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LETTER TO THE EDITOR

A Case of Pseudogout Successfully Treated with Prednisolone and Clarithromycin

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To the Editor pseudogout (PG) is an acute arthritis induced by calcium pyrophosphate dihydrate crystal deposition, and it frequently develops in the elderly population. Non-steroidal anti-inflammatory drugs and/or corticosteroids, such as prednisolone (PSL), administered either by local intra-articular injection or systemic therapy remain the mainstay of PG treatment.[1] Macrolide antibiotics (MACs), such as clarithromycin (CAM) and azithromycin (AZM) provide not only antibacterial activity but also anti-inflammatory properties. Several recent studies reported the successful treatment of rheumatoid arthritis[2] and polymyalgia rheumatica[3] using CAM as an anti-inflammatory agent. Herein, we describe a case of PG treated with PSL in combination with CAM, considering their anti-inflammatory properties. A 92-year-old female with Alzheimer's disease, hypertension and osteoporosis presenting with fever, arthralgia and articular swellings in her bilateral shoulders, elbows, and wrists, was referred to our hospital. At the age of 91, she was diagnosed with PG by an orthopedic doctor. So far, she has experienced two PG attacks. The laboratory findings on this visit were as follows: white blood cell count, 8,990/μL (basophils, 0.3%; eosinophils, 1.0%; neutrophils, 78.4%; lymphocytes, 12.7%; and monocytes, 7.6%); C-reactive protein (CRP), 11.81 mg/dL; rheumatoid factor, 5 IU/L; anti-cyclic citrullinated peptide antibody, 0.5 U/mL; and antinuclear antibody titer, 1:40. A blood culture yielded negative results. No abnormal findings suggestive of infection could be found in the systemic survey, including the chest roentgenogram and urinalysis. Bone roentgenogram revealed intra-articular calcification compatible with PG. The aforementioned symptoms were similar to those of the previous PG attacks; therefore, the patient was diagnosed with PG attack. Initially, she was treated with acetaminophen (800 mg/day), which was unsuccessful, although it was effective in the previous PG attacks. Therefore, she was treated with PSL (10 mg/day). Seven days after PSL treatment, fever and articular symptoms rapidly improved with a decreased CRP of 1.12 mg/dL. Therefore, the PSL dosage was decreased to 7.5 mg/day. A week later, fever and articular symptoms relapsed with an

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increased CRP of 16.01 mg/dL. The PSL dosage was again increased to 10 mg/day. Furthermore, she received CAM (400 mg/day), considering its anti-inflammatory properties. Ten days after CAM treatment, the symptoms improved with a decreased CRP of 0.2 mg/dL. Therefore, CAM was ceased and the PSL dosage was decreased to 9 mg/day. Thereafter, the PSL dosage could be gradually decreased to 5 mg/day without any recurrence of fever and articular symptoms. However, 2 weeks after receiving PSL (5 mg/day), articular symptoms relapsed with CRP increasing to 5.81 mg/dL. As an alternative to increasing the PSL dosage, she received CAM (400 mg/day) again. Two weeks after CAM retreatment, articular symptoms improved with CRP decreasing to 0.2 mg/dL. Thereafter, the PSL dosage could be gradually decreased to 2 mg/day. As stated above, MACs provide anti-inflammatory properties as well as antibacterial activity. MACs reportedly affect several pathways of the inflammatory process, including the production of proinflammatory cytokines. In fact, AZM inhibits the production of interleukin (IL)-1.[4] It has been reported that IL-1 is associated with PG attack[5]; therefore, the efficacy of CAM treatment in the present case might be because of its anti-inflammatory properties that inhibit the production of IL-1.

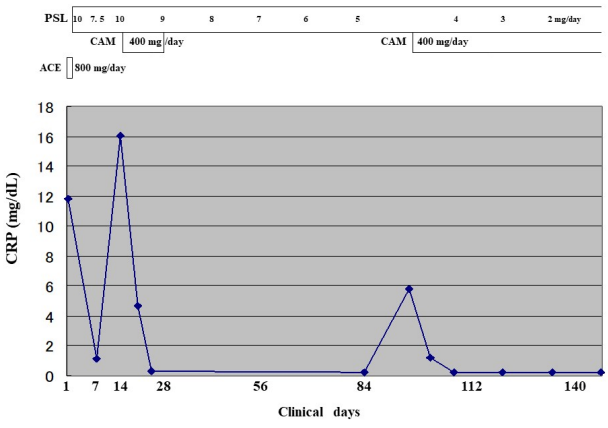

Figure 1. The laboratory data and prescribed agents on clinical days. ACE: acetaminophen, PSL: prednisolone, CAM: clarithromycin, CRP: C-reactive protein

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