controlled, longer RA duration) sustained the lowest mean SJC through-out follow-up. Cluster 4 patients (“health low–moderate, moderate RA”) exhibited the greatest improvement in mental health symptoms of pain and inflammation among OA patients. However, only a handful of observational studies evaluated the risk of a specific NSAID type among OA patients for a specific CVD event such as myocardial infarction (MI) (3). The overall cardiovascular safety of NSAIDs used in treating OA in the real-world therefore remains unknown.

**Objectives:** To evaluate the comparative safety of various NSAIDs against CVD when treating patients with OA.

**Methods:** We used linked health administrative data (HAD) of a previously as other follow population-based cohort of 720,055 British Columbians from Canada. We identified individuals with OA who received at least one NSAID prescription from January 1996 to December 2013. Eligible study subjects were at least 20 years old, did not have CVD and had not received an NSAID prescription within the last 90 days from their OA diagnosis date. We defined composite CVD outcome from hospital discharge abstract database, payment information file of Medical Services Plan and vital statistics death's file using ICD-9 or ICD-10 codes. We created an NSAID exposure variable in a time-dependent fashion in which individuals were considered at risk for the duration of NSAID prescriptions. We used time-dependent Cox regression analysis to estimate CVD risk associated with NSAID use overall as well as four unique groups of NSAIDs, i.e., coxibs, naproxen, ibuprofen and other conventional NSAIDs.

**Results:** Our cohort included 3,806 OA individuals. There were 1,147 CVD events. After adjusting for age, sex, SES, COPD, diabetes, hypertension, hyperlipidemia, peptic ulcer disease and Romano comorbidity score, the hazard ratio (HR) and 95% confidence interval (CI) from the time-dependent Cox regression model was 1.48 (1.27, 1.73). When exposure to different groups of NSAID was compared with unexposed person-time, CVD risk were similar among coxibs and naproxen followed by other conventional NSAIDs and ibuprofen, adjusted HR (95% CI) were 1.58 (1.24, 2.00), 1.58 (1.11, 2.24), 1.39 (1.10, 1.75) and 1.36 (0.75, 2.47), respectively.

**Conclusion:** Our study is the first retrospective cohort study using BC HAD that looked at the overall CVD risk of NSAID use in treating OA in a real-world setting. After modelling exposure to NSAIDs as time-dependent, we found that exposure to NSAIDs substantially increased overall CVD risk compared to non-exposed periods. We also found that coxibs and naproxen may increase CVD risk more than conventional NSAIDs.

**REFERENCES**


**Disclosure of Interests:** Mohammad Atoquzzaman: None declared, Ehsan Karim: None declared, Hubert Wong: None declared. Jacek Kopeć: None declared. Mohammad Atoquzzaman: None declared, Ehsan Karim: None declared, Hubert Wong: None declared. Jacek Kopeć: None declared. Scientific Abstracts

**SA0600 COMPARATIVE CARDIOVASCULAR SAFETY OF NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS) AMONG INDIVIDUALS WITH OSTEOARTHRITIS; FINDINGS FROM PROVINCIAL PRESCRIPTION CLAIM RECORDS IN BRITISH COLUMBIA, CANADA**

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**Background:** Osteoarthritis (OA) has been reported as an independent risk factor for cardiovascular diseases (CVD) (1). Furthermore, mediating role of NSAIDs in the observed OA-CVD association has also been noted (2). Thus, a substantial proportion of the total risk of CVD among OA patients compared to non-OA controls was attributable to NSAID use (2). This is particularly worrisome as there is no cure for OA and NSAIDs are the mainstay of treatment in controlling the primary symptoms of pain and inflammation among OA patients. However, only a handful of observational studies evaluated the risk of a specific NSAID type among OA patients for a specific CVD event such as myocardial infarction (MI) (3). The overall cardiovascular safety of NSAIDs used in treating OA in the real-world therefore remains unknown.

