DOWNLOAD PDF THE YEAR IN RHEUMATIC DISORDERS VOLUME 4

Chapter 1 : Cardiovascular co-morbidity in rheumatic diseases â€" Mayo Clinic

This is inevitably , rather than , and in possible recognition of this the book is entitled 'The Year in Rheumatic Disorders, Volume 4' rather than ' ' unlike previous volumes in the series.

Diseases of these valves are more prevalent than disease of the pulmonary or tricuspid valve due to the higher pressures the left heart experiences. It is typically the result of aging, occurring in Aortic insufficiency, or regurgitation, is characterized by an inability of the valve leaflets to appropriately close at end systole , thus allowing blood to flow inappropriately backwards into the left ventricle. Causes of aortic insufficiency in the majority of cases are unknown, or idiopathic. Processes that lead to aortic insufficiency usually involve dilation of the valve annulus , thus displacing the valve leaflets, which are anchored in the annulus. Mitral stenosis is caused largely by rheumatic heart disease , though is rarely the result of calcification. In some cases vegetations form on the mitral leaflets as a result of endocarditis, an inflammation of the heart tissue. Mitral stenosis is uncommon and not as age dependent as other types of valvular disease. In these cases the left ventricle of the heart becomes enlarged and causes displacement of the attached papillary muscles , which control the mitral valve. Mitral insufficiency is significantly associated with normal aging, rising in prevalence with age. Pulmonary valve diseases are the least common heart valve disease in adults. Unless the degree of stenosis is severe individuals with pulmonary stenosis usually have excellent outcomes and treatment options. Often patients do not require intervention until later in adulthood as a consequence of calcification that occurs with aging. Pulmonary valve insufficiency occurs commonly in healthy individuals to a very mild extent and does not require intervention. Additionally, insufficiency may be the result of carcinoid syndrome , inflammatory processes such a rheumatoid disease or endocarditis, or congenital malformations. Tricuspid valve stenosis without co-occurrent regurgitation is highly uncommon and typically the result of rheumatic disease. It may also be the result of congenital abnormalities, carcinoid syndrome, obstructive right atrial tumors typically lipomas or myxomas , or hypereosinophilic syndromes. Minor tricuspid insufficiency is common in healthy individuals. Dysplasia[edit] Heart valve dysplasia is an error in the development of any of the heart valves, and a common cause of congenital heart defects in humans as well as animals; tetralogy of Fallot is a congenital heart defect with four abnormalities, one of which is stenosis of the pulmonary valve. Certain medications have been associated with valvular heart disease, most prominently ergotamine derivatives pergolide and cabergoline. Damage to the heart valves follows infection with beta-hemolytic bacteria, such as typically of the respiratory tract. Pathogenesis is dependent on cross reaction of M proteins produced by bacteria with the myocardium. Involvement of other heart valves without damage to the mitral are exceedingly rare. Many developing countries, as well as indigenous populations within developed countries, still carry a significant burden of rheumatic fever and rheumatic heart disease and there has been a resurgence in efforts to eradicate the diseases in these populations. In pregnancy[edit] The evaluation of individuals with valvular heart disease who are or wish to become pregnant is a difficult issue. Issues that have to be addressed include the risks during pregnancy to the mother and the developing fetus by the presence of maternal valvular heart disease as an intercurrent disease in pregnancy. Comparison[edit] The following table includes the main types of valvular stenosis and regurgitation. Major types of valvular heart disease not included in the table include mitral valve prolapse , rheumatic heart disease and endocarditis.

Chapter 2 : Clinical Publishing

volume, devoted to an in-depth description of the major rheumatic diseases ranging from epidemiology, pathophysiology and clinical presentation to treatment modalities.

