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Nimesulide: Is it only an anti-inflammatory, analgesic drug?

Sir,

Nimesulide (4-nitro-2-phenoxy-methanesulphonanilide) is a non-steroidal anti-inflammatory drug (NSAID), which is considered to be a preferential cyclo-oxygenase-2-blocker and has other anti-inflammatory actions in addition to its action on prostanoid formation (1-7). NSAIDs are largely used for relief of symptoms of rheumatic diseases, but they are considered to be ineffective in modifying disease activity; they are also considered to be ineffective (or at least to exhibit only slight and inconstant effects) on acute-phase reactants, such as the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). A decrease in the values of acute-phase reactants is instead a typical effect induced by slow-acting antirheumatic drugs (SAARDs). We observed a case of polymyalgia rheumatica (PMR) which appeared especially interesting because nimesulide administration might have induced a marked decrease of acute-phase reactants.

We examined a 68-year-old Caucasian male who reported persistent pain localised in the muscles of the neck, shoulder and pelvic girdle. The onset of pain had been about 1 year before. Muscle or joint tenderness was not observed on clinical examination. ESR and CRP values were within the normal range (ESR: 10 mm/h; CRP serum level: 0.6 mg/dl). Rheumatoid factor was detected, with a titre of 441 IU. The patient had been taking low dose nimesulide (100 mg daily) for one month. Nimesulide induced only a slight and transient decrease in pain intensity. Nimesulide administration was then stopped. Seven days after nimesulide withdrawal ESR was 22 mm/h, the CRP level was 1.5 mg/dl, rheumatoid factor titre was 532 IU, and the fasting plasma glucose level was 157 mg/dl. The patient complained of strong pain interfering with daily activities and with nocturnal sleep. ESR and the CRP level measured again after 10 days were 80 mm/h and 7 mg/dl, respectively. The patient reported persistent strong pain, mainly felt during the night and the morning, localised in the shoulder and pelvic girdle, with morning stiffness lasting about 2 hours. Joint or muscle tenderness or reduced muscle strength were not detected on clinical examination. PMR was diagnosed and deflazacort was prescribed, with a daily dosage of 30 mg. Two months after the onset of deflazacort therapy, the patient reported complete pain relief. ESR and CRP values were in the normal range.

This case appears to be interesting for many reasons. The patient was affected by PMR: the diagnosis was based on typical clinical and laboratory findings, and was confirmed by the effect of the specific therapy for this disease, i.e. the administration of corticosteroids. When we first saw the patient, the diagnosis was not clear because the values of acute-phase reactants were within the normal range; the patient was taking nimesulide, which had induced only a slight analgesic effect. An increase in the values of acute-phase reactants occurred only after nimesulide withdrawal, which was also followed by an increase of pain. It may be deduced that nimesulide, though it was not effective in providing complete pain relief, could have a marked effect on acute-phase reactants. Complete relief of pain was achieved only by the administration of corticosteroids, which are considered to be the drugs of choice in PMR. We may suppose that the effect of nimesulide on acute-phase reactants is due to an as yet unknown mechanism of action, quite different from those studied previously and not related to the anti-inflammatory and analgesic actions.

Further investigations are necessary to confirm our hypothesis. A careful comparison between the effects of nimesulide and the effects of other NSAIDs in different clinical conditions may be useful to identify the pathophysiological mechanisms via which nimesulide exerts its specific actions.

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