uncertain: systemic inflammation and anti-rheumatic therapy may be of important. The use of methotrexate (MTX) has been linked to a reduced presence of MetS, via an assumed generic anti-inflammatory mechanism. We aimed to assess the prevalence of the MetS in RA; to identify factors that associate with its presence; and to investigate how they may interact with the potential influence of methotrexate.
Methods: MetS prevalence was assessed cross-sectionally in 400 consecutive RA patients, using five MetS definitions (National Cholesterol Education Programme (NCEP) 2004 and 2001, International Diabetes Federation (IDF), World Health Organisation (WHO) and European study Group for Insulin Resistance (EGIR)). Logistic regression was used to identify independent predictors of the MetS. Further analysis established the nature of the association between MTX and the MetS in RA.
Results: Prevalence rates of the MetS varied from 12.1% to 45.3% according to the definition used, mirroring similar differences reported in the general population. Factors associated with the presence of the MetS in RA included older age, a higher Health Assessment Questionnaire -(HAQ) score and less methotrexate use. Use of MTX, but not of other disease modifying anti-rheumatic drugs (DMARDs) or glucocorticoids (GC), associated with significantly reduced chance of having the MetS in RA (OR = 0.517) (CI 0.33-0.81), p=0.004)
Conclusions: The prevalence of the MetS in RA varies greatly according to the definition used. RA disease-specific factors, including disease severity associates with its presence. MTX therapy, but not other DMARDs or GC, independently associates with reduced propensity to MetS, suggesting a drug-specific, rather than generic anti-inflammatory mechanism and makes MTX a good choice as the first line DMARD in RA patients at high risk of developing the MetS.
Disclosure: All authors have declared no conflicts of interest.

TABLE 1. Odds ratio for developing the metabolic syndrome in patients receiving methotrexate compared to those not on methotrexate

	Odds Ratio (95% confidence interval)
Crude	OR = 0.483 (0.32–0.73), p = 0.001
Model a	OR = 0.505 (0.33–0.77), p = 0.001
Model b	OR = 0.525 (0.34–0.80), p = 0.003
Model c	OR = 0.517 (0.33–0.80), p = 0.004

Crude: Unadjusted model. Model a: Adjusted for age and sex. Model b: Adjusted for age, sex, disease. duration, ESR, and health assessment questionnaire score Model c: Adjusted for age, sex, disease duration, ESR, health assessment questionnaire score, sulphasalazine, hydroxychloroquine, leflunomide, andi-tumour necrosis factor therapy, and glucocorticoid use.

OP54. FACTORS ASSOCIATED WITH WORK DISABILITY IN RHEUMATOID ARTHRITIS PATIENTS: RESULTS FROM THE BSR BIOLOGICS REGISTER (BSRBR)

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Background: There is limited data available on the effect of anti-TNF therapy on working status in patients with rheumatoid arthritis (RA) in the UK. The aim of this study was, (i) to describe working status at start of anti-TNF treatment, and (ii) to identify predictors of onset of new work disability.
Methods: 2,703 patients with RA registered with the BSRBR between 01/10/2001 and 01/09/2008 (aged<62 yrs) as starting anti-TNF therapy were included in this study. Patients were asked to describe their working status at baseline and again at 3 yrs. Data collected at baseline included age, gender, disease duration, HAQ-score, and DAS28. DAS28 score was also collected at 6 months. Baseline characteristics were compared between the group of patients who were working and those who reported that they were not able to work/or retired due to ill health (i.e. work disabled). Predictors of new work disability at follow-up among patients initially working at start of anti-TNF therapy were identified using univariate and multivariate logistic regression analyses. Covariates included baseline characteristics, EULAR good response, and remission at 6 months.
Results: At baseline, 37.6% of the patients reported they were working, 48.7% was work disabled, 5.3% was working in the home, 0.6% was unemployed, 0.7% was student and 7.1% was retired. Except for gender, all baseline characteristics were significantly different between working patients and work disabled patients, resp.: mean (SD) age (48 (9) vs 52 (7)), median disease duration (10 vs 12 yrs), percentage female (74% vs 78%), mean (SD) HAQ-score (1.7 (0.6) vs 2.2 (0.4)), and mean (SD) DAS28 score (6.4 (1.0) vs 6.7 (1.0)). Among those patients who reported that they were not able to work at baseline due to ill health, 53 patients were working 3 yrs after starting anti-TNF therapy. Of 903 patients working at start of anti-TNF therapy, 90% was still working at 3 yrs and 10% went on to develop work disability. Factors unable to predict new onset work disability (OR; 95% confidence interval) included gender (OR 0.92; 0.56, 1.51), age (OR 1.02; 0.99, 1.05), and log disease duration (OR 0.91; 0.49, 1.68). However, a higher HAQ-score at baseline was associated with new work disability at follow-up (OR 2.46; 1.62, 3.76). There was also a trend towards less new work disability in those patients fulfilling the EULAR good response criteria (OR 0.67; 0.41, 1.09) and those in remission at 6 months (OR 0.55; 0.29, 1.04). In the multivariate regression analysis, only a high HAQ-score was associated with new work disability at follow-up (OR 2.51; 1.62, 3.91).
Conclusions: There is a trend towards less future work disability in working patients who responded to anti-TNF therapy. With a median disease duration of 12 yrs, 49% was already work disabled at start of anti-TNF therapy. Earlier introduction of anti-TNF therapy may prevent patients with RA from becoming work disabled in the future.
Disclosure: All authors have declared no conflicts of interest.