Highlight and copy the desired format. Emerging Infectious Diseases, 15 6 , Abstract We report 2 cases of leishmaniasis in patients with autoimmune rheumatic diseases in Greece. To assess trends in leishmaniasis reporting in this patient population, we searched the literature for similar reports from Europe. Reports increased during â€", especially for patients treated with antiâ€"tumor necrosis factor agents. We report 2 new cases of leishmaniasis involving patients with autoimmune rheumatic diseases who received antiâ€"tumor necrosis factor anti-TNF agents. We also reviewed all similar cases from Europe reported in the literature, and we discuss the implications of leishmaniasis in the setting of anti-TNF therapy, which is associated with increased risk for opportunistic infections 1. The Study Patient 1, a year-old man who had received a diagnosis of ankylosing spondylitis 7 years previously, was admitted to Laikon Hosptal, Athens, Greece, in May for evaluation of encrusted vesicular lesions on the face. The lesions were painless but mildly pruritic. He was living in a leishmaniasis-endemic area in Athens, had no pets in his house, and had no history of recent travel abroad. The central scale was removed from one of the lesions, and scrapings from the base of the lesion were stained with Giemsa stain, which showed intracellular amastigotes with peripheral nuclei and rod-shaped kinetoplasts. Results of indirect immunoflorescent antibody IFA testing were positive for Leishmania parasites titer 6, Patient 2, a year-old woman who had giant cell arteritis, was admitted to the Euroclinic Hospital, Athens, in May with a high fever and fatigue. The patient had been treated with infliximab 0. She was also living in an Athens suburb, which is leishmaniasis-endemic, and had 4 dogs. The examination of Giemsa-stained smears from bone marrow aspirate demonstrated abundant Leishmania parasites, and IFA was marginally positive for Leishmania antibodies titer PCR was positive for the detection of the Leishmania genome in peripheral blood. Two days later, the fever subsided, and within the next few days, the patient recovered from pancytopenia, while the inflammatory markers showed a gradual decrease. We searched the reference list of each resulting report for additional publications. We used no language or time restrictions. Reported cases of leishmaniasis in patients with autoimmune rheumatic diseases in Europe, indicated by stars 1 case from Israel not shown. Dark gray shading, distribution of leishmaniasis; light gray shading, distribution All retrieved articles were case reports. We found 13 additional cases of leishmaniasis in patients with autoimmune rheumatic diseases 2 â€" 14 , all published after the introduction of anti-TNF agents in Table. Two of the 15 reported patients had been treated with a recombinant interleukinâ€"1 receptor antagonist anakinra; Amgen Inc. In most of the patients, visceral leishmaniasis developed 13 patients, All patients were living in leishmaniasis-endemic areas of Europe Figure. The anti-TNF agents were introduced into clinical practice in , and the first case of leishmaniasis associated with anti-TNF blockade occurred in 4. During the 6-year period â€" , a total of 6 reports were made of leishmaniasis in patients with rheumatic diseases; 1 During the ensuing 5 years â€" , 9 cases of leishmaniasis were reported, 6 For the 7 patients who received anti-TNF agents, the median duration of anti-TNF treatment was 18 months range 9â€"60 months. Six of these 7 patients were receiving anti-TNF agents when symptoms and signs of leishmaniasis occurred. In 1 patient 5 , biologic treatments had been discontinued 6 months before the diagnosis of leishmaniasis Table. Only 1 patient had been tested for antibodies against Leishmania spp. Therefore, this is the only case with compelling evidence that leishmaniasis was a primary infection and not reactivation of a latent infection. Conclusions Our data suggest that the introduction of TNF blockade into the clinical practice is associated with increasing reports of leishmaniasis in patients with autoimmune rheumatic diseases who live in leishmaniasis-endemic areas of Europe. Notably, in most reported cases, patients had not received anti-TNF agents but other immunosuppressants. This increase coincides with the increasing use of anti-TNF agents during the same period, as prescription practice started changing toward treating patients with lower disease activity Another indirect piece of evidence that TNF blockade may increase the risk for leishmaniasis is that the median duration of previous anti-TNF treatment before the diagnosis of leishmaniasis was significantly shorter than

