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OSTEOARTHRITIS and CARTILAGE

Chondroitin sulfate: S/DMOAD (structure/disease modifying anti-osteoarthritis drug) in the treatment of finger joint OA

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Summary

A total of 119 patients were included in a randomized, double-blind, placebo-controlled trial in order to assess the S/DMOAD properties in OA of chondroitin sulfate (CS 4&6, 3×400 mg/day, Condrosulf® IBSA, Lugano, CH).

Posteranterior roentgenographies of the interphalangeal (IP) joints were carried out at the start of the study and at yearly intervals. This enabled the investigators to document the radiological progression of the anatomical lesions in the pathological finger joints over a 3-year period. It was shown that the progression of OA in the IP finger joints in an individual can be determined by the evolution of his finger joints through previously described anatomical phases: 'N' (not affected), 'S' (classical OA), 'J' (loss of joint space), 'E' (erosive OA) and 'R' (remodeled joint).

Structure/disease-modifying anti-OA drug (S/DMOAD) properties were searched for by assaying the number of patients developing OA in previously normal IP joints ('N' > 'S'), or progressing through the described anatomical phases of the disease ('S' > 'J', 'S' > 'E', 'J' > 'E', 'S' > 'R', 'J' > 'R', 'E' > 'R'). In the CS 4&6 group we observed a significant decrease in the number of patients with new 'erosive' OA finger joints. This result is particularly important since OA of the finger joints becomes a clinical problem (pain, functional loss) when 'S' joints progress to 'J' and especially 'E' phases. During and after these 'E' phases, joints will remodel and show the nodular deformities characteristic of Heberden's and Bouchard's nodes.

Treated patients were protected against erosive evolution.

Key words: Chondroitin sulfate, Finger joint OA, DMOAD, Roentgenography, Heberden's and Bouchard's OA.

THIS randomized, double-blind, placebo-controlled study, including 119 patients, was designed to assess the S/DMOAD properties in osteoarthritis (OA) of chondroitin sulfate (CS 4&6, Condrosulf® IBSA, Lugano, CH). Thirty-four of the patients received chondroitin sulfate 4&6 orally administered: 400 mg, three times daily. Placebo medication was administered to 85 patients in total: 46 of them belonged to a previous clinical trial and 39 to the present study. All 119 patients were followed during 3 years.

Posteranterior roentgenographies of the DIP and PIP joints and of the MCP joints of the 2nd, 3rd, 4th and 5th fingers were obtained at the start of this prospective study and at yearly intervals. This enabled the investigators to document the radiological progression of the anatomical lesions in the pathological finger joints over a 3-year period.

It was shown that the progression of OA in the finger joints in an individual can be assessed by the evolution of his/her finger joints through previously described anatomical phases: 'N' (not affected), 'S' (classical OA), 'J' (loss of joint space), 'E' (erosive OA) and 'R' (remodeled joint) [1].

A structure/disease-modifying anti-OA drug (S/DMOAD) should decrease: (1) the number of patients developing OA in previously normal finger joints; (2) the number of patients developing 'erosive' OA in osteo-arthritic finger joints. This last definition is particularly important since 60–80% of individuals over 70 years of age have so-called 'S' OA and have been asymptomatic throughout their lives. OA of the fingers joints becomes a clinical problem (pain, functional loss) when 'S' joints progress to 'J' and especially 'E' phases. During and after these 'E' phases, joints will remodel and show the nodular deformities characteristic of Heberden's and Bouchard's nodes.

Structure and disease modifying anti-OA drug (S/DMOAD) properties were searched for by assaying the numbers of patients developing OA in previously normal finger joints (N > S), or progressing through the described anatomical phases

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of the disease (S > J, S > E, J > E, S > R, J > R, E > R, S > N). The numbers of patients in the different treatment groups showing particular changes in their anatomical phases were compared using the chi-square test.

It is obvious that similar pathogenetic mechanisms lie behind the aggressive progression of osteoarthritis of the DIP and PIP joints. Therefore, DIP and PIP joints were considered as one single group of finger joints when the progression through the anatomical phases in the individual patients was studied. The evolution in the interphalageal (IP) joints during 3 years of follow-up is discussed.

MCP joints, which were separately evaluated, were not influenced by CS 4&6.

An analysis of the changes in the anatomical phases in each individual showed that comparable numbers of patients in both treated and placebo groups developed OA in non-affected joints. However, in the CS 4&6-treated group, we observed a significant decrease in the number of patients with new 'erosive' OA finger joints.

Some 8.8% of the patients had 'S' or 'J' joints becoming 'erosive' (2.9% of the patients had 'S' joints becoming 'erosive'; 5.9% of the patients had 'J' joints becoming 'erosive'). In the untreated group 29.4% of the patients had 'S' or 'J' joints becoming 'erosive' (20.0% of the patients had 'S' joints becoming 'erosive'; 22.4% of the patients had 'J' joints becoming 'erosive').

The pharmacologic mechanisms behind this effect can be a matter of discussion. Progression to the 'E' phase is characterized by erosive changes in the subchondral bone of these finger joints. Direct or indirect pharmacologic effects on the physiology of this tissue could have led to protection of the joint (the so-called S/DMOAD effect).

References

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