### CME Rheumatological and immunological disorders - II

- *Inhibitors*. 7th International Congress. NEGMA Laboratoires 2000:22 Abstract.
- 15 Reginster J-Y, Deroisy R, Paul I, Lee RL, et al. Glucosamine sulfate significantly reduces progression of knee osteoarthritis over 3 years: a large, randomized, placebocontrolled, double-blind, prospective trial. Arthritis Rheum 1999;4 (Suppl 9):S400.
- 16 Pavelka K, Gatterova J, Olejarova M, Machacek S, et al. A long-term randomized, placebo-controlled, confirmatory trial on the effects of glucosamine sulfate on knee osteoarthritis progression. Osteoarthritis Cartilage 2000;8(Suppl):S6–7.
- 17 Mazzuca S, Brandt KD. Plain radiography as an outcome measure in clinical trials of OA. Rheum Dis Clin North Am 1999;25: 467–80
- 18 Buckland-Wright JC. Quantitation of radiographic changes. In: Brandt KB, Doherty M, Lohmander LS (eds). Osteoarthritis. Oxford: Oxford University Press, 1998: 459–72.
- 19 Buckland-Wright JC, Wolfe F, Ward RJ, Flowers N, Hayne C. Substantial superiority of semiflexed (MTP) views in knee osteoarthritis: a comparative radiographic study, without fluoroscopy, of standing extended, semiflexed AP, and schuss views. J Rheumatol 1999;26:2664–74.
- 20 Peterfy CG, Li J, Duryea J, Lynch JA, et al. Nonfluoroscopic method for flexed radiography of the knee that allows reproducible joint space width measurement. Arthritis Rheum 1998;41(Suppl 9):S361.
- 21 Dieppe PA, Cushnaghan J, Shepston L. The Bristol 'OA 500' study: progression of osteoarthritis (OA) over 3 years and the relationship between clinical and

- radiographic changes at the knee joint. *Osteoarthritis Cartilage* 1997;**5**:87–97.
- 22 Brandt KD. The role of analgesics in the management of osteoarthritis pain. Am J Ther 2000;7:75–90.
- 23 Whelton A. Renal and related cardiovascular effects of conventional and Cox-2specific NSAIDs and non-NSAID analgesics. Am J Ther 2000;7:63–74.
- 24 Levinson DJ, Rubinstein HM. Double-blind comparison of fenoprofen calcium and ibuprofen in osteoarthritis of large joints. Curr Ther Res 1983;34:280–4.
- 25 Tyson VC, Glynne A. A comparative study of benoxaprofen and ibuprofen in osteoarthritis in general practice. *J Rheumatol* 1980;**6**(Suppl):132–8.
- 26 Lister BJ, Poland M, DeLapp RE. Efficacy of nabumetone versus diclofenac, naproxen, ibuprofen, and piroxicam in osteoarthritis and rheumatoid arthritis. Am J Med 1993;95:28–9S.
- 27 Kvien TK, Brors O, Staff PH, Rognstad S, Nordby J. Improved cost-effectiveness ratio with a patient self-adjusted naproxen dosing regimen in osteoarthritis treatment. *Scand J Rheumatol* 1991;**20**:280–7.
- 28 Scholes D, Stergachis A, Penna PM, Normand EH, Hansten PD. Nonsteroidal anti-inflammatory drug discontinuation in patients with osteoarthritis. *J Rheumatol* 1995;**22**:708–12.

Address for correspondence: Dr Kenneth D Brandt, Rheumatology Division, Indiana University School of Medicine, USA

## SELF-ASSESSMENT QUESTIONNAIRE

# Rheumatological & immunological disorders

- Twenty self-assessment questions (SAQs) based on the published articles will appear at the end of each CME specialty featured in *Clinical Medicine*. The questions have been validated for the purpose of CME by independent experts. Three (3) CME credits will be awarded to those achieving 80% correct answers. This opportunity is open only to RCP Fellows and Collegiate Members in the UK who are registered for CME\*.
- A loose leaf answer sheet is enclosed, which will be marked electronically at the Royal College of Physicians. Answer sheets must be returned by 21 May 2001 to:

CME Department (SAQs), Royal College of Physicians, 11 St Andrews Place, London NW1 4LE.

Correct answers will be published in the next issue of *Clinical Medicine*.