the median duration of immunosuppressive therapy for all 15 patients 18 vs. Our report has limitations. It is unclear for all cases with 1 exception presented in this article whether leishmaniasis was primary infection or reactivation of latent disease. We cannot also exclude the possibility that the concomitant, long-term use of other immunosuppressants, and not the anti-TNF agents per se, played a crucial role in the development of leishmaniasis. Different prescribing patterns of anti-TNF agents might influence the number of cases reported from each disease-endemic European country. However, the small number of reported cases and the lack of data on differences in the anti-TNF prescribing policies do not allow any conclusions to be reached. Finally, due to underreporting, the reported cases may underestimate the real incidence of leishmaniasis among patients with autoimmune rheumatic diseases. Prospective studies to estimate the incidence of the disease, the impact of risk factors and the need for serologic screening for leishmaniasis before initiation of anti-TNF agents or any other immunosuppressive treatment are clearly needed. This is particularly important since currently only a few patients with autoimmune rheumatic diseases receive anti-TNF agents Therefore, the use of anti-TNF treatment is likely going to increase, possibly causing a parallel increase in opportunistic infections such as leishmaniasis. His research interests include the study of infections in patients with autoimmune rheumatic diseases. Risk and prevention of tuberculosis and other serious opportunistic infections associated with tumor necrosis factor. Nat Clin Pract Rheumatol. Visceral leishmaniasis related to infliximab administration. Enferm Infecc Microbiol Clin. Visceral leishmaniasis infection in a rheumatoid arthritis patient treated with infliximab. Visceral leishmaniasis infection in a rheumatoid arthritis patient treated with adalimumab. Visceral leishmaniasis infection in a patient with rheumatoid arthritis treated with etanercept. Visceral leishmaniasis in a patient with psoriatic arthritis treated with infliximab: Visceral leishmaniasis associated with Wegener disease. Use of lipid complex amphotericin B and liposomal amphotericin B. Relapse of cutaneous leishmaniasis in a patient with an infected subcutaneous rheumatoid nodule. Enfermedad oportunista poco frecuente en enfermo tratamiento immunosupresor por arthritis reumatoide. Anales de Medicina Interna Madrid. Visceral leishmaniasis in a patient with systemic juvenile arthritis treated by IL-1RA agonist anakinra. Visceral leishmaniasis in a rheumatoid arthritis patient treated with methotrexate. Int J Infect Dis. US perspectives on indications and monitoring.

Chapter 3 : Valvular heart disease - Wikipedia

The Year in Rheumatic Disorders Volume 4. CRC Press. Used - Good. Shows some signs of wear, and may have some markings on the inside.

Arachnoiditis, Pituitary Adenoma Opioid Withdrawal: Koster, MD and Kenneth J. Warrington, MD Page 1 of 3 Polymyalgia rheumatica PMR is a chronic inflammatory condition that predominantly involves large joints and periarticular structures. Additionally, women are affected 1. Although the etiology and pathogenesis of PMR are not known, epidemiologic studies suggest that there are both genetic and environmental factors involved in the development of this disease. This is further evidenced by a geographic variance in the incidence of PMR, with the highest rates seen among inhabitants of and descendants from Northern European countries. Stiffness typically lasts greater than 30 minutes and is worse after rest or inactivity. Patients often describe difficulty getting dressed or discomfort when turning in bed at night that interferes with sleep. Shoulder range of motion may be limited, causing difficulty in performing activities at or above head level. Compared to symptoms of non-inflammatory conditions, symptoms of PMR typically are symmetrical and get better with activity. In clinical practice, it can be challenging to distinguish PMR from elderly-onset rheumatoid arthritis. Indeed, inflammatory arthritis of peripheral joints eg, wrists, knees can be seen in up to one-third of patients with PMR. Similarly, diffuse muscle tenderness is not a prominent feature and should raise suspicion for electrolyte abnormalities, metabolic disturbances, or fibromyalgia. It is well recognized that PMR is associated with giant cell arteritis GCA , a systemic vasculitis affecting the large arteries that can cause blindness, stroke, and aortic aneurysm. Therefore, when evaluating patients for PMR, health care providers also should evaluate for symptoms of GCAâ€”headache, scalp tenderness, jaw claudication, diplopia, vision loss, and upper or lower extremity claudication. If any symptoms are present, testing for GCA should be undertaken, including the consideration of temporal artery biopsy. Making the Diagnosis Because of the broad differential diagnosis and potential mimicking disease states Table 2 , clinicians evaluating patients for PMR should obtain a detailed history and conduct a comprehensive physical examination, focusing particularly on the musculoskeletal, vascular, and neurologic systems. Laboratory parameters in PMR are not specific but typically show evidence of a systemic inflammatory state. Such abnormalities can include a mild normocytic anemia due to chronic inflammation, leukocytosis, and thrombocytosis. Additional laboratory studies that are useful in the differential diagnosis include liver transaminases, creatine kinase, calcium, sodium, potassium, magnesium, creatinine, thyroid-stimulating hormone, serum protein electrophoresis, and urinalysis with microscopy. Several sets of diagnostic criteria for PMR have been proposed, but they have not been validated or universally accepted. Common features among these criteria have included a minimum age 50â€”65 years , bilateral shoulder girdle and hip girdle aching, morning stiffness, and elevated inflammatory markers. Role of Imaging In recent years, there has been an increased use of imaging modalities to evaluate patients with suspected PMR. Ultrasonography and magnetic resonance imaging MRI commonly have identified abnormalities in large joints and periarticular structures. Although these findings also can be seen in other forms of inflammatory arthritis, identification of these abnormalities can be helpful to differentiate PMR from other conditions. The lifetime risk of adult-onset rheumatoid arthritis and other inflammatory autoimmune rheumatic diseases. Epidemiology of giant cell arteritis and polymyalgia rheumatica. Advances and challenges in the diagnosis and treatment of polymyalgia rheumatica. Ther Adv Musculoskelet Dis. Polymyalgia rheumatica and giant-cell arteritis. Polymyalgia rheumatica with low erythrocyte sedimentation rate at diagnosis. Polymyalgia rheumatica without significantly increased erythrocyte sedimentation rate. A more benign syndrome. Erythrocyte sedimentation rate and C-reactive protein in the evaluation of disease activity and severity in polymyalgia rheumatica: Repetitive fluorodeoxyglucose positron emission tomography in isolated polymyalgia rheumatica: Ultrasound of proximal upper extremity arteries to increase the diagnostic yield in large-vessel giant cell arteritis. Incidence of temporal arteritis in patients with polymyalgia rheumatica: What is the best approach to diagnosing large-vessel vasculitis? Best Pract Res Clin Rheumatol. Clinical outcomes, quality of life, and diagnostic uncertainty in the first year of polymyalgia rheumatica.

