

ARP36
SIR BENJAMIN COLLINS BRODIE (1783–1862) – A PIONEER RHEUMATOLOGIST

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Aim: Benjamin Brodie was a London surgeon who investigated joint disease by observation and morbid anatomy for over thirty years in the first half of the 19th century. He summarised his rheumatology experience in his book 'Pathological and Surgical Observations on the Diseases of Joints'. Others have briefly mentioned his contributions, but to date no detailed analysis has been done.

Methods: The authors used the first (1818), third (1834) and fifth (1850) editions of Brodie's book to give a more comprehensive picture of his achievements.

Results: The descriptions of disease given before about 1850 are often unintelligible now, yet Brodie's are admirably clear. He describes reactive arthritis in the first edition of his book more completely than authors before him, or even Reiter a century later. He recognised that the conjunctivo-urethral-synovial syndrome can occur independently of gonorrhoea, that there are often repeated attacks, and that iritis is a complication – the first indication that this syndrome is part of what we now call seronegative spondyloarthritis. Brodie showed that there were causes of joint inflammation other than tuberculosis or pyogenic infection, and was proud of having saved many limbs that would otherwise have been amputated.

The fifth edition (1850) has among the earliest descriptions in English of ankylosing spondylitis and rheumatoid arthritis.

Conclusions: Brodie was showered with honours (Fellowship of the Royal Society, Presidency of the Royal College of Surgeons, a Baronetcy) and had a very lucrative private practice, but none of this detracted from his work and his clinical teaching at St. George's Hospital. His appreciation of rheumatic disease demands him to be widely recognised as a pioneer rheumatologist.

ARP37
STATIN ASSOCIATED NECROTIZING AUTOIMMUNE MYOPATHIES IN THE INDIGENOUS POPULATION: A CASE SERIES FROM NORTH QUEENSLAND

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Aim: To describe clinical and histopathological features of statin associated necrotizing autoimmune myopathies (NAM) in Indigenous Australians and increase awareness of this condition amongst treating physicians.

Methods: Cases were collected through the Rheumatology Department at The Townsville Hospital between March 2012 and January 2015. A chart review was performed to obtain retrospective information about each case. We detail patient demographics, presenting features, histopathological findings, autoimmune profile, treatment and outcomes.

Results: 4 Indigenous Australians were identified as having a biopsy confirmed statin associated NAM. All patients had been on atorvastatin for at least 2 years and had significant proximal weakness with average CK level on presentation 16,820 U/L. Predisposing factors for myopathy included vitamin D deficiency and diabetes mellitus (all cases), with primary hypothyroidism and liver cirrhosis identified in two other cases. Two individuals were positive for the auto-antibody anti-HMGCR. Histopathological findings included muscle necrosis with varying degrees of inflammation, membrane attack complex (MAC) deposition and MHC-I upregulation. Treatment involved various combinations of prednisolone, IVIG, methotrexate and mycophenolate. Recovery was slow but favourable in all cases with an average length of inpatient stay of 54 days. There was a significant delay in diagnosis of 1–3 months in two of the cases.

Conclusions: The statin associated necrotizing autoimmune myopathies are rare but important disorders that cause significant morbidity to affected individuals. Given the prevalence of cardiovascular disease in Indigenous

Australians, further research is required to facilitate earlier diagnosis and improved treatment outcomes.

ARP38
EPIDEMIOLOGY OF ANCA-ASSOCIATED VASCULITIS IN TOWNSVILLE, NORTH QUEENSLAND

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Aim: The primary aim of the study was to obtain the prevalence of ANCA-associated vasculitis (AAV) in Townsville. A secondary aim was to compare this study with other centres with different latitudes in Australia and New Zealand. A latitudinal variance in disease rates for ANCA-associated vasculitis has been shown in Europe and New Zealand.

Methods: The pathology database at Townsville Hospital was respectively searched for all positive ANCA tests between 2009 and 2013. Patient files were reviewed for all positive results to determine whether they met the ACR criteria for granulomatosis with polyangiitis (GPA) or Chapel Hill criteria for microscopic polyangiitis (MPA) or eosinophilic granulomatosis with polyangiitis (EGPA).

Results: 5013 ANCA tests were requested at Townsville Hospital in the 5 year study period. 61 patients had positive ANCA results. 23 patients met criteria for ANCA-associated vasculitis with 13 cases of MPA, 6 cases of GPA, 2 cases of EGPA and 2 others. 14 out of those 23 patients lived in the Townsville Health District. Townsville Health District had a population of 230,000 in 2010 giving a prevalence of 61 cases of AAV/million population over 5 years. MPA had a rate of 34/million and GPA 22/million.

Conclusions: This study shows lower rates of AAV than other southern hemisphere studies in Christchurch (151/million/5 years) or Australian Capital Territory (148/million/5 years). These results add further evidence to the observed latitudinal difference in disease. The study may have underestimated the disease burden due to a number of factors. Firstly, it did not include a local private hospital and secondly, it did not include cases that had ANCA tests performed at private laboratories or before the study began.

ARP39
STATIN ASSOCIATED AUTOPHAGIC VACUOLES IN IDIOPATHIC INFLAMMATORY MYOPATHIES AND NECROTISING MYOPATHY

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Aim: The aim of this study was to review and compare the clinical, pathological and ultrastructural features of patients with biopsy proven idiopathic inflammatory myositis (IIM) and necrotising myopathy (NM) exposed to statin with those who were statin naive.

Methods: All muscle biopsies (myositis and non-myositis) performed in South Australia between January 2012 and August 2013 inclusive were reviewed with respect to statin exposure. One author blinded to biopsy results, collected data related to statin exposure using standardised biopsy forms and obtained missing data by checking medical records and contacting health providers. Patients were classified on the basis of histological criteria and routine electron microscopy was performed on all specimens.

Results: IIM and NM comprised 78/186 of biopsies i.e. 42% of all cases reviewed. (17/78 PM, 16/78 IBM, 10/78 DM, 21/78 MNOS and 14/78 NM) 41/78 (53%) were exposed to statin in this group. 'Non-myositis' control cases comprised 108/186 and 21/108 (19.4%) had been exposed to statin. In 9/78 'myositis' biopsies distinctive electron dense granules (0.2–0.5 μm) within single membrane bound vacuoles were noted (type 2 autophagic vacuoles).

Conclusions: We report a distinctive ultrastructural abnormality with features of a late autophagic vacuole observed in 9/41 cases of immune mediated myopathies. All of these patients were exposed to statins. We speculate that the statin induced changes in cholesterol metabolism of cell membranes in some way interferes with the multistep process of autophagy in the setting of muscle fibre necrosis/regeneration and inflammation. The LM observation of vacuolar changes in otherwise normal fibres in all 9 cases showing AV-2s