HIV and rheumatology: a practical guide

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- Human immunodeficiency virus (HIV) is a treatable chronic disease: mortality rates are the same in people with well controlled HIV infection as in HIV-negative adults. However, one quarter of HIV-positive adults in the UK are unaware of their HIV status. There should be a low threshold for testing for HIV in a rheumatological setting.

- Like chronic rheumatological diseases, long-standing HIV is characterised by increased risks of multiple co-morbidities, including metabolic, bone and psychological ill health.

- Patients with HIV can develop musculoskeletal symptoms due to HIV itself or due to antiretroviral therapy (ART).

- HIV and its treatment influence the course and outcomes of pre-existing rheumatological conditions. In practice, most rheumatological treatments can be used but particular care is required with corticosteroids and potent immunosuppressants.

- Close collaboration between rheumatologists and HIV clinicians is crucial in managing dual pathology.

HIV overview

Human immunodeficiency virus (HIV) is a retrovirus and the cause of the acquired immunodeficiency syndrome (AIDS). The first cases of AIDS were reported in 1981 in the United States and HIV itself was identified as the cause of this syndrome in 1983.1 Prognosis was initially poor, with severe opportunistic infections or malignancies (‘AIDS-defining conditions’) occurring in 7.7–11.0 years and death within 7.9–12.5 years of seroconversion, depending on age.2

The prognosis of HIV has improved dramatically since the introduction of effective combination antiretroviral therapy (ART) in the mid-1990s. Indeed, a recent observational study found no evidence of increased mortality rates (compared to the general population) among adults with well controlled HIV infection and CD4 counts above 500/μL enrolled in international clinical trials of ART.3

In 2012 the number of people living with HIV globally was estimated at 34 million, with most (around 70%) living in sub-Saharan Africa.4 In the UK in the same year, the number of people living with HIV was estimated to be 98,000,5 of whom approximately 22,000 (24%) were unaware of their HIV status. Late diagnosis is the principal risk factor for mortality among HIV-positive individuals in the UK and widespread HIV testing in a diverse range of medical settings is therefore key to improving survival.6

Management of HIV follows similar principles to other chronic illnesses, including rheumatological conditions; it is essential that there is mutual understanding and collaboration between the
healthcare team and people with HIV. Adverse effects and consequences of long-term medication must be considered and there is a need to acknowledge the socioeconomic and psychological impact of having a chronic condition. In addition, as an infectious disease, contacts and family members may also be affected and healthcare workers’ responsibilities extend to individuals other than the patient.

The aim of treatment is to reduce mortality and risk of opportunistic conditions by maintaining a well-functioning immune system, using timely ART to achieve and maintain an undetectable plasma HIV viral load. In this review we focus on rheumatological issues in the context of good access to ART. The content of the review would be very different if written from the perspective of resource-limited settings in many countries with high HIV prevalence.

Primary HIV infection

Primary HIV infection is characterised by high levels of viral replication and a fall in CD4 count, before and during the formation of an antibody response. The high viral load is a risk factor for onward transmission of HIV, particularly if the individual is unaware of their recent HIV acquisition. Symptoms during primary infection predict faster disease progression and death so early diagnosis is essential. Most people with primary HIV report symptoms, typically a glandular fever-type illness, with fever in most patients, with other common features being pharyngitis, rash and arthralgia in about a third of cases. Acute rhabdomyolysis or a raised creatine kinase (CK) have been reported as a manifestation of primary HIV infection. Those consulting rheumatologists may present with arthralgia, myalgia or rash, with raised inflammatory markers. Hyperferritinaemia has been reported, mimicking adult-onset Still’s disease. Diagnostic HIV testing should be requested if primary HIV infection is suspected. If the lab is alerted to the possibility of primary HIV then polymerase chain reaction (PCR) for viral ribonucleic acid (RNA) can be performed if standard combined antibody/p24 antigen tests are negative. HIV tests currently in use are capable of detecting viral RNA as soon as a week after infection, and are positive during acute HIV infection. If testing is negative and clinical suspicion is high, the test can be repeated after two weeks.

Current British HIV Association (BHIVA) guidelines recommend initiating antiretroviral treatment in primary infection only if there is neurological involvement, in the presence of AIDS-defining illness or if the CD4 falls to below 350/μL. Treatment should also be considered in the event of severe symptoms. As in rheumatoid arthritis (RA), early diagnosis and intervention in HIV improves outcome, so if primary HIV infection is suspected prompt referral to an HIV physician is essential. Some have argued that ART should be offered in less advanced disease, to almost everyone infected with HIV, and this is the approach recommended in the USA.