Prednisone plus methotrexate for polymyalgia rheumatica: Can methotrexate be used as a steroid sparing agent in the treatment of polymyalgia rheumatica and giant cell arteritis? Long-term follow-up of polymyalgia rheumatica patients treated with methotrexate and steroids. Infliximab plus prednisone or placebo for the initial treatment of polymyalgia rheumatica: September 27, 1.

Chapter 4 : Latest Advances In the Diagnosis And Treatment Of Polymyalgia Rheumatica

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This is an open access article distributed under the Creative Commons Attribution License , which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. Abstract The age at onset refers to the time period at which an individual experiences the first symptoms of a disease. In autoimmune diseases ADs , these symptoms can be subtle but are very relevant for diagnosis. They can appear during childhood, adulthood or late in life and may vary depending on the age at onset. Early age at onset is a worst prognostic factor for some ADs i. The age at onset varies widely depending on the disease. For example, sixty-five percent of patients with systemic lupus erythematosus SLE start manifesting their symptoms between ages 16 and 55 [3]. Another 20 percent manifest them before age 16 and the remaining 15 percent after age 55 [4]. Rheumatoid arthritis RA can begin at any age but has its peak between ages 30 and 55 [5]. Juvenile idiopathic arthritis JIA is a term used to describe the autoimmune, inflammatory joint condition that develops in children. Multiple Sclerosis MS usually appears between ages 20 and 40, and it is very rare during adolescence [7]. Type 1 diabetes mellitus T1D is considered a childhood and adolescent disease with two peaks of onset, one between ages 5 and 9 and a second between ages 10 and 14 [8]. On the other hand, an adult onset would be considered to be in a range of 25â€”61 years old [9]. Finally, autoimmune thyroiditis AITD is thought to be a disease that can appear in childhood but is more prevalent during adulthood [10]. Herein, we analytically reviewed the effect of age at onset on the most prevalent ADs, their clinical differences Table 1 , and their genetic and immunologic relationships Table 2. Clinical differences between early and adult onset. Genetic and immunological factors related to age at onset. It is more common in adults, but it may be diagnosed during childhood as well [11]. Childhood disease onset is characterized by a high degree of morbidity compared with adult SLE populations [12]. The patients have a higher frequency of renal disease, malar rashes, pericarditis, hepatosplenomegaly, and hematologic alterations [13]. In treatment, they usually have a higher use of prednisone and the need for additional immunosuppressive therapies [14]. Patients at this age are susceptible to a longer lifetime of damage from the disease flares and the treatment side effects and a mortality rate that is 2 times higher [11 , 12]. Other associations found in childhood onset are growth failure, delayed puberty, and fibromyalgia. In contrast, adult onset patients are more likely to develop pulmonary disease [11] and may have an increased rate of simultaneously developing another AD such as SS [15]. Additionally, Webb et al. In both cases, better disease outcome can be achieved if an early diagnosis is made by a better recognition of age-specific manifestations and the use of a good treatment algorithm [15]. As other ADs, SLE is a complex disease in which several polymorphic genes and environmental factors over time influence the onset and course of disease. No association between the number of SLE risk alleles and age at onset in Hispanic patients and European American was found [16]. Rheumatoid Arthritis and Juvenile Idiopathic Arthritis RA is a chronic, systemic, and destructive inflammatory AD that involves both small and large diarthrodial joints. It usually develops in middle-aged adults but may also appear during childhood or late in life [32]. Patients who are diagnosed between ages 16 and 65 are considered young onset and after 65, late onset with each of them having different semiologic characteristics. In contrast, Deal et al. It has been reported that older patients have more acute onset in both large specially the shoulders and small joints and usually present polymyalgia rheumatica-like symptoms [20]. Classical hand deformities, interstitial lung disease, and SS presented less commonly at late onset. However, these patients can present more constitutional features like weight loss, myalgia, rheumatic nodules, and neuropathy [18]. Anti-CCP seropositivity and elevated inflammatory markers at onset are associated with poor radiological outcome in both early and late onset [33 , 34]. Studies differ when talking about prognosis and report favorable, similar, or worse outcome. Most of them conclude that treatment should be instituted equally on both. Juvenile idiopathic arthritis JIA is a term that describes a group of disorders that share the clinical manifestation of chronic joint inflammation from unknown causes