Concurrent Oral 9 – Case Reports

OP55. RITUXIMAB IS EFFECTIVE IN REFRACTORY INFLAMMATORY EYE DISEASE ASSOCIATED WITH RHEUMATOID ARTHRITIS

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Background: Scleritis is an inflammatory eye condition that can develop secondary to rheumatoid arthritis (RA). The standard treatment for severe disease comprises steroids (topically and/or systemic) and cyclophosphamide and there are variable responses to TNFalpha inhibitors (TNFi). The management of scleritis that is refractory to the above therapies, or in situations where they are contraindicated, is problematic.

Rituximab is a depleting anti-CD20 monoclonal antibody licensed for the treatment of RA that is unresponsive to TNFi. We report a series of four consecutive patients with RA and refractory scleritis who were successfully managed by rituximab.
Methods: All patients had seropositive erosive RA and had severe scleritis. Two developed scleritis whilst on etanercept, which was subsequently discontinued to allow a course of cyclophosphamide, which proved to be ineffective. In the other two patients, the use of a TNFi or cyclophosphamide was contraindicated because of co-morbidities. Each patient was treated with a standard cycle of two infusions of rituximab at 1000mg/infusion, two weeks apart.
Results: Following the cycle of rituximab, the scleritis resolved fully in all four patients (requiring between 2 weeks and 3 months for reduction in activity) and in parallel there was a significant improvement in activity of RA. There has not been a relapse in scleritis to date and no further immunosuppression has been required, with the period of remission ranging from two months to two years.
Conclusions: We have found rituximab to be a valuable treatment option for patients with RA and severe scleritis, when conventional therapy has failed, or where such treatment is contraindicated. Formal trials will be required to determine the optimal timing for such therapy in the future.
Disclosure: All authors have declared no conflicts of interest.

OP56. SPONTANEOUS PNEUMOMEDIASTINUM SECONDARY TO REFRACTORY DERMATOMYOSITIS SUCCESSFULLY TREATED WITH RITUXIMAB: A CASE REPORT AND REVIEW OF THE LITERATURE

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Background: Spontaneous pneumomediastinum has been described in patients with dermatomyositis (DM) and polymyositis. Literature reviews suggest that this complication has a mortality of between 27 and 41%. Rituximab has been shown to be effective in treating refractory DM, but as yet there have been no reports of it being used as part of the treatment for pneumomediastinum.
Methods: A 25-year-old female was referred for an out-patient rheumatology opinion with a widespread erythematous rash and mild proximal muscle weakness. Biochemistry revealed a creatinine kinase (CK) of 358 iu/l and lung function tests were normal. A chest, abdomen and pelvic CT scan excluded malignancy. A skin biopsy was consistent with a diagnosis of DM. Clinical features improved with methotrexate (MTX) and prednisolone therapy.

