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Fig. Assessment of cartilage markers in synovial fluid at the 4 cycles. A) Study overview. B) type II collagen formation, PRO-C2. C) Aggrecan degradation, huARGS. D) Fibronectin degradation, FBN-C. Data are shown as median of available samples at each timepoint.

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OP0190 UNDERSTANDING CURRENT PRESCRIPTION DRUG TREATMENT PARADIGMS FOR PATIENTS WITH OSTEOARTHRITIS IN EUROPE

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Background: Joint pain is the most prevalent symptom for sufferers of osteoarthritis (OA). Pharmacological management of OA is restricted by limited efficacy and considerable toxicity, with growing fears about opioid use.

Objectives: To understand the current real-world prescribed drug treatment paradigm related to OA disease severity for patients in 5 EU countries; France, Germany, Italy, Spain and the UK.

Methods: Data were drawn from the Adelphi OA Disease Specific Programme (2017-18), a point-in-time study of physicians and their patients. Physicians classified their patients as currently having mild, moderate or severe disease severity, and provided details on currently prescribed OA therapy and physician satisfaction with therapy, rated from very satisfied to very dissatisfied. Patients were excluded from these analyses if they suffered from back and neck OA only, and shoulder OA that had not been diagnosed by X-ray. Comparisons among disease severity groups were made using analysis of variance and chi-squared tests.

Results: The study included 489 physicians (primary care physicians, rheumatologists, orthopaedists) reporting on 3596 of their OA patients: 24% mild (n=874), 53% moderate (n=1904), and 23% severe (n=818). Overall, 73% patients were prescribed at least one drug for their OA (65% of mild; 76% of moderate; 77% of severe patients [<0.001]). Paracetamol (34%) was the most commonly prescribed OA treatment. NSAIDs (31%) and opioids (27%) were also frequently prescribed treatments, and worsening severity was associated with an increase in opioid use (11% of mild; 26% of moderate, 47% of severe patients [<0.001]), but not NSAID (Table 1). The mean number of prescription medications increased (0.9 for mild; 1.4 for moderate; 1.6 for severe patients [<0.001]) and physician satisfaction with treatment decreased (86% for mild; 70% for moderate; 41% for severe [<0.001]) with worsening OA disease severity.

Table 1. Prescribed treatment by physician-reported OA severity

	Mild (n=874)	Moderate (n=1904)	Severe (n=818)
Current class of medication prescribed for OA, n (%)			
Paracetamol	186 (21.3)	663 (34.8)	313 (38.3)
NSAIDs	267 (30.5)	605 (31.8)	237 (29.0)
Any opioid	93 (10.6)	501 (26.3)	386 (47.2)
Weak opioid	82 (9.4)	407 (21.4)	255 (31.2)
Strong opioid	11 (1.3)	99 (5.2)	146 (17.8)
Opioid + analgesic (combined)	6 (0.7)	15 (0.8)	7 (0.9)
Corticosteroid	31 (3.5)	150 (7.9)	92 (11.2)
Glycosaminoglycan	50 (5.7)	149 (7.8)	62 (7.6)
Viscosupplement	12 (1.4)	93 (4.9)	42 (5.1)
Number of currently prescribed drug classes, mean (SD)	0.9 (0.8)	1.4 (1.1)	1.6 (1.2)

Conclusion: Physicians reported decreasing satisfaction with treatment for their OA patients as disease severity increased, despite increasing use of opioids and numbers of classes of prescribed drugs.

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OP0191 ASSOCIATION OF TRAMADOL WITH ALL-CAUSE MORTALITY, CARDIOVASCULAR DISEASE, VENOUS THROMBOEMBOLISM AND HIP FRACTURES AMONG PATIENTS WITH OSTEOARTHRITIS. A POPULATION-BASED STUDY

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Background: Both tramadol (narcotic-like drug) and nonsteroidal anti-inflammatory drugs (NSAIDs) are prescribed for pain relief among osteoarthritis (OA) patients. Evidence comparing risks of adverse events between tramadol and NSAIDs users is inconclusive.

Objectives: To examine the association of tramadol with all-cause mortality, cardiovascular disease (CVD), venous thromboembolism (VTE) and hip fractures (HFs) compared with NSAIDs and codeine in OA.

Methods: Design: Sequential propensity score-matched cohort study. Sample: All patients with OA who received medical care from 2005 to 2014 in the entire province of British Columbia, Canada. Tramadol cohort: Initial prescription of tramadol (n=56325). Four comparator cohorts: the initiation of the following: naproxen (n=13798), diclofenac (n=17675), cyclooxygenase-2 [Cox-2] inhibitor (n= 17039), or codeine (a weak opioid) (n=7813). Patients required to be prescribed neither tramadol nor its comparators during the year before the initial prescription date (i.e., index date). Outcomes: 1) all-cause mortality; first ever 2) CVD, 3) VTE, 4) HF within the 1st year after the initiation of tramadol or its comparators. Follow-up: from index date until the event occurred, disenrollment, or the end of a 1-year follow-up period. Statistical analysis: We created baseline covariates (demographics, comorbidities, medications and health resource utilization) from the year prior to the index date. Calendar years from 2005 to 2014 were divided into 10 blocks; propensity scores were calculated using logistic regression within each block. We used 1:1 greedy matching method. We estimated hazard ratios (HRs) using Cox proportional hazard models.

Results: After propensity score matching, 112650 patients with OA were included (mean age of 68 years, 62.8% were females). During the 1-year follow-up, 296 deaths (21.5/1000 person-years) occurred in the tramadol cohort and 246 (17.8/1000 person-years) in the naproxen cohort (Table 1). All-cause mortality was higher for tramadol compared with all NSAIDs cohorts, but not with the codeine cohort (Table 1, Figure 1). Tramadol initiators have also a higher risk of CVD and VTE compared with the diclofenac and Cox-2 inhibitor initiators with HRs ranging from 1.2 to 1.7. Furthermore, tramadol was also associated with a higher risk of HF compared with all NSAIDs cohorts (HRs ranging from 1.4 to 1.5). No significant difference was found between tramadol and codeine (Table 1).

Conclusion: OA patients initiating tramadol have an increased risk of mortality, CVD, VTE, and HF within 1 year compared with NSAIDs, but no statistically significant difference in the risk was observed between tramadol and codeine.