that begins before 16 years of age [21]. PJIA is defined by the presence of more than four affected joints during the first six months of illness. There is a bimodal distribution of the age at onset: Children with polyarticular disease with onset after 10 years of age are divided into negative rheumatoid factor RF polyarthritis and positive RF polyarthritis. Positive RF polyarthritis tends to be associated with anti-CCP antibodies and a more severe disease than their adult counterpart [36]. They usually have a rapid onset of inflammation in multiple joints, especially hands, wrists, elbows, and feet. A big difference between it and the adult form of the disease is the effect of the disease on their growing skeleton that can lead to growth retardation or accelerated growth of an affected joint [21]. Anti-CCP antibodies can be detected at early disease stages and may be used as indicators of RA progression and prognosis [38]. This finding may be useful to predict early RA onset in genetically predisposed patients [39]. It is very rare during childhood [46]. In contrast, Nakamura et al. However, this findings warrant further replication and confirmation. No age at onset relationship was found. B-cell damage may be induced at any age [50]. Childhood- and late-onset patients are characterized by symptoms like polydipsia, polyuria, and weight loss, but younger patients suffer more from diabetic ketoacidosis and ketosis as the initial presentation [24 , 27]. Studies indicate that late-onset patients have better preserved B-cell function than early-onset patients. They are also characterized by a longer symptomatic period before diagnosis and a lower frequency of insulin autoantibodies and HLA class II susceptibility alleles [25 , 26]. Adult onset can be associated with milder signs of metabolic decompensation and a lower glycated hemoglobin level at diagnosis [27 , 28]. This polymorphism could be associated with development of T1D at an early age [42]. Multiple Sclerosis Age at onset of MS as in most of ADs is defined as the age when the first symptoms appear, although the disease process may have begun earlier [52]. Studies are consistent with the idea that, in early onset, mild and severe disability levels are reached after a longer time than in the case of adult onset. However, these disability levels are also reached at a lower age in comparison with adult cases. This can be translated into more disability in early-onset patients than their corresponding adult counterpart at the same age. Bad prognostic factors for a worse outcome include disability in the first year, high relapse rate, short interattack intervals, the relapsing progressive course, or a shift of the progressive phase [7 , 54 â€” 56]. Associated HLA-class alleles of the disease are also identified. Autoimmune Thyroiditis AITD is an inflammatory state of the thyroid gland that results from interaction between genetic and environmental factors. Hashimoto thyroiditis is the most frequent form of chronic autoimmune thyroiditis. This is the most common cause of hypothyroidism in children [59]. Some of its manifestations at this age are effects on growth, bad school performance, bradykinesia, and delayed pubertal development. A goiter is the main symptom that indicates AITD. Patients that receive proper treatment at this age will probably experience normal growth and puberty [30 , 31]. Both young and adult onset patients may show symptoms of lethargy, intolerance to cold, constipation, dry skin, brittle hair, and muscle pain. Late-onset hypothyroidism, once it begins, is permanent but, in some young onset patients and postpartum women, it is often transient [10]. A good example is T1D. That is why they have to be screened annually. Conclusions Age at onset varies among ADs and so do their manifestations. Some of them, for example, MS and SS, are rare during childhood but others such as T1D primarily occur during this period. Early age at onset cannot always be associated with a worse prognosis. Knowledge of the early-age symptoms will help physicians to provide better treatment which, coupled with educational and transition support, might improve outcome. Understanding of genetic influences and association studies between diseases are required to determine the role of genes in age at onset. Studies with a larger number of people with nontypical ages at onset would bring further insights. View at Google Scholar H. View at Google Scholar A. View at Google Scholar M. View at Google Scholar S. View at Google Scholar E. View at Google Scholar N. View at Google Scholar J. View at Google Scholar K. View at Google Scholar C. View at Google Scholar P. View at Google Scholar B. View at Google Scholar W. View at Google Scholar Follow Us.