HIV testing

It is crucial that all clinicians, including rheumatologists, have a very low threshold for testing for HIV. When and how to test is summarised in boxes 1 to 3. BHIVA does not make specific recommendations for testing with rheumatological presentations but Box 2 includes clinical situations that are commonly encountered in rheumatology clinics. This list is by no means exhaustive. Screening for HIV should also be considered before starting potent immunosuppressants. This approach is supported by national and international guidelines, which suggest

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**BOX 1. Indications for HIV testing**

An HIV test should be considered in the following settings where diagnosed HIV prevalence in the local population exceeds 2 in 1000 population (these prevalence data are available from local Public Health services):

1. all men and women registering in general practice
2. all general medical admissions.

HIV testing should be also routinely offered and recommended to the following patients:

1. all patients presenting for healthcare where HIV, including primary HIV infection, enters the differential diagnosis (see table of indicator diseases (Box 2) and section on primary HIV infection)
2. all men and women known to be from a country of high HIV prevalence (>1%*).
testing prior to treatment with TNF inhibitors in RA, and prior to treatment with any immunosuppressive therapy including glucocorticoids in systemic lupus erythematosus (SLE). Many departments, including the authors’, test all patients commencing biologic therapy although not usually with lesser degrees of immunosuppression. However, the 2012 American College of Rheumatology (ACR) guidelines for treatment of RA make no reference to HIV testing.

Drug interactions relevant to rheumatology

Antiretroviral drugs are responsible for a complex and extensive range of drug interactions, including with drugs commonly used in rheumatology – although in practice most DMARDs and biologic drugs do not have major interactions. We strongly advise that, when prescribing any new or unfamiliar medication, treating physicians should always check for interactions using the free tool at www.hiv-druginteractions.org. It should be noted that access to clinic letters related to HIV treatment may be limited as these are often stored separately (in genitourinary or sexual health services), so a careful drug history is essential.

One of the most common causes of interactions is the widely used protease inhibitor (PI), ritonavir. This drug was originally developed as a specific inhibitor of HIV protease but is now used to inhibit the metabolism, and therefore boost the action, of other PIs in HIV. Ritonavir is a potent inhibitor of liver enzymes CYP3A4 (which metabolises many commonly used glucocorticoids) and CYP2D6. Ritonavir can potently increase the action and duration of action of corticosteroids. Severe Cushing’s syndrome has been reported on several occasions following single injections of triamcinolone (usually as Kenalog) for musculoskeletal disease. Triamcinolone preparations should not be administered to patients on ritonavir. While a similar effect might be predicted in patients given methylprednisolone, in practice this does not seem to induce the same degree of hypercorticoid changes. Nevertheless, care needs to be exerted in the use of all corticosteroids in patients taking ritonavir.

Ritonavir and other protease inhibitors can also greatly increase the toxicity of colchicine. Colchicine should therefore probably be avoided in patients taking PIs.

DMARDs and other rheumatological drug treatments in HIV

Broadly speaking, the approach to drug treatment of chronic rheumatological disease is similar in patients with and without HIV. Perhaps surprisingly, many immune-modulating and moderately immunosuppressive drugs seem to be safe, at least for short-term use. It should be emphasised that this impression of safety is based upon use in small numbers of patients without prolonged follow-up – hence rare and delayed adverse effects cannot be excluded. Some principles are suggested:

- Treatment decisions should be made jointly with the patient and HIV physicians.
- Immunosuppressive drugs should generally only be used when the HIV is well controlled (CD4 count greater than 350/μL with undetectable viral load.

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**BOX 2. MSK and related conditions where HIV testing should be offered, adapted from BHIVA guidelines**

<table>
<thead>
<tr>
<th>Neurological:</th>
<th>Peripheral neuropathy</th>
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<tbody>
<tr>
<td>Dermatological:</td>
<td>Severe or recalcitrant seborrhoeic dermatitis</td>
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<tr>
<td></td>
<td>Severe or recalcitrant psoriasis</td>
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<td></td>
<td>Multidermatomal or recurrent herpes zoster</td>
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<tr>
<td>Respiratory:</td>
<td>Bronchiectasis</td>
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<tr>
<td>Other:</td>
<td>Pyrexia of unknown origin</td>
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<td></td>
<td>Lymphadenopathy of unknown cause</td>
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<td></td>
<td>Chronic parotitis</td>
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<td></td>
<td>Lymphoepithelial parotid cysts</td>
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<td></td>
<td>Mononucleosis-like parotid syndrome (primary HIV infection)</td>
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<tr>
<td></td>
<td>Hepatosplenomegaly</td>
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<td></td>
<td>Tuberculosis at any site</td>
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<tr>
<td>Associated with reactive arthritis:</td>
<td>Any sexually transmitted infection</td>
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<td></td>
<td>Salmonella, shigella or campylobacter</td>
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<tr>
<td>Laboratory abnormalities:</td>
<td>Unexplained blood dyscrasia (thrombocytopenia, neutropenia, lymphopenia)</td>
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<tr>
<td></td>
<td>Unexplained autoantibodies</td>
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</tbody>
</table>