The patient represented 2 years later with worsening proximal muscle weakness and a CK of 2441 iu/l. She was treated with IV methylprednisolone and IV immunoglobulin in addition to MTX.

The patient was admitted 3 days after completing IV immunoglobulin therapy with worsening dysphagia and dyspnoea. The CK had risen to 5369 iu/l. Blood gases revelaed type 1 respiratory failure and she was transferred to ITU. A CXR revealed a right-sided pneumothorax and a HRCT scan of the thorax demonstrated free gas in the mediastinum with no underlying interstitial lung disease. A diagnosis of spontaneous pneumothorax and pneumomediastinum secondary to DM was made and a regimen of rituximab (1g IV on days 1 and 14), cyclophosphamide (740mg IV on days 2 and 15) and methylprednisolone (500mg IV on days 2 and 15) was commenced.
Results: The patient's condition improved and the CK normalised within 2 weeks of commencing the rituximab regimen. A CD19+ cell count indicated effective depletion of CD20+ B cells. A PET scan excluded an underlying malignancy.

The patient's muscle disease remains in remission on MTX maintenance almost 2 years after rituximab therapy. However, her skin disease is still problematic.
Conclusions: Case series of spontaneous pneumomediastinum in DM have suggested that this complication tends to occur in younger patients with recent onset disease, DM-specific skin disease, interstitial lung disease and negative

anti-Jo1 antibodies. Rituximab has been shown to be an effective therapy for cases of DM refractory to conventional immunosuppressive therapy.

This is the first reported case of spontaneous pneumomediastinum in DM successfully treated with a rituximab regimen. The patient demonstrated DM-specific skin disease and anti-Jo1 antibody negativity. However, there was no evidence of interstitial lung disease on HRCT.

Rituximab should be considered in the treatment of refractory DM, particularly if complicated by potentially life-threatening manifestations such as pneumomediastinum.

Disclosure: All authors have declared no conflicts of interest.

OP57. RITUXIMAB AS PRIMARY THERAPY FOR PULMONARY HAEMORRHAGE IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Pulmonary Haemorrhage (PH) is a rare and potentially life-threatening complication of Systemic Lupus Erythematosus (SLE). Early initiation of combination treatment with high-dose steroids and cyclophosphamide(CYC) is recommended. Some studies suggest additional benefit with plasma exchange but limited treatment options exist for those who fail to respond. We report the first use of Rituximab(RTX) for the primary treatment of SLE associated PH in combination with high dose steroids and CYC.

Methods: A 52 year old woman with a 12 year history of SLE (malar rash, leucopenia, ANA+ve, dsDNA+ve and previous WHO grade IV lupus nephritis) presented to our hospital with fever, dry cough, abdominal pain, intermittent diarrhoea, and a new onset vasculitic rash on her leg. There was serological evidence of active SLE with low complement (C3 0.47g/L, C4<0.04g/L) and raised anti-dsDNA(178 IU/ml) but normal full blood count and admission chest radiograph. A CT abdomen revealed small and large bowel thickening with free peritoneal fluid. A diagnosis of lupus related vasculitis was made and she was managed conservatively with intravenous (iv) fluids and steroids. Three days later she became increasingly tachycardic and tachypnoeic with bibasal crepitations and type 1 respiratory failure. A CTPA demonstrated areas of pulmonary infiltrate in the upper lobes consistent with an infective or haemorrhagic process, bibasal pleural effusions and collapse with no pulmonary emboli. Laboratory investigations revealed new onset anaemia (Hb 8.9 g/dL) and thrombocytopaenia (108×109/l). Treatment was commenced with iv antibiotics, pulsed iv methylprednisolone (total 3g) and co-trimoxazole, despite which on day 7 her condition deteriorated with worsening type I respiratory failure and cytopenia.

Results: On day 8, bronchoscopy confirmed PH, with negative culture and microscopy on BAL. She was transferred to the ITU for non-invasive ventilatory support and 3 unit blood transfusion. On day 9 (and day 25) she received iv Rituximab 1g (750mg/m2) and on day 10, a single dose of 750mg iv CYC. Her 7 day ITU admission was characterised by a rapid clinical improvement in respiratory function and cytopaenias. She was subsequently discharged home on prednisolone 40mg/day, tapered to 5mg over 6 months. She remains asymptomatic.