Chapter 5 : Epidemiology of the rheumatic diseases â€" University of Arizona

The Year in Rheumatic Disorders, Volume 6 by Rajan Madhok (Editor), Hilary A Capell (Editor), Harvinder Luthra (Editor) starting at. The Year in Rheumatic Disorders, Volume 6 has 0 available edition to buy at Alibris.

Advanced Search Abstract Patients who have chronic rheumatic or autoimmune diseases, such as rheumatoid arthritis, systemic lupus erythematosus, or vasculitides, show a risk of infection that is at least 2-fold greater than that for healthy individuals. This increased risk is not only a result of the aberrant immunologic reaction itself but also can be attributed to the immunosuppressive therapy required to control the activity of the underlying disease and the associated organ complications. Vaccination is an option for a substantial number of these infections. In this context, pneumococcal and influenza vaccines are the best evaluated and are recommended by standard vaccination guidelines. Some studies have found mildly impaired immune responses to vaccines among patients receiving long-term immunosuppressive therapy, but postvaccination antibody titers are usually sufficient to provide protection for the majority of immunized individuals. The accumulated data on the safety and effectiveness of vaccines warrant immunization with the majority of vaccines for patients with chronic autoimmune or rheumatic diseases, especially vaccination against influenza and pneumococci. Vaccination protocols for this population should be better implemented in daily clinical practice. Risk of Infection in Patients Receiving Long-Term Immunosuppressive Therapy In past years, substantial progress has been made in the treatment of chronic rheumatic and autoimmune diseases. Corticosteroids, azathioprine, and low-dose weekly methotrexate are widely used for these indications and are appropriate for most patients. Without questioning the usefulness and effectiveness of immunosuppressive treatment for patients with chronic rheumatic or autoimmune diseases, it must be kept in mind that the manipulation of the immune system that is inherent to these therapies may increase the risk of infection for the patients. Bone and joints, skin, soft tissues, and the respiratory tract are sites frequently involved in infectious processes in patients who have rheumatoid arthritis [1]. In patients with chronic inflammatory rheumatic or autoimmune diseases without arthritis, infections of the respiratory tract are the most common. The risk of infection is also dependent on the degree of immunosuppression associated with disease-modifying antirheumatic drugs DMARDs , most of which also have cytostatic effects [2â€"4]. Corticosteroids play an important role in this context because they are often used together with DMARDs and appear to increase cytotoxic effects, the degree of immunodeficiency, and the risk of infection in a dose-dependent manner [2 , 3]. These new agents amplify the immunosuppressive effects of traditional DMARDs by additional lymphocyte toxicity and inhibition of important cytokine and noncytokine activation pathways. Data from a large retrospective US study [5] and a German national database [6] demonstrated an increased infection rate among patients during treatment with TNF antagonists, compared with during treatment with traditional DMARDs. The use of TNF antagonists has also been identified as a risk factor for tuberculosis and other infections associated with a dysregulated Th1 response and also for mortality in patients with pneumococcal infections. Such patients have the highest risk of infection, not only for typical opportunistic infections, such as tuberculosis or Pneumocystis pneumonia, but also for infections that are common in the general population [3]. Common infections, in particular, are responsible for the majority of infectious episodes in patients receiving long-term immunosuppressive therapy [7]. Vaccines are available for a number of these infections and are a window of opportunity for prophylaxis. However, given the treatment-associated immune defects described above, several questions need to be addressed with regard to the immunogenicity, efficacy, and safety of standard vaccine preparations for these patients. In this review, the current evidence on these topics is summarized. Pneumococcal Vaccines Two pneumococcal vaccines are commercially available. Immunogenicity of pneumococcal vaccine in patients with chronic rheumatic or autoimmune diseases. Since the s, immune responses to the polysaccharide vaccine have been tested in several small studies [8â€"13] and have been reviewed by Elkayam et al. The most detailed investigation by Elkayam et al. The influence of prednisolone therapy on vaccine responses has not been evaluated in patients with chronic rheumatic or autoimmune diseases. In patients with chronic obstructive pulmonary disease or asthma, however,