**Objectives:** To evaluate the comparative safety of various NSAIDs against CVD when treating patients with OA.

**Methods:** We used linked health administrative data (HAD) of a previously as other follow population-based cohort of 720,055 British Columbians from Canada. We identified individuals with OA who received at least one NSAID prescription from January 1996 to December 2013. Eligible study subjects were at least 20 years old, did not have CVD and had not received an NSAID prescription within the last 90 days from their OA diagnosis date. We defined composite CVD outcome from hospital discharge abstract database, payment information file of Medical Services Plan and vital statistics death's file using ICD-9 or ICD-10 codes. We created an NSAID exposure variable in a time-dependent fashion in which individuals were considered at risk for the duration of NSAID prescriptions. We used time-dependent Cox regression analysis to estimate CVD risk associated with NSAID use overall as well as four unique groups of NSAIDs, i.e., coxibs, naproxen, ibuprofen and other conventional NSAIDs.

**Results:** Our cohort included 3,806 OA individuals. There were 1,147 CVD events. After adjusting for age, sex, SES, COPD, diabetes, hypertension, hyperlipidemia, peptic ulcer disease and Romano comorbidity score, the hazard ratio (HR) and 95% confidence interval (CI) from the time-dependent Cox regression model was 1.48 (1.27, 1.73). When exposure to different groups of NSAID was compared with unexposed person-time, CVD risk were similar among coxibs and naproxen followed by other conventional NSAIDs and ibuprofen, adjusted HR (95% CI) were 1.58 (1.24, 2.00), 1.58 (1.11, 2.24), 1.39 (1.10, 1.75) and 1.36 (0.75, 2.47), respectively.

**Conclusion:** Our study is the first retrospective cohort study using BC HAD that looked at the overall CVD risk of NSAID use in treating OA in a real-world setting. After modelling exposure to NSAIDs as time-dependent, we found that exposure to NSAIDs substantially increased overall CVD risk compared to non-exposed periods. We also found that coxibs and naproxen may increase CVD risk more than conventional NSAIDs including ibuprofen.

**REFERENCES**


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**SA0601 MORTALITY RATE IN PATIENTS TREATED WITH BIOLOGICS: DATA FROM THE ROMANIAN REGISTRY OF RHEUMATIC DISEASES**

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**Background:** Rheumatoid arthritis (RA) is associated with increased mor-tality, with longitudinal studies averaging a standardised mortality ratio of

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**Background:** Rheumatoid arthritis (RA) is associated with increased mor-tality, with longitudinal studies averaging a standardised mortality ratio of
1.5 (95% CI 1.2 to 1.8) for RA patients compared to the general population [1]. In contrast, information on mortality in antikylosis spondylitis (AS) is scarce, whereas there are conflicting reports of the mortality risk among patients with psoriatic arthritis (PsA), but it is accepted that patients with PsA do not have a significantly elevated risk of mortality [2].

**Objectives:** To estimate the Mortality Rate in RA, SA and PsA for patients (pts) treated with biologics in Romania.

**Methods:** Data were gathered from the Romanian Registry of Rheumatic Diseases (RRBR), which comprises all patients treated with biologics in Romania for RA, AS and PsA. We have studied demographic data, disease exposure, treatment in person-years (PY), and all-cause mortality until 01.01.2018.

**Results:** The cohort included 9577 pts (35596.83 PY) as following: 5224 RA pts (18676.75PY), 3469 AS pts (12680.63PY) and 884 PsA pts (4212.45PY). The mean age of the cohort was 54.86 yrs (62.87 yrs for RA, 45.54 yrs for AS, 56.18 yrs for PsA); 3196 pts (33.37%) were men: 79 (1.5%) RA pts, 2681 (77.28%) AS pts and 436 (49.3%) PsA pts. Mean disease duration for the cohort was 16.46 yrs for RA, 11.46 yrs for AS and 10.99 yrs for PsA. The number of all-cause deaths was 89 (74 deaths in RA, 11 in AS and 4 in PsA group). The mortality rate for the entire cohort was 0.25/100PY, and the rate varied across the diseases: 0.39/100PY in RA pts, 0.08/100PY in AS pts and 0.09/100PY in PsA. Infections were the major mortality cause in RA (0.09/100PY) and AS (0.04/100PY). Cardiovascular fatal events occurred in 0.06/100PY in RA population, significantly higher than 0.007/100PY in AS pts. Rate of s of neoplasm death in RA cohort was 0.004/100PY, compared to 0.007/100PY in AS; non-Hodgkin lymphoma was recorded only in RA pts (0.005/100PY). All deaths reported in PsA cohort (4 deaths) were of unknown cause.

