161-8.
14. Gran JT, Myklebust G. The incidence and clinical characteristics of peripheral arthritis in polymyalgia rheumatica and temporal arteritis: a prospective study of 231 cases. Rheumatology 2000;39:283-7.
15. Bird HA, Esselinckx W, Dixon ASTJ, Mowat AG, Wood PHN. An evaluation of criteria for polymyalgia rheumatica. Ann Rheum Dis 1979;38:434-9.
16. Hamrin B. Polymyalgia rheumatica. Acta Med Scand 1972;533 Suppl:1-13.
17. Chuang T-Y, Hunder GG, Ilstrup DM, Kurland LT. Polymyalgia rheumatica. A 10-year epidemiologic and clinical study. Ann Intern Med 1982;97:672-80.
18. 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.
19. SAS/STAT user's guide. Version 6, 4th ed. Gary: SAS Institute; 1990.
20. von Knorring J, Somer T. Malignancy in association with polymyalgia rheumatica and temporal arteritis. Scand J Rheumatol 1974;3:129-35.
21. Mackenzie AH. The polymyalgia rheumatica syndrome. Geriatrics 1969;24:158-66.
22. Hunder G, Disney T, Emmerson L. Polymyalgia rheumatica. Mayo Clin Proc 1969;44:849-75.
23. Andersson R, Malmvall B-E, Bengtsson B-Å. Long-term survival in giant cell arteritis including temporal arteritis and polymyalgia rheumatica. Acta Med Scand 1986;220:361-4.
24. Salvarani C, Macchioni PL, Tartoni PL, et al. Polymyalgia rheumatica and giant cell arteritis: a 5-year epidemiological and clinical study in Reggio, Italy. Clin Exp Rheumatol 1987;5:205-15.
25. Schaufelberger C, Bengtsson B-Å, Andersson R. Epidemiology and mortality in 220 patients with polymyalgia rheumatica. Br J Rheumatol 1995;34:261-4.
26. Matteson EL, Gold KN, Bloch DA, Hunder GG. Long-term survival of patients with giant cell arteritis in the American College of Rheumatology giant cell arteritis classification criteria cohort. Am J Med 1996;100:193-6.
27. Gonzalez-Gay MA, Garcia-Porrura C, Vázquez-Caruncho M, Dababneh A, Hajeer A, Ollier WER. The spectrum of polymyalgia rheumatica in Northwestern Spain: incidence and analysis of variables associated with relapse in a 10 year study. J Rheumatol 1999;26:1326-32.
28. Gonzalez-Gay MA, Blanco R, Abraira V, et al. Giant cell arteritis in Lugo, Spain, is associated with low longterm mortality. J Rheumatol 1997;24:2171-6.
29. Shimamoto Y, Matsunaga C, Suga K, Fukushima N, Nomura K, Yamagushi M. A human T-cell lymphotropic virus type I carrier with temporal arteritis terminating in acute myelogenous leukemia. Scand J Rheumatol 1994;23:151-3.
30. Gabriel S, Conn D, Phyliky R, Pittelkow M, Scott R. Vasculitis in hairy cell leukemia: review of literature and consideration of possible pathogenic mechanisms. J Rheumatol 1986;13:1167-72.
31. Greer JM, Longley S, Edwards NL, Elfenbein GJ, Panush RS. Vasculitis associated with malignancy: experience with 13 patients and literature review. Medicine 1988;67:222-30.
32. Case records of the Massachusetts General Hospital. N Engl J Med 1990;322:1656-65.
33. Caldwell DS, McCallum RM. Rheumatological manifestations of cancer. Med Clin North Am 1986;70:385-417.
34. Kalra L, Delamere JP. Lymphoreticular malignancy and monoclonal gammopathy presenting as polymyalgia rheumatica. Br J Rheumatol 1987;26:458-9.
35. Kyle RA. Monoclonal gammopathy of undetermined significance (MGUS): a review. Clin Haematol 1982;2:123-50.
36. Lie JT. Simultaneous clinical manifestations of malignancy and giant cell temporal arteritis in a young woman. J Rheumatol 1995;22:367-9.
37. Montanaro M, Bizzari F. Non-Hodgkin's lymphoma and subsequent acute lymphoblastic leukaemia in a patient with polymyalgia rheumatica. Br J Rheumatol 1992;31:277-8.
38. Tabata M, Kobayashi T. Polymyalgia rheumatica and thyroid papillary carcinoma. Intern Med 1994;33:41-4.