Background: SM04690, a small molecule intra-articular (IA) Wnt pathway inhibitor is in development as a potential disease modifying knee osteoarthritis drug. A \textit{SM04690, a small molecule intra-articular (IA) Wnt pathway inhibitor...}

Methods: Subjects with ACR-defined knee OA, Kellgren-Lawrence (KL) grades 2–3, received 2 mL IA SM04690 (0.03, 0.07, or 0.23 mg) or placebo (PBO) in the target (most painful) knee. WOMAC Pain [0–50] and Function [0–170] were assessed at Weeks 0, 4, 13, 26, 39 and 52 and knee radiographs at Weeks 0, 26 and 52. Baseline-adjusted logistic regression group analyses estimated concordance between mJSW change and pain and function changes for responders who achieved both WOMAC Pain and Function improvements of $\geq 50\%$ and $>20$ [scaled to $100$] units. Receiver-operator characteristic (ROC) curves were generated with area under curve (AUC) to estimate concordance (AUC $>0.7=\text{excellent}$, $0.7=\text{good}$, and $0.5=\text{poor}$) 

Conclusions: In this post-hoc analysis, treatment with SM04690 maintained or increased mJSW in the 0.03 and 0.07 mg doses compared to PBO over 52 weeks. In UNI and UNI-WP 0.07 mg cohorts, changes in mJSW were concordant with WOMAC Pain and Function response.
Methods: The study involved 60 female patients with knee OA having neuro-pathic pain component (NPC; DN4 >4). Mean age 59.82±4.46 y (min 49, max 65 years). All patients were randomly divided into two groups to be treated with 2 therapeutic regimens: acetaminophen +pregabalin (Group I) or acetaminophen (Group II) for 5 weeks (3 visits). All patients were subjected to clinical and neurological examination, total WOMAC score assessment, verification of neuropathic pain (NP) (questionnaire DN4 and Pain DETECT), and VAS pain intensity assessment at rest.

Results: The therapy was successful in both groups with respect to WOMAC score [figure 1] (Group I – 1385.30±365.83 vs 1034.70±402.37 vs 886.64±456.31; Group II – 1206.04±358.72 vs 1016.45±428.52 vs 976.55±408.02 respectively, p=0.01). Significant reduction of pain intensity at rest was documented in both groups [figure 2]. Group I 61.60±14.91 vs 45.34±16.14 vs 36.24±18.09; Group II 56.07±22.58 vs 44.86±18.68 vs 41.96±24.04, p=0.01, respectively. Therapeutic regimens in both groups had positive impact on NPC based on DN4 questionnaire and Pain DETECT scores. However, a combination of a NSAID with anticonvulsants agent (pregabalin) resulted in a more pronounced effect. Changes in DN4 values in Group I (visit1/visit3) were: 5.97±1.24/2.97±1.83 p=0.001; and in Pain DETECT values – 17.93±3.87/9.34±6.18, p=0.001; while in Group II DN4 scores were 5.35±0.93/3.79±2.29, p=0.001; and Pain DETECT – 15.03±5.26/12.24±6.29, p=0.02. [figures 3–4]

Conclusions: Complex therapy with the use of Pregabalin in patients with OA of the knee, with signs of NPC, allows not only effectively reduce pain intensity and improve functional activity of patients and, consequently, the quality of life.

Disclosure of Interest: None declared


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NEUROPHYSIOLOGICAL DATA IN PATIENTS WITH CHRONIC PAIN IN KNEE OSTEOARTHRITIS

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Background: Traditionally, chronic pain in OA is considered to be a classical model of nociceptive pain. Nociceptive mechanism can’t explain the presence: referred pain, secondary hyperalgesia and other sensitive phenomena.1 Recent studies has shown that besides nociceptive pain there is another mechanism that takes place in chronic pain OA – central sensitisation.2,3 Exploring of chronic pain OA can be reached only by a complex approach in examining patients with chronic knee OA that includes not only a rheumatological examination, but examination of the neurological sphere and algometria. At the moment there are only few studies dedicated to neurophysiological changes in pain OA.

Objectives: to assess pain system with neurophysiological examination in chronic pain OA

Methods: 46 chronic knee pain OA and 23 healthy group control women, 45–65 years old, were included. The study included clinical rheumatologic, neurological examinations, neuropathic pain scales (DN4 and Pain DETECT). Knee X-ray and ultrasound studies. Neurophysiological examination included algomertia with algometer and wind-up phenomena observed by Neuropen. Five test sites in the peripatellar region and one control site on tibialis anterior (5 cm distal to the tibial tuberosity) were located and marked for examination

Results: Neuropathic pain scales demonstrated neuropathic descriptors present in OA patients. Neurological examination revealed no somatosensory deficit. But examination of the sensitive sphere indicated hyperalgesia: primary hyperalgesia (increased sensitivity to pain in the damaged joint) and secondary hyperalgesia. Chronic OA is a complex of mechanisms and includes nociceptive pain, referred pain, secondary hyperalgesia and other sensitive phenomena.1 Recent studies has shown that besides nociceptive pain there is another mechanism that takes place in chronic pain OA – central sensitisation. Mechanism-oriented treatment should also target CNS, including anticonvulsant, and antidepressant agents.

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[2] Kosek E, Ordeberg G. Abnormalities of somatosensoryperception in patients revealed low pressure pain threshold(PPT) above injured knee and intact region compared with healthy group. (tab.1) PPT in intact region was compared between OA patients and control group by ROC-analysis. Max of PPT in intact region in patient with OA was – 14.70, min – 1.80, mean value – 7.34. Mean value of PPT in control group was 15.18. Area under curve was: 0888. Sensitivity – 70%. Specificity – 83%. ROC-analysis demonstrated that low PPT in OA patients is a specific feature of central sensitisation (figure 1) Wind-up phenomena examination in intact region revealed significant difference of data in OA patients with reffered pain and control group (4.3±2.1 vs 2.44±1.3 p=0.003)and OA patients without reffered pain and control group (3.67±1.43 vs 2.44±1.3 p=0.011),

Figure 1 Diagonal segments are produced by ties.

Conclusions: Chronic OA is a complex of mechanisms and includes nociceptive and central sensitisation. Neurophysiological changes: low ppt in demaged area and even intact region and wind-up fenomena were revealed in all OA patients and charactercises central sensitisation. Mechanism-oriented treatment should also target CNS, including anticonvulsant, and antidepressant agents.

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