\* Further details on CME are available from the CME department at the Royal College of Physicians (address above or telephone 020 7935 1174 extension 306 or 309).

### Guidelines on completing the answer sheet

Your completed answer sheet will be scanned to enable a quick and accurate analysis of results. To aid this process, please keep the following in mind:

- 1 Please print your GMC Number firmly and neatly
- 2 Only write in allocated areas on the form
- 3 Only use pens with black or dark blue ink
- 4 For optimum accuracy, ensure printed numbers avoid contact with box edges
- 5 Please shade circles like this: Not like this: •
- 6 Please mark any mistakes made like this:
- 7 Please do not mark any of the black squares on the corners of each page
- 8 Please fill in your full name and address on the back of the answer sheet in the space provided; this will be used to mail the form back to you after marking.

### Q1 Cytoplasmic anti-neutrophil antibodies (c-ANCA) in vasculitis:

- a) Are a sensitive marker for Wegener's
- b) Are directed against myeloperoxidase (MPO)
- c) In a rising titre always indicates a need to increase immunosuppressive therapy
- d) Are found in >75% of classical PAN patients
- e) Have a role in endothelial cell activation

### Q2 In the treatment of systemic vasculitis:

- a) Cyclophosphamide should be maintained for at least one year after remission induction
- b) The risk of haemorrhagic cystitis increases with the cumulative dose of cyclophosphamide received
- Eradication of nasal staphylococcus aureus decreases the relapse rate in Wegener's
- d) Plasma exchange is helpful in Wegener's where there is rapidly progressive renal failure
- e) The is still a 20% five year mortality in systemic necrotizing vasculitis

### Q3 Marfan syndrome:

- a) Is always associated with mutilations in the fibrillin gene
- b) Can be diagnosed by echocardiography alone
- c) Is associated with a family history 30% of cases
- d) Can diagnoses by fibrillin analysis
- e) Typically causes dural ectasia

### Q4 Skeletal dysplasias:

- a) Are not commonly genetic in origin
- b) Are usually caused by recessive mutations
- c) May cause rhizomelic disproportion
- May be caused by fibroblast growth factor receptor mutations

e) May be caused by Type V collagen defects

### Q5 Congestive heart failure in rheumatoid arthritis:

- a) Is most commonly due to myocarditis
- b) Echocardiography is seldom useful
- c) Never responds to steroids
- d) Is an indication for angiotensin converting enzyme inhibitor therapy
- e) Can occur with normal left ventricular systolic function

### Q6 Cardiovascular mortality in rheumatoid arthritis:

- a) Accounts for 10% of all deaths in RA
- b) Is predominantly due to rheumatoid heart disease
- c) Is the same as in the general population
- d) May associate with the severity of RA
- e) May be due to heart failure

### Q7 Systemic Lupus Erythematosus disease in humans:

- a) Is mostly genetically determined
- b) Is likely to relapse after infection
- c) Is associated with a reduced rate of apoptosis
- d) Is more common in people originating from the Caribbean
- e) May be associated with false positive tests for syphyllis

### Q8 Autoantibodies in Systemic Lupus Erythematosus:

- a) Only target nuclear antigens
- b) May cause abnormal clotting tests
- c) Are usually of IgM type
- d) Reflect a failure of immune tolerance
- e) Often include anti-smooth muscle antibodies

### Q9 The following are usually associated with anti-cardiolipin antibodies:

- a) Fetal loss
- b) Thrombocytopenia
- c) Erosive arthritis
- d) Venous and arterial thrombosis
- e) Coomb's test positive hemolytic anemia

### Q10 The following are the recommended treatment of recurrent DVT in the Antiphospholipid Syndrome:

- a) Subcutaneous heparin for three months
- Prednisone followed by cyclophosphamide for six months
- c) Warfarin for six months aiming for an INR of 1.8
- Heparin followed by warfarin indefinitely aiming for an INR of 3 to 3.5
- e) IV heparin, IV corticosteroid, IV cyclophosphamide and plasmapheresis

#### Q11 In reactive arthritis:

- a) Organisms cannot be cultured from affected joints
- b) Long term antibiotics alter the course of disease
- c) HLA B27 influences disease severity
- Enteric infection preceding arthritis is always symptomatic
- e) Symmetrical small joint involvement is common