**Conclusion:** This is the first report on mortality rate in biological treated patients in Romania. These data support data from the literature, showing that RA patients have a higher mortality risk compared to AS and PsA.

**REFERENCES**


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**SAT0602 SHOULD ALL PATIENTS WITH ANTI-CENTROMERE ANTIBODIES BE REFERRED FOR A RHEUMATOLOGY ASSESSMENT?**

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**Background:** Anti-centromere antibodies (ACA) are commonly associated with systemic sclerosis (SSc). The presence of ACAs in patients with SSc is known to increase the likelihood of developing Pulmonary Hypertension, which has a high mortality rate. ACAs are also seen in patients with other connective tissue diseases (CTD) and can sometimes be identified after testing for antinuclear antibodies in patients who have not reported any rheumatological symptoms. It is possible that connective tissue diseases are being under diagnosed due to a lack of awareness by physicians of the significance of positive ACAs.

**Objectives:** To investigate whether patients with ACAs are being appropriately referred to the Rheumatology service.

**Methods:** All patients who were positive for ACAs in Southend university hospital between April 2016 and October 2018 were included in this single centre retrospective observational study. We identified patients demographics, diagnosis, ANA titre, additional diagnosis and immunosuppressive therapy. We also captured their monitoring with pulmonary function testing and echocardiography.

**Results:** A total of 75 patients were identified with ACAs. The average age was 65 years, 61 females, 14 Male. Fifty-six patients were referred to rheumatology team and were found to have the following diagnosis, LcSSc (21), Sjogren’s syndrome (10), undifferentiated connective tissue disease (UCTD) (6), Rheumatoid arthritis (RA) (5), ANCA associated vasculitis (AAV) (3), Raynaud’s phenomenon (3), Lupus (2), Antiphospholi- pid syndrome (APS) (1). Primary biliary cirrhosis with Sjogren’s syndrome (SS) (1), Juvenile idiopathic arthritis (1), Antisynthetase syndrome (AS) (1), Autoimmune hepatitis (1) and osteoarthritis (1). Of those 33 patients had a routine screening with an Echocardiogram and 26 had pulmonary function tests. One patient with LcSSc developed pulmonary hypertension. The remaining 19 patients were not referred and did not have the adequate screening for pulmonary hypertension. ANA titre was 1:80 in one patient, 1:320 for 4 patients, unknown for three and 1:640 for the remaining 67. 11 patients were treated with Hydroxochloroquine (4 SS, 4 UCTD, 1 lupus, 1 APS and 1 Lc Ssc), 5 on Methotrexate (4 RA and 1 AS), 2 on MMF and steroid (RAA).

**Conclusion:** Nearly all patients with ACAs that were seen in the Rheumatology clinic had an autoimmune rheumatic disease. However, we found that 25% of people with ACAs were not referred to the Rheumatology service. The reasons for this are unclear. It is possible that patients did not report symptoms that would have prompted a referral. Some of the CTDs in which ACAs are typically found (LcSSc and SS) are associated with symptoms that can be mild and might not be reported by the patients or general physicians do not associate them with rheumatological disorders (e.g. sicca symptoms, gastro-esophageal reflux and Raynaud’s phenomenon). Early diagnosis might enable earlier treatment and prevent complications from these diseases. General physicians should therefore be made aware of these antibodies and the disorders that they can be associated with.

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