Conclusions: This case reports the first use of RTX for the primary treatment of SLE associated PH. Given the early (within a week) and sustained improvement (to 6 months) in both the clinical and laboratory features of our patient's illness it is likely that B cell depletion ameliorates disease by a number of different mechanisms (such as loss of B cell mediated antigen presentation, cytokine production or T cell regulation) in addition to a reduction in pathogenic autoantibodies. Therefore, we believe further studies are warranted.

Disclosure: All authors have declared no conflicts of interest.

OP58. RASBURICASE IN TREATMENT FAILURE GOUT

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Background: Severe chronic tophaceous gout can be a challenging condition to treat in the presence of true hypersensitivity to, or intolerance of allopurinol and in the presence of chronic kidney disease. Rasburicase (recombinant urate-oxidase enzyme) is currently licensed in the treatment and prevention of chemotherapy-induced hyperuricaemia at 0.2 mg/kg for seven days. It has recently been shown to be effective in reducing serum urate and tophus bulk.

Methods: A 47 year old male, with a thirty year history of chronic tophaceous gout and intermittent polyarticular involvement, presented with widespread discharging subcutaneous tophi and systemic upset. Over recent months he had been unable to work due to severe gouty arthritis. He had a background of alcohol excess. He had previously been treated with Allopurinol producing a rash and at smaller doses, disease flares. Sulphinpyrazone caused unacceptable nausea.

Results: He was overweight (BMI = 31kg/m2) and normotensive (126/77). Large, discharging tophi were noted over the small joints of the hands and feet, both pinnae and olecranon. There were bilateral knee effusions and inflamed subcutaneous deposits over the extensor aspects of both arms and thighs. He had normal renal function, serum urate was 0.55mg/l, CRP 277mg/l and X-rays confirmed juxta-articular erosions. Treatment with anti-inflammatories (NSAIDs), oral prednisolone and broad spectrum antibiotics was commenced although subsequent cultures were negative. In view of the extreme nature of his presentation in addition to previous intolerance of and ineffectiveness of Allopurinol and Sulphinpyrazone, Rasburicase was given in the form of six, monthly infusions at 0.2mg/kg. During treatment he has experienced one short-lived disease flare but has now discontinued NSAIDs and all analgesia. He has returned to full-time employment. Urate remains at 0.56mg/l. Tophus volume has significantly improved with near complete resolution of subcutaneous deposits. Allopurinol has been commenced in a low-dose, desensitisation fashion.

Conclusions: This gentleman's successful treatment to date with Rasburicase adds to the evidence this is an effective 'bridge' therapy in severe and difficult-to-treat cases of gout. Although hypersensitivity and loss of efficacy have been concerns with repeated infusions in trial subjects with haematological malignancy, this was not the case in our patient. Rasburicase may offer an effective means to bringing such cases under rapid symptomatic control before commencing definitive long term urate lowering therapy.

Disclosure: All authors have declared no conflicts of interest.

OP59. THE VITAMIN D AND CALCIUM CHALLENGE TEST

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Background: Hyperparathyroidism is common in patients with vitamin D deficiency. Rarely this relationship can become dysfunctional with inappropriate autonomous hyperparathyroidism. We present 2 cases of autonomous parathyroid function and hypercalcaemia revealed by treatment of vitamin D deficiency.

Methods: Case one is a 53 year old Somali woman resident in the UK for the last 5 years. She presented with back pain and difficulty climbing stairs. She had unrecordable levels of vitamin D, elevated alkaline phosphatase(ALP) 209 IU/L(30-115) and calcium of 2.49 mmol/L(2.20-2.60). A diagnosis of osteomalacia was made and the patient commenced on calcium and vitamin D supplementation. The patient was admitted following 5 days of treatment with a calcium of 3.69 mmol/L(2.20-2.60) and PTH of 75 pmol/L(0.9-5.4). She received intravenous pamidronate and hydration. A left sided neck mass was confirmed on ultrasound and sestamibi scan as a parathyroid adenoma. She underwent parathyroidectomy with subsequent normalisation of calcium levels.