corticosteroid therapy did not compromise the immune response to the pneumococcal polysaccharide vaccine [15]. De Roux et al. There are no studies of the effects of cyclosporine in patients with autoimmune diseases and chronic immunosuppression; however, in patients receiving cyclosporine therapy after heart transplantation, no impaired immune responses to the pneumococcal polysaccharide vaccine were observed [17]. The introduction of the biological DMARDs for treatment of rheumatic diseases has prompted several investigations with respect to the effects of these agents on immune responses after immunization with the pneumococcal vaccine. Postvaccination antibody levels in patients given treatment with a TNF antagonist alone or a combination of a TNF antagonist and a DMARD other than methotrexate were similar to those in healthy control individuals but were significantly higher than those in patients receiving only methotrexate. Immune responses to 5 of the 23 antigens of the polysaccharide vaccine were investigated by Mease et al. Logistic regression analysis identified methotrexate therapy and older age but not treatment with etanercept as independent factors for attenuated immune responses [20]. On the other hand, Visvanathan et al. That study found similar response rates of Abatacept and rituximab have been licensed recently for the treatment of rheumatoid arthritis, and detailed studies of immune responses to pneumococcal vaccines in patients receiving treatment with these agents are not yet available. Rituximab results in a long-lasting depletion of B cells. A decreased or blunted antibody response to vaccines, therefore, should be expected and has been demonstrated in patients with lymphoma who received treatment with rituximab. Of note, plasma cells appear to be less affected. Preliminary data did not show a decrease in preexisting antibody titers against tetanus, influenza, or pneumococcal antigens in patients receiving rituximab therapy during a week period [23], but studies evaluating longer periods are not currently available. Abatacept acts by inhibiting costimulatory pathways, which are essential for generation of an immune response to protein and peptide antigens. Decreased responses to such antigens, therefore, can be predicted, whereas T cellâ€”independent responses to polysaccharide e. However, a study involving volunteers revealed decreased immune responses after tetanus and pneumococcal vaccination [24]. Similar effects can also be expected for patients with chronic rheumatic and autoimmune diseases, but data are not available. Important issues are how long the protective titers after pneumococcal vaccination last for patients receiving immunosuppressive therapy and when such patients should be revaccinated. However, similar decreases in titers were seen in healthy persons [26]. The matter of revaccination, a topic of dispute even for healthy populations, is still unclear for immunocompromised patients. The pneumococcal conjugate vaccine has been licensed only for children to date. No studies are available on the immune response to this vaccine in patients receiving immunosuppressive therapy for rheumatic or autoimmune diseases. However, the conjugate pneumococcal vaccine has been shown to induce protective antibody titers in children with generally impaired immune responses at 6â€”9 months after bone marrow transplantation [27]. Effectiveness of pneumococcal vaccines in patients with chronic rheumatic or autoimmune diseases. No data are available on the effectiveness of pneumococcal vaccination in patients with autoimmune diseases. In other populations, however, vaccination against pneumococci has proved to be effective. The effect was most pronounced for infections caused by the serogroups covered by the heptavalent vaccine. A significant protective effect of pneumococcal vaccination has also been shown recently for elderly people [31]. Several studies have investigated the various aspects of influenza vaccination for patients receiving DMARD therapy. All these investigations were performed with the inactivated influenza vaccine, not with the live, intranasally administered attenuated vaccine Flumist; MedImmune. Immunogenicity of influenza vaccine in patients receiving long-term immunosuppressive therapy. Six weeks after vaccination, the patients with SLE generated immune responses against a mean of 1. Treatment with azathioprine was found to be associated with reduced vaccine response [33]. In contrast, Chalmers et al. TNF antagonists appear to have little impact on the response to influenza vaccines. Nevertheless, the majority of patients developed protective antibody levels after vaccination: Similarly, Gelinck et al. Preliminary data also presented by Gelinck et al. Effectiveness of influenza vaccine in patients with chronic rheumatic or autoimmune diseases. Two small studies found lower infection rates after influenza vaccination in patients with SLE or rheumatoid arthritis and in children with rheumatic diseases [40 , 41]. In other patient populations, however, the beneficial effects of influenza vaccination have been clearly demonstrated. Effectiveness for elderly people

