The Structural, Psychological, Functional and Pain Sensitization Characteristics of Presurgical Knee Osteoarthritis Patients with Evidence of Neuropathic Pain. A Prospective Observational study

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ABSTRACT INTRODUCTION: Neuropathic pain in patients with osteoarthritis (OA) listed for total knee replacement (TKR) surgery is clinically under-recognized by orthopedic surgeons worldwide. Recent evidence has reported that up to 34% patients with chronic OA have neuropathic pain.1 In the US it is estimated that 3.5 million primary TKR procedures will be performed by 20302 but up to 20% of these patients will continue to suffer from chronic postoperative pain in spite of objective measures of surgical success.3 Pre and post TKR treatment with Pregabalin (pharmacological treatment for neuropathic pain) can inhibit the development of chronic postoperative pain thereby inferring that preoperative neuropathic pain may predict outcome after TKR surgery.4 With the recent knowledge that pre TKR OA pain is not wholly due to joint pathology but also central and peripheral sensitization, it is clear that as orthopedic surgeons, an improved understanding of the mechanism based approach to pain in OA is required to improve patient outcomes after joint replacement.5 We conducted a prospective observational study in patients awaiting TKR surgery to identify those with neuropathic pain and to see the structural, psychological, functional and pain sensitization characteristics in those patients with this pain phenotype. The aims of this study were to identify the preoperative parameters of neuropathic pain in knee OA that possibly predict the development of chronic postoperative pain after TKR.

METHODS: Fifty patients (mean age 66.4 years [8.3 SD]) with chronic knee OA awaiting TKR surgery were recruited from orthopedics clinics in Nottingham, UK. Twenty-two healthy volunteers (mean age 56.7 years [9.0 SD]) with no OA or chronic pain condition were also recruited for comparison. The study was approved by the local ethics committee REC reference:10/H0408/115 and all participants took informed consent. All subjects underwent a 3-Tesla Knee MRI which was scored using the semi-quantitative MOAKS (MRI Osteoarthritis Knee Score) for severity of synovitis, effusion size and the total bone marrow lesions (BML) all of which are know peripheral drivers of pain in OA and were graded by one MSK radiologist and one orthopedic physician trained to complete the MOAKS score. All subjects completed self reported questionnaires to assess for neuropathic pain (PainDETECT®), depression (Beck Depression Inventory), anxiety (State-Trait Anxiety), pain catastrophizing (Pain Catastrophizing Scale) and function (Oxford Knee Score) as well as an objective assessment of the subject’s somatosensory system using Quantitative Sensory Testing to identify peripheral and central sensitization. The OA patients were divided into two age- and sex-matched groups according to neuropathic pain determined by the PainDETECT® questionnaire with a High PainDETECT® Score ≥19 indicating neuropathic pain assigned to Group A, and those with nociceptive or unclear pain based on PainDETECT® score ≤18 assigned to Group B. The 22 healthy volunteers with no chronic pain condition or osteoarthritis were assigned to Group C. The data was analyzed using Prism 7.0. A mixed-model analysis of variance (ANOVA) or Kruskal-Wallis ANOVA by Ranks Test was conducted. Pain Duration, Visual Analogue Pain Score and Oxford Knee Score was assessed between Groups A and B using the unpaired t-Test and Mann Whitney U Tests. P<0.05 was considered significant.

RESULTS SECTION: Fifteen out of 50 (30%) pre TKR OA patients had a PainDETECT® score ≥ 19 indicating neuropathic pain. Facilitated temporal summation and widespread hyperalgesia indicative of central sensitization was higher in the neuropathic pain patients (Group A) than those OA patients with predominantly nociceptive mixed pain phenotype (Group B) or healthy volunteers (Group C) (Kruskall Wallis Rank Test (Group A-B, p<0.0010), Group A-C, p=0.0005) and ANOVA (Group A-B, p=0018, Group A-C, p< 0.0001). Interclass correlation coefficient between the 2 radiological scorers for the MOAKS MRI score was 0.929 (0.79-0.98 95% CI). There was no difference between total BML, synovitis or effusion size scores, all of which are known peripheral drivers of pain in OA, using the MOAKS criteria between Groups A or B. Anxiety assessed using the State-Trait Anxiety questionnaire demonstrated higher levels of anxiety in Group A than Group B or C (Kruskall Wallis Rank Test Group A-B p <0.062, Group B-C p= 0.796 Group A-C, p <0.0005).

DISCUSSION: Neuropathic pain in OA is clinically under recognized in patients undergoing TKR in orthopaedic centres worldwide. In our cohort of patients, those that had neuropathic pain also had evidence central sensitization indicative of wide spread hyperalgesia and facilitated temporal summation. Those patients with neuropathic pain appear to have equivocal subchondral damage (BMLs), synovitis and effusion scores to those OA patients with purely nociceptive or mixed pain pattern. This work also complements recently published functional brain MRI work looking to central sensitization in knee OA linking the emotional aspect of pain to central sensitization.1 Enhanced neuropathic pain in chronic knee OA pain may also be linked to augmented responses to emotional circuitry evidence by high levels of anxiety within this cohort.

SIGNIFICANCE: 20% of all patients undergoing TKR surgery continue to suffer debilitating chronic postoperative pain despite objective measures of surgical success. Identification of neuropathic pain in OA and individualized medical treatment for those patients prior to TKR surgery may improve clinical outcome for this under-recognized cohort of patients.

REFERENCES:

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