Results: Case two is a 68 year old woman who presented with a waddling gait and difficulty climbing stairs. She had a raised Alkaline phosphatase level of 261 IU/L(30-120),an undetectable vitamin D level and a markedly elevated PTH of 68.7 pmol/L, suggestive of osteomalacia with secondary hyperparathyroidism. Calcium and liver biochemistry were normal. She was started on calcium and vitamin D supplements. Repeat biochemistry at 3 months revealed a normal ALP and a PTH of 20.9, but an elevated corrected Calcium of 2.87 mmol/l (2.10–2.60). She had no symptoms of hypercalcaemia. Ultrasound of the neck was unremarkable. Despite having stopped calcium and vitamin D supplementation for 2 years, her calcium level remains mildly elevated.

Conclusions: Initially it appears both patients have primary hyperparathyroidism unmasked by vitamin D supplementation however the clinical presentations are contrasting. The large adenoma and marked hypercalcaemia in the first case is more representative of hyperparathyroidism in the developing world. In this population there is a theorised progression from four gland hyperplasia to single gland adenoma formation as a consequence of prolonged severe vitamin D deficiency. It has been suggested that this type of hyperparathyroidism should be termed 'quaternary hyperparathyroidism'.

The second case is more typical of the presentation of primary hyperparathyroidism in western populations with mild hypercalcaemia in an older woman. The clinical features in this case suggest primary hyperparathyroidism that has been masked by vitamin D deficiency.

These two cases highlight the potential difficulties in treating severe vitamin D deficiency and the importance of monitoring calcium and parathyroid hormone levels. With interest in treating vitamin D deficiency growing we propose the inadvertent use of the calcium and vitamin D challenge test may uncover underlying hyperparathyroidism be it primary or quaternary.

Disclosure: All authors have declared no conflicts of interest.

OP60. SLE WITH C1Q DEFICIENCY TREATED WITH FRESH FROZEN PLASMA (FFP): A TEN-YEAR EXPERIENCE

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Background: We present the case of a 24 year old Pakistani female with systemic lupus erythematosus (SLE)-like illness secondary to homozygous complement C1q deficiency, who has been treated successfully with fresh frozen plasma (Octaplas®) therapy. She presented aged 6 years with cutaneous lupus and later developed Raynaud's, alopecia, oral ulceration and seizures secondary to cerebral lupus. She was poorly controlled on prednisolone and azathioprine and aged 15 years presented with fatigue, fever, mucosal ulcers, headaches, malar rash, weight loss, arthralgia, nail dystrophy and recurrent infections requiring prolonged antibiotics. Investigations revealed a normocytic anaemia, neutrophilic leucocytosis and a prominent acute phase response. Hereditary homozygous C1q deficiency is a rare, autosomal recessive condition associated with a severe SLE-like illness. Since she remained unwell despite immunosuppressants we attempted to restore complement activity. Most complement proteins are synthesised by the liver, but serum

C1q is predominantly produced from bone marrow-derived cells. Bone marrow transplantation could theoretically offer a permanent cure and has successfully reconstituted serum C1q levels in murine C1q deficiency. However, she was ineligible for an allograft due to incompatible HLA-matched sibling donors. Hence we elected to restore C1q activity using Octaplas®.
Methods: Complement activity was measured using standard assays and C1q quantified by ELISA.
Results: Total haemolytic complement activity was absent and serum C1q undetectable. Antinuclear antibodies were positive (1:640) but antibodies against dsDNA, extractable nuclear antigens, cardiolipin and C1q were absent. We demonstrated that Octaplas® contained normal C1q levels and she responded to three weekly infusions of Octaplas® (10ml/kg) such that by six months her symptoms had almost completely resolved. Attempts to increase the infusion interval to five weeks resulted in symptom recurrence. This patient has remained well with monthly Octaplas® infusions for the last decade, with only intermittent and mild hypersensitivity reactions during infusions. Development of anti-C1q antibodies is an important consideration with this therapy but to date she remains anti-C1q antibody negative. Octaplas® provided symptomatic relief for four weeks but restored normal complement haemolytic activity for less than 48 hours. Previous reports of plasma therapy in C2-deficient SLE patients suggested that temporary complement repletion results in significant clearance of circulating immune complexes. It is likely that a similar mechanism explains the therapeutic efficacy of Octaplas® in our case.
Conclusions: We conclude that long term Octaplas® therapy is an effective treatment option for SLE associated with C1q deficiency.
Disclosure: All authors have declared no conflicts of interest.

