Polymyalgia Rheumatica Is Not Seasonal in Pattern and Is Unrelated to Parvovirus B19 Infection

PILAR PERIS

ABSTRACT. Objective. To prospectively analyze seasonal distribution in the onset of symptoms of polymyalgia rheumatica (PMR) and its relationship to parvovirus B19 infection.

Methods. Over a 4-year period (September 1997 to September 2001), 68 patients were prospectively diagnosed with PMR in an outpatient rheumatology department, of which only 55 patients (38 women, 17 men) aged 50 to 90 years (mean 74.1 ± 8.1) were able to specify the month of onset of symptoms. During the last year parvovirus B19 IgM serologies were determined in all new cases.

Results. No significant seasonal variation in disease onset was observed during the 4-year period; 17 cases were observed in spring, 10 in summer, 15 in autumn, and 13 in winter (p = 0.625). Nevertheless, almost 50% of all cases of PMR were diagnosed in the months of May, February, and August. All of the evaluated patients (14 of 14) had negative parvovirus B19 IgM serologies.

Conclusion. Onset of symptoms in PMR is unrelated to seasonal pattern. Yet almost 50% of cases occurred in the months of May, February, and August. Parvovirus B19 infection was unrelated to the onset of PMR. (J Rheumatol 2003;30:2624–6)

Key Indexing Terms: POLYMYALGIA PARVOVIRUS SEASONAL

Polymyalgia rheumatica (PMR) is a common illness, with an average annual incidence of 17.8 cases/100,000 people over age 50 years. Although the etiology of this disorder is unknown, genetic and environmental factors may play a role. The abrupt onset of symptoms and the periodic clustering of cases observed in some series suggest a precipitating infectious factor. Indeed, infectious processes such as parainfluenza virus, Mycoplasma pneumoniae, Chlamydia pneumoniae, and parvovirus B19 have been related to this disease. Interestingly, reports indicate a seasonal or monthly clustering of cases of PMR, whereas others failed to confirm any seasonal trend. Nevertheless, it should be noted that most of these studies are retrospective, which makes evaluation of the precise onset of symptoms and the possible coincident viral infection difficult.

The aim of this study was to prospectively analyze the seasonal distribution of PMR in an outpatient rheumatology unit and its relationship to recent parvovirus B19 infection.

MATERIALS AND METHODS

Over a 4-year period (September 1997 to September 2001) all cases diagnosed with PMR were prospectively included in the study. Only the patients who were able to specify the month of onset of symptoms were analyzed.

PMR was diagnosed based on established criteria. Diagnosis was confirmed by a followup at least 10 months.

No differences in seasonal pattern were found: 17 cases started symptoms in spring, 10 in summer, 15 in autumn, and 13 in winter (p = 0.625). Almost 50% of patients started symptoms in the months of May (18%), February (15%), and August (13%). Figure 1 shows the monthly rate of the onset of symptoms during the 4-year period. During this period, a total of 19,710 additional visits were made, of which 7971 were first visits. The total monthly number of visits remained stable throughout the study (Figure 2).

All patients were evaluated by the same rheumatologist (PP) and were routine referrals from general practitioners. This department is the referral unit for an urban population of about 180,000 people in Barcelona. The data of the first visit and the monthly onset of symptoms were noted for all patients. In addition, the total number of visits to this department during the same period of time was recorded.

Serological analysis of parvovirus B19 was performed during the last year of the study in all new incident cases. Detection of IgM antibodies to parvovirus B19 was performed by ELISA at the time of clinical diagnosis; IgG antibodies were not performed.

To evaluate a seasonal effect, the month of onset of the first symptoms was used to calculate the season-specific incidence rates. Differences between season incidence rates were assessed by the chi-square test. Statistical significance was defined as p < 0.05.

RESULTS

A total of 68 patients were diagnosed with PMR, of which 55 were able to specify the month of onset. There were 38 postmenopausal women and 17 men, aged 50 to 90 years (mean ± SD, 74.1 ± 8.1 yrs). The mean duration of symptoms prior to visit was 63.8 ± 43 days.

No differences in seasonal pattern were found: 17 cases started symptoms in spring, 10 in summer, 15 in autumn, and 13 in winter (p = 0.625). Almost 50% of patients started symptoms in the months of May (18%), February (15%), and August (13%). Figure 1 shows the monthly rate of the onset of symptoms during the 4-year period. During this period, a total of 19,710 additional visits were made, of which 7971 were first visits. The total monthly number of visits remained stable throughout the study (Figure 2). When the monthly incidence of cases of PMR was evaluated each year, the periodic clustering of cases differed slightly between years. Most of the cases that presented in May and February were observed in 1999 and 2000, whereas most of
the cases presenting in August were observed in 1998 and 2001 (Figure 3).

The serological study for IgM parvovirus B19 was negative in all cases studied (14/14): 7 cases occurred in winter, 3 in spring, 3 in autumn, and one in summer.

**DISCUSSION**

This study shows that PMR is not seasonal in pattern.

Nevertheless, a periodic clustering of cases in May, February, and August was observed. No relationship between parvovirus B19 infection and the onset of disease was found during the last year of the study.

Some studies indicate a seasonal pattern in the incidence of PMR³, whereas others do not⁶,⁷. The reasons for these discrepancies may be due to several factors. Most of these studies were retrospective, thus the specific recall about the
onset of symptoms is absent. This point is important, since the disease onset does not usually coincide with the month of visit. In this study, the mean duration of symptoms prior to visit was about 2 months, and almost 20% of patients were unable to specify the month of onset. Moreover, the variable periodic clustering of cases may interfere with the evaluation of a seasonal pattern. Thus, the incidence of PMR cases may peak at different months of the year and change between years. In this sense, a previous 12-year study indicated quarterly and annual variations of incidence of giant cell arteritis and PMR. In the present series, symptoms in almost half the patients occurred in May, February, and August, 3 different seasons, with variations between years. However, the low number of cases included in the study may have influenced these results.

The acute onset of symptoms of PMR suggests a precipitating environmental factor as one of the causes. Indeed, an increased prevalence of antibodies against parainfluenza virus type 1 has been reported in PMR, as well as a close temporal relationship between the epidemics of M. pneumoniae, C. pneumoniae, and parvovirus B19 infections and the incidence peaks of PMR and giant cell arteritis. Nevertheless, the relationship between PMR or giant cell arteritis and parvovirus infection seems to be controversial. Cimmino, et al. in a preliminary study indicated a relationship between PMR and B19 infection, whereas Hemauer, et al. did not confirm these results. The results of my study suggest that parvovirus B19 does not play a role in PMR. However, it should be recognized that parvovirus infection was analyzed during the last year of the study, and B19 infection is known to occur in periodic outbreaks, with peaks of disease activity occurring from 3 to 6 years apart. Therefore, it cannot be totally ruled out that this virus may have been related to other clustering peaks in previous years.

PMR does not appear to be associated with a seasonal pattern. Parvovirus B19 infection was not associated with the onset of symptoms of PMR in this study. Nevertheless, longer prospective studies with larger numbers of patients are necessary to confirm these results.

REFERENCES