kidney cancer (HR 1.60 [95%CI 1.15-2.23]), sarcoma (HR 2.14 [95%CI 1.41-3.24]), acute leukemia (HR 1.81 [95%CI 1.06-3.07]), chronic leukemia (HR 1.62 [95%CI 1.19-2.27]), Hodgkin’s lymphoma (HR 2.42 [95%CI 1.12-5.20]), non-Hodgkin’s lymphoma (HR 1.66 [95%CI 1.21-2.28]) and multiple myeloma (HR 2.40 [95%CI 1.63-3.53]) (Table 1). The time (mean [months] ± SD) to the diagnosis of any malignancy was significantly shorter in GCA patients (48.6 ± 41.3) compared to controls (58.1 ± 43.6; p<0.001).

Conclusion: GCA patients are at increased risk for sarcoma, kidney cancer, hematological malignancies and overall malignancies compared to age-and-sex matched controls from the general population.

References:

Disclosure of Interests: None declared

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Figure 1. Kaplan-Meier cancer-free survival curve

OP0144

EFFECT OF TOCILIZUMAB ON VASCULAR INFLAMMATION BY 18F-FLUORODEOXYGLUCOSE POSITRON EMISSION TOMOGRAPHY: A PROSPECTIVE, LONGITUDINAL STUDY

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Background: Two randomized controlled trials have demonstrated clinical efficacy of tocilizumab for the treatment of giant cell arteritis (GCA)(1, 2). In these trials, clinical and laboratory measures were used to define the outcome measures. The direct effect of tocilizumab on vascular inflammation remains poorly characterized.

Objectives: To prospectively evaluate vascular inflammation as measured by 18F-fluorodeoxyglucose (FDG) positron emission tomography (PET) in a longitudinal cohort of patients with GCA treated with tocilizumab over a several year follow-up period.

Methods: Patients with GCA who were recruited into a prospective, observational cohort. All patients fulfilled modified 1990 American College of Rheumatology (ACR) Classification Criteria for GCA. All patients underwent FDG-PET computed tomography (CT) prior to initiation of tocilizumab. A single reader reviewed all PET scans, blinded to clinical data. Qualitative assessment of FDG uptake relative to liver uptake by visual assessment (scale 0-3) was assessed in 9 arterial territories. A summary score, PET Vascular Activity Score (PETVAS), was calculated (scale 0-27).

Patients underwent imaging at 6-12 month intervals per a standardized imaging protocol. In a subset of patients in whom tocilizumab was discontinued due to established remission, a repeat FDG-PET scan was obtained within 6 months of drug discontinuation.

Change in PET activity over time was measured by linear regression. PET activity during established remission was compared to PET activity after discontinuation of tocilizumab. For some patients, tocilizumab was added to the existing treatment regimen without a substantive change in concomitant glucocorticoid dose. In a secondary analysis, patients were stratified by prednisone dosing (high dose prednisone >10mg/day prednisone, low dose prednisone ≤10mg/day prednisone during the imaging interval) to determine if tocilizumab had an effect on vascular inflammation independent of glucocorticoids.

Results: 22 patients were included in the study. All patients had clinically active disease at baseline with median baseline PETVAS 24.5 (23-27). There was a significant reduction in PETVAS over 2 years follow up (p<0.01 for linear trend) (Figure). Of note, there was continued progressive improvement in PETVAS in both year 1 and year 2 of treatment. Eight patients received concomitant high dose glucocorticoids and 14 patients remained on low dose glucocorticoids with the addition of tocilizumab. In patients who only received low dose prednisone, significant reduction in PETVAS was still observed with addition of tocilizumab (PETVAS 25.5 to 19.5, p=0.04). In a subset of 5 patients who discontinued tocilizumab due to established remission (median PETVAS 19 (17.3-22) at time of remission), a repeat FDG-PET scan within 6 months after treatment discontinuation showed worsening PET activity in 4 out of 5 patients (median PETVAS 23 (20-23)) Two of these patients subsequently experienced a clinical disease relapse.

Conclusion: Tocilizumab was associated with improved vascular inflammation as assessed by FDG-PET. There was continued improvement of vascular inflammation at both year 1 and year 2 of treatment, and early evidence suggests a rebound of vascular inflammation when tocilizumab was discontinued. These data provide rationale for long-term tocilizumab therapy in patients with GCA and for consideration of FDG-PET as an outcome measure in future clinical trials.

References:

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Figure. XXX

OP0145

MALIGNANCY IN ANCA-ASSOCIATED VASCULITIS AND POLYARTERITIS NODOSA: AN AUSTRALIAN POPULATION-BASED STUDY

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Background: The increased risk of malignancy in patients with ANCA-associated vasculitis (AAV) and polyarteritis nodosa (PAN) has been attributed to late aortic vasculitis (AAV) and polyarteritis nodosa (PAN) has been attributed to late

Figure. XXX

Disclosure of Interests: None declared

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OP0145

MALIGNANCY IN ANCA-ASSOCIATED VASCULITIS AND POLYARTERITIS NODOSA: AN AUSTRALIAN POPULATION-BASED STUDY

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Background: The increased risk of malignancy in patients with ANCA-associated vasculitis (AAV) and polyarteritis nodosa (PAN) has been attributed to late

Figure. XXX

Disclosure of Interests: None declared

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MALIGNANCY IN ANCA-ASSOCIATED VASCULITIS AND POLYARTERITIS NODOSA: AN AUSTRALIAN POPULATION-BASED STUDY

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Background: The increased risk of malignancy in patients with ANCA-associated vasculitis (AAV) and polyarteritis nodosa (PAN) has been attributed to late
Long term outcome and prognosis factors of isolated aortitis

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Background: Aortitis is a group of disorders characterized by the inflammation of the aorta. The most common causes of aortitis are the large-vascular vasculitis i.e. giant cell arteritis (GCA) and Takayasu arteritis (TA). However, aortitis may be isolated. Because of the wide variation in the course of aortitis, predicting outcome is challenging. The optimal management strategy of isolated aortitis (IA) is still unclear as IA is poorly defined, with data consisting of small retrospective and case control studies.

Objectives: To assess the long-term outcome and prognosis factors for vascular complications in patients with isolated aortitis.

Methods: Retrospective multicenter study of 353 patients with non-infectious aortitis including 138 giant cell arteritis (GCA), 96 Takayasu arteritis (TA) and 73 isolated aortitis (IA). Factors associated with event-free survival, vascular event-free survival and revascularization-free survival were assessed. Risk factors for vascular complications were identified in multivariate analysis.

Results: After a median follow up of 52 months, vascular complications were observed in 32.3% , revascularization in 30% and death in 76%. The 5-year cumulative incidence of vascular complications was 58% (41; 71), 20% (13; 29), and 19 % (11; 28) in IA, GCA and TA, respectively. In multivariate analysis, IA [HR, 1.85 (1.19 to 2.88), p=0.017] and male gender [1.77 (1.26 to 2.49), p<0.0001] were independently associated with vascular events. The 5-year surgery-free survival was 45% (31; 65), 71% (62; 81) and 76% (68; 86) in IA, TA and GCA, respectively.

Conclusion: IA has a worse vascular prognosis than GCA and TA. Sixty percent of IA patients will experience a vascular complication within 5 years from diagnosis.

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