**BOX 3. How to do an HIV test**

**Pre test**

1. Obtain consent and confirm how test result will be given to the patient.
2. Further counselling not required unless patient has more questions or is considered very likely to have a positive test

**Interpret result**

If primary HIV infection is suspected, request PCR for viral RNA if standard combined antibody/p24 antigen tests are negative.

All positive HIV tests should be confirmed by further laboratory testing.

**Action if positive result:**

Refer immediately to HIV specialist for management and contact-tracing.
on ART), and should be closely monitored. A falling CD4 count or rising viral load should prompt review of immunosuppressive drug use.

- Extreme caution should be exercised in the use of aggressive immunosuppression.
- Vigilance for infection is essential, and immunisation should be used to reduce the risk where possible. The varicella zoster vaccine is safe in well controlled HIV infection.21

Hydroxychloroquine and sulphasalazine seem safe and well tolerated. In vitro22 (but not in vivo23) evidence suggests hydroxychloroquine may inhibit HIV replication. Sulphonamide drug allergy is generally more common in HIV infection, but reactions to sulphasalazine do not seem to be more common. There are no major drug interactions for these drugs. Methotrexate was associated with major infection in case reports from the pre-ART era.24,25 but seems to be well tolerated in well controlled HIV.26,27

More potent immunosuppressive drugs (for example mycophenolate, azathioprine and ciclosporin) have been used without major short-term toxicity.26,28-30 Longer-term use raises concerns about the risk of infection and lymphoma (the risk of lymphoma is increased in HIV even in the ART era31). Cyclophosphamide and aggressive chemotherapy have been used in life-threatening vasculitis32 and lymphoma in HIV,31 and can be tolerated, but extreme caution is advised, especially as safer alternatives may now be available.

Corticosteroids (other than triamcinolone) seem relatively safe and well tolerated in short-term use in HIV infection, but caution is needed in terms of the increased risk of infection and the metabolic consequences of corticosteroid use (including bone loss) that have the potential to exacerbate the metabolic syndrome characteristic of chronic HIV.33

TNF inhibitors seem well tolerated14,34-36 but the strong association between HIV and tuberculosis suggests the risk of mycobacterial (and other intracellular infection) may be particularly increased.

Use of rituximab in rheumatic disease in HIV seems to have been minimal, although rituximab has been widely used in the treatment of HIV-associated Castleman’s disease and lymphoma and seems to be well tolerated, but with a particular risk of reactivation of Kaposi’s sarcoma.37 It should be noted that infections typically associated with HIV, such as pneumocystis pneumonia, cryptococcal infection and progressive multifocal leukoencephalopathy have been described in patients with RA treated with rituximab without HIV. This suggests particular caution may be needed in use of rituximab in this context.

There is no published experience with other biologics such as tocilizumab and abatacept.

Musculoskeletal co-morbidity in HIV

The spectrum of musculoskeletal co-morbidity has changed considerably as HIV has evolved from a progressive fatal disorder to a manageable chronic disease. Reviews of HIV and rheumatology from the 1980s and 1990s24,38 focus on infection, spondyloarthritis, myositis and multisystem disorders associated with HIV, such as the diffuse infiltrative lymphocytosis syndrome (DILS). More recent accounts27,29,30 have a strong focus on bone disease and the immune reconstitution inflammatory syndrome (IRIS) associated with treatment. This evolving picture, coupled with differential access to treatment across the world, makes it difficult to draw overarching conclusions about the epidemiology of musculoskeletal co-morbidity, so in this review we draw some practical conclusions based on UK practice. It is clear that rheumatic illnesses can antedate HIV infection, occur as part of HIV seroconversion, HIV itself, or as part of the immune reconstitution inflammatory syndrome. It is also clear that pain is common in patients with HIV, and has a significant impact on quality of life. Non-specific arthralgias, back pain and fibromyalgia are probably more common than in the general population.30 Soft tissue rheumatic complaints such as carpal tunnel syndrome, tendinitis, and shoulder capsulitis also appear to be more common in patients with HIV.30

Musculoskeletal presentations in HIV and musculoskeletal complications of HIV treatment

Arthralgia

Joint pain is very common in HIV-infected patients and is usually mild, intermittent and polyarticular. Arthralgia predominantly affects the knees and shoulders. Although there are limited data, the introduction of ART does not seem to have reduced the prevalence of arthralgia.30 The painful articular syndrome seems to be specific to HIV, and describes a severe, sharp, articular pain of 2–24 hours’ duration, without signs of inflammation, requiring NSAIDs and/or narcotic drugs. This syndrome is most often seen in advanced HIV infection. It is speculated that this may be due to bone oedema related to the increased incidence of avascular necrosis.