OP61. A CASE OF SJOGREN'S SYNDROME WITH RENAL TUBULAR ACIDOSIS AND CENTRAL PONTINE MYELINOLYSIS

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Background: Sjogren's syndrome (SS) is a chronic autoimmune disease characterized by lymphocytic infiltration classically presenting with keratoconjunctivitis sicca, xerostomia, parotid gland enlargement and arthralgia. However, virtually any organ can be affected. Renal involvement occurs in up to 60%, with distal renal tubular acidosis (dRTA) being the most common manifestation and CNS involvement in 25%.
Methods: We describe an unusual case of a 64 year old woman presenting with a 3 month history of fatigue and nausea, with unexplained hypercalcaemia (3.27 mmol/L) and hypokalemia (2.1 mmol/L). Background history included osteoporosis, depression and long-term smoking. Intravenous potassium, fluid therapy and Pamidronate successfully corrected her electrolyte imbalances and resolved her symptoms. Extensive investigations failed to identify an underlying pathology, with an appropriately suppressed PTH, normal thyroid USS, negative autoimmune profile and myeloma screen. MRI spine showed compression fractures consistent with osteoporosis. Two months later she represented in the same way. Hypercalcaemia was successfully corrected but the hypokalemia was refractory. Despite IV replacement her potassium fell (2.01 mmol/L) and she developed an acute generalised paralysis. She was also acidotic (pH 7.21) with an inappropriately alkali urine, (pH 7.2) and a diagnosis of type 1 RTA (dRTA) was made. She was transferred to ITU for IV bicarbonate. Her symptoms fully resolved with successful correction of her electrolyte and acid-base balance. Throughout her serum sodium was normal. Renal ultrasound showed nephrocalcinosis.

Two months later she developed a unilateral abducens nerve palsy progressing to affect both eyes, with xerostoma and xeropthalmia.
Results: Repeat autoimmune profile showed strongly positive RF 1:1280, ANA 1:320 and positive Ro-SSA. La-SSB, Sm, RNP, Jo-1, and SCL-70 were all negative. MRI brain showed central pontine myelinolysis (CPM).

A diagnosis of Sjogren's syndrome (SS) was made and both this and the dRTA successfully managed with Hydroxychloroquine and oral sodium bicarbonate. Her electrolytes have remained stable and she wears prism spectacles to correct her diplopia.
Conclusions: We postulate a case of Sjogren's Syndrome as a cause of renal tubular acidosis leading to severe electrolyte disturbance, nephrocalcinosis, and central pontine myelinolysis. Whilst the CPM may be secondary to the rapid correction of potassium, it could also be a direct consequence of SS. Patients presenting with hypokalemic paralysis secondary to dRTA before a diagnosis of SS is unusual but cases have been reported. CPM is a rare manifestation of SS with only two reported cases. CPM has been attributed in a few reported cases to rapid potassium shifts. The CPM may explain the abducens nerve palsy, but this may be a neurological sequelae of SS. Proposed mechanisms by which SS causes a dRTA and CPM will be discussed.
Disclosure: All authors have declared no conflicts of interest.