### CME Rheumatological and immunological disorders - II

### Q12 In ankylosing spondylitis:

- More than 90% of patients with classical disease are B27+
- All B27+ individuals are equally at risk of developing the disease
- B27 restricted CD8+ T cells have been shown to play a critical role in pathogenesis
- B27 positive transgenic rats only develop disease if gut flora are present
- e) Patients have elevated titres of antibodies to Klebsiella

### Q13 In spondyloarthropathy:

- a) Uveitis can complicate all forms
- b) Grade 3 sacroilitis is always present
- c) The arthritis is resistant to anti-TNF therapies
- d) A diagnosis can be made on the basis of inflammatory spinal pain and alternating buttock pain
- e) Inflammatory bowel disease may be clinically silent

### Q14 Concerning structure-modifying drugs:

- Structure-modifying drugs for OA have been shown not only to be 'chondroprotective', but to have favourable effects on joint pain
- We do not know if structure-modifying drugs have a beneficial effect on OA pain
- c) Structure-modifying drugs have been shown to exacerbate joint pain in humans with OA
- d) Structure-modifying drugs which result in a reduction in osteophyte size are accompanied by a decrease in joint pain, but those which retard joint space narrowing are not
- e) Because they permit an increase in load bearing on the OA joint, structure-modifying drugs are associated with an increase in joint pain

### Q15 In osteoarthritis:

- a) Conventional radiography (eg, a standing anteroposterior knee radiograph) is the preferred outcome measure for evaluating structure-modifying drugs in OA clinical trials
- Ultrasonography and magnetic resonance imaging are validated outcome measures for clinical trials of structure-modifying drugs in OA
- c) An increase in knee flexion, as may occur with an increase in joint pain, may result in a decrease in the width of the medial tibiofemoral compartment joint space in a standing anterposterior knee radiograph
- d) Fluoroscopic alignment of the knee joint with the X-ray beam results in poorer standardization of radiographic positioning than conventional radiography
- e) It has been shown that progressive narrowing of the joint space in knee radiographs of patients with OA predicts progressive increase in joint pain and disability

### Q16 Concerning doxycycline:

- Doxycycline therapy has been shown to accelerate the progression of articular cartilage damage in OA
- b) The putative benefits of doxycycline in OA have been attributed to its antimicrobical effects on Chlamydia
- Doxycycline stimulates the transcription of mRNA involved in synthesis of cartilage matrix metalloproteinases, such as collagenase
- d) Doxycycline stimulates the synthesis of inducible nitric oxide synthase, enhancing blood flow to the OA joint and thereby facilitating cartilage repair
- Protective effects of doxycycline in animal models of OA provide no assurance that it will have a similar effect in humans

### Q17 In the context of primary systemic vasculitic syndromes:

- a) ANCA assays have equal sensitivity in limited disease and systemic disease involving the lung/kidney
- Two major staining patterns can be identified on indirect immunofluorescence – cytoplasmic and peri-nuclear which respectively correspond to myeloperoxidase and proteinase 3 specificities using solid phase ELISA assays
- c) A cANCA staining pattern is observed most frequently in Wegener's Granulomatosis
- Acute respiratory failure with diffuse bilateral alveolar infiltrates is almost always due to pulmonary haemorrhage
- e) A high CRP is strongly suggestive of infection

### Q18 In Systemic Lupus Erythematosus:

- a) Fever is almost always associated with a raised CRP
- b) Rigors in a patient with active disease most commonly indicates infection
- Bronchoscopy and bronchoalveolar lavage may help to distinguish pulmonary haemorrhage from opportunistic pulmonary infection
- Raised complement levels and high titres of anti ds DNA antibodies correlate with active renal disease
- e) There is an increased susceptibility to disseminated neisserial and salmonella infection

### Q19 TNF blockade works by:

- a) Binding and inactivation of soluble TNF
- b) Binding and inactivation of membrane bound TNF
- c) Cytotoxicity of TNF producing cells
- d) Inhibition of cell migration
- e) Inhibition of neovascularisation

#### Q20 TNF blockade is contraindicated in case of:

- a) Active bacterial infection
- b) Viral infection
- c) History of malignancy
- d) Pregnancy
- Allergic reaction to mouse murine antigen-binding part of monoclonal antibodies