has been evaluated and validated in several meta-analyses. Immunogenicity of the Hepatitis B Vaccine Vaccination against hepatitis B virus is generally recommended by numerous national immunization guidelines. Immunity against hepatitis B virus is especially relevant for patients beginning immunosuppressive regimensâ€”for example, treatment for malignant diseases or long-term immunosuppressive therapyâ€”because fulminant reactivation of latent chronic hepatitis B virus may develop under such conditions as a life-threatening complication. Safety and efficacy of vaccination against hepatitis B virus in patients with rheumatoid arthritis have been evaluated by Elkayam et al. Immunogenicity and Safety of the Varicella Zoster Vaccine Varicella zoster vaccine has been licensed recently in the United States and in some European countries for elderly patients at risk for herpes zoster virus infection, to prevent postherpetic neuralgia [43]. Patients receiving long-term immunosuppressive therapy are also at increased risk for developing herpes zoster. In patients with systemic lupus erythematosus, herpes zoster has been identified as one of the most common infections associated with immunosuppression [7 , 44 , 45]. Use of a live attenuated vaccine in immunocompromised patients is of potential concern, and the vaccine has not been approved for use in such patient groups. However, the risks associated with live vaccines presumably depend on the degree of immunodeficiency. As an example, varicella zoster vaccine has proved to be immunogenic, well tolerated, and effective in prevention of herpes zoster episodes in children with HIV infection and after renal or bone marrow transplantation. An inactivated varicella zoster vaccine has been evaluated in patients planning to undergo autologous stem-cell transplantation. It was found to be immunogenic and effective. Vaccine recipients showed significantly increased specific T cell responses and significantly fewer clinical herpes zoster episodes in a month period after transplantation than did patients who did not receive the vaccine [46]. Because the vaccine contains live attenuated viruses, the same theoretical concerns as for the varicella zoster vaccine apply, which potentially precludes its use for immunocompromised individuals. However, in a retrospective study involving vaccinated and nonvaccinated children with juvenile idiopathic arthritis, Heijstek et al. Antibody titers were not measured in that study. Similar safety experience has been reported for the vaccine in children with DiGeorge syndrome, in children after bone-marrow or solid-organ transplantation, and in HIV-positive children without severe immune defects. However, in HIV-positive children with severe immune defects, cases of measles, mumps, and rubella after vaccination have been observed. Vaccine Safety in Patients with Chronic Rheumatic or Autoimmune Diseases Numerous case reports indicate that vaccination may trigger or worsen autoimmune and rheumatic diseases, which is suggested by a timely relation between vaccination and disease exacerbation. Proinflammatory cytokines released during the immune response to vaccine antigens or mechanisms of molecular mimicry may be responsible for the initiation of a hitherto hidden rheumatic or autoimmune inflammatory process. There is still controversy about whether hepatitis B vaccination is associated with increased risk of development of demyelinating CNS disorders. Early reports that indicated an increased risk [49] were not confirmed by more-recent analyses [50]. Furthermore, whenever controlled, prospective vaccine-safety studies involving patients with autoimmune diseases were performed, no evidence of the exacerbation of existing autoimmune diseases or of the induction of new rheumatic or autoimmune processes was found [8 , 14 , 33 , 34 , 36]. However, the relatively small numbers of patients included in these studies may have precluded the detection of rare events. Vaccine Acceptance among Patients with Rheumatic Diseases.

Chapter 6 : Rajan Madhok (Author of The Year in Rheumatic Disorders, Vol. 5)

Rajan Madhok is the author of The Year in Rheumatic Disorders, Vol. 5 (avg rating, 0 ratings, 0 reviews, published), The Year in Rheumatic Disor.

Chapter 7 : Pain, catastrophizing, and depression in the rheumatic diseases â€” Johns Hopkins University

Beginning with v(1), January 1, , only those articles published via the "BMJ Open Access" option are available in PMC.

Chapter 8 : How Does Age at Onset Influence the Outcome of Autoimmune Diseases?

38th European Workshop for Rheumatology Research, February , Geneva, Switzerland.

Chapter 9 : Archive of "Annals of the Rheumatic Diseases".

title = "Pain, catastrophizing, and depression in the rheumatic diseases", abstract = "Persistent and disabling pain is the hallmark of osteoarthritis, rheumatoid arthritis, fibromyalgia, and various other rheumatologic conditions.