OP62. PERSISTANT PAIN AT THE 1ST CMC JOINT MISDIAGNOSED FOR YEARS

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Background: Pain at the 1st CMC joint, in elderly people, is most commonly caused by OA. The usual treatment is simple analgesia, Non-Steroidal Anti-Inflamatory Drugs and/or intra-articular steroid injection. We would like to report a rare cause of pain at this site which is usually curable.
Methods: Case report of 68 years old man who presented with painful paraesthesia at the dorsum of his left thumb for more than 6 months before referral to the hospital. He has long standing history of neck ache and stiffness otherwise quite well. He was initially diagnosed as C6 nerve root impingement as a result of MRI. Treatment with a diagnostic C6 nerve impingement, in the form of C6 nerve root block, was ineffective. As a result, the diagnosis was revised to thumb CMC joint OA. However, injection therapy failed again to produce any response. The patient developed a trigger point in the left triceps muscle which was imaged by MRI and was identified as a venous varicosity. Injection of the trigger spot failed to produce benifit and the patient remarked that,curiously, the thumb pain had temporarily worsened following the injection. Pain management team diagnosed a chronic regional pain syndrome. The patient was told that he has to learn to live with it. The pain worsened. Repeat MRI after two years confirmed a highly vascular growth at the radial groove which have enlarged over the course of more than two years.
Results: MRI of the left upper arm showing highly vascular tumour in the left radial groove with Gadolinium enhancement. The tumour was surgically removed and histopathology confirmed it to be a Scwannoma of the radial nerve. 80% of the symptoms disappeared within 3 months.The patient was completely symptom-free one year after surgury.
Conclusions: Pain at the 1st CMC joint in elderly is not always due to OA. The presence of a trigger point in the upper arm with worsening of the pain at the base of the thumb on pressing this point should alert the physician to the possible diagnosis of radial nerve tumour which can be cured by surgical resection.
Disclosure: All authors have declared no conflicts of interest.

Concurrent Oral 10 – Education Research

OP63. USE OF 'GALS' IN CLINICAL PRACTICE. WHY ARE WE STILL FAILING?

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Background: Musculoskeletal (M-S) disorders are one of the commonest causes of delayed discharge following hospital admission. Earlier studies from teaching hospitals have shown the lack of GALS screening and M-S examination clerking in patients admitted to hospital. We undertook a prospective study at two sites of our Trust and compared with a teaching hospital in an attempt to ascertain the use of GALS among the junior doctors and also examined clerking of M-S system in hospital case notes.
Methods: The study was undertaken for 3 months with review of 50 case notes from each hospital site (1 Non-Teaching & 1 Teaching) from 3 different wards of each site such as MAU, acute medical and surgical wards. The study also included 'face to face' interview with the junior doctors in charge of the patients, with a view to assess their confidence and explore their feelings with regard to GALS assessment. The wards and the days of the week for the case note review were chosen randomly at all sites The focus was on GALS screening questions besides documentation of previous M-S disease, history of falls, fractures and assessment of M-S examination. Targeted questionnaire were used for the face-to-face interview of the junior doctors as in previous studies.
Results: Of the 50 case notes from each centre, 25 were from MAU, 15 and 10 from acute medical and surgical wards respectively. Fifty five percent were males and 45% were female. The mean age 67.6 years; range 18-98 yrs. Only 12% patients had a working diagnosis of M-S disorder on admission. Only 6 of the 150 (4%) patients were asked the 3 GALS questionnaire. Review of clerking showed that only 16% patients had documentation of M-S examination despite 42% had reported history of M-S disease with disability. One third of patients had recurrent falls and 15% had fractures. Overall 16% patients had M-S examination in MAU, 22% and 10% in acute medical and surgical ward. Thirty junior doctors were interviewed across 3 sites and only 3 had screened for M-S disease. Only 4 (13%) felt confident to do M-S examination. There was no difference in attitudes in doctors between teaching and non teaching hospitals.
Conclusions: This prospective study reaffirms that despite years after original reporting, paucity of GALS and M-S examination continues in hospital inpatients and little progress has been made in real practice. Despite a large number of patients being admitted with M-S symptoms very few doctors practise what is taught at undergraduate level. The lack of confidence amongst the junior doctors needs to be addressed and there is an impending need to ensure that all clinical assessments in A&E and other clinical areas have a schedule for GALS and M-S examination. Only then rheumatologists may achieve the ultimate goal of teaching the M-S examination at undergraduate level.
Disclosure: All authors have declared no conflicts of interest.

OP64. AN EVIDENCE AND PRACTICE BASED REGIONAL MUSCULOSKELETAL EXAMINATION FOR SCHOOL-AGED CHILDREN - PREMS

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