Polyarthritis including RA

The existence of an HIV-specific arthritis remains a matter of debate. Certainly, if it exists, this is not a major clinical problem.
Early case reports suggested that RA became quiescent with the progressive immunodeficiency caused by HIV infection. However, while some cases may improve as HIV worsens, symmetrical, polyarticular, erosive RA has been reported in patients with low CD4 counts and certainly occurs in patients with well controlled HIV infection. Subject to the caveats above, RA can be managed conventionally in the context of HIV. Although some would argue for a bias towards the use of sulfasalazine and hydroxychloroquine as first-line treatments.

Immunological abnormalities associated with RA can also occur in HIV-infected patients without arthritis, for example, hypergammaglobulinaemia, and positive tests for rheumatoid factor and anti-CCP. These are rarely of clinical significance, are usually low titre and associated with low CD4 counts. These serological changes often decrease or resolve with ART.

Gout
Pre-ART data suggested that around 1 in 200 people with HIV develop gout annually. There have been no epidemiological studies of gout in the ART era, but hyperuricaemia is associated with several antiretroviral drugs (particularly PIs) and there is a strong clinical impression that gout is now much more common, and associated with low CD4 counts. These serological changes often decrease or resolve with ART.

Spondyloarthritis
Spondyloarthritis, particularly psoriatic and reactive arthritis, featured prominently in earlier reports of rheumatological disorders seen in HIV. An increased risk of SpA was linked to the associations between these rheumatological disorders and infection. Inflammatory back pain may be a presenting feature of HIV infection even in populations where HLA-B27 is rare.

Ankylosing spondylitis seems less common perhaps due to difficulty in making a radiological diagnosis without longer-term follow-up, or perhaps because of the suggested protective effect of HLA-B27 on HIV progression.

The incidence of reactive arthritis (ReA) varies between series, probably reflecting variation in the mode of acquisition of HIV since other sexually transmitted infections are common triggers for reactive arthritis. Overall, the data regarding the effect of ART on ReA are inconclusive: for instance there are cases of severe ReA resolving as the advanced HIV improved with ART and conversely new ReA occurring as part of IRIS.

The prevalence and severity of psoriatic arthritis (PsA) also differ between studies. Overall, PsA seems to be more common and more severe in the later stages of HIV. Some authors quote an improvement of PsA with ART, although this is not confirmed by others. Established HIV infection can present with articular and extra-articular features (such as keratoderma blennorrhagica) of severe spondyloarthritis, although anecdotally this presentation seems less common than in the earlier years of the pandemic.

Multisystem presentations
Most systemic autoimmune diseases have been described in the context of HIV infection (for example, SLE, giant cell arteritis, Behçet’s disease and many other forms of vasculitis). Presentation and treatment are usually not altered by the presence of HIV, although caution is needed with aggressive immunosuppression.

The diagnosis and management of SLE in the context of HIV can be complex: HIV and SLE can have similar clinical presentations (oral ulcers, alopecia, fever, and arthralgia), can be associated with cytopenias and are associated with autoantibodies. SLE itself can occasionally cause false positive HIV antibody testing, although this has been less of a concern since the widespread use of combined antibody and antigen testing.

Antinuclear antibodies are often present in HIV (as in other chronic infections), but high-titre dsDNA antibodies seem rare (although have been described). Complement levels are usually normal. Most serological data are from the pre-ART era, and it is unclear whether newer HIV therapies have changed this autoantibody profile.

Anticardiolipin antibodies are seen commonly in HIV infection (as in other chronic infections) and anti-β2 glycoprotein I antibodies are also found frequently. However, lupus anticoagulant is only rarely detected. Again, most serological studies were performed before the introduction of ART. Antiphospholipid antibodies in HIV are not usually associated with thromboembolic disease and may resolve with ART.

As noted above, the diffuse infiltrative lymphocytosis syndrome (DILS) was prominent in earlier reviews of rheumatological aspects of HIV. This is a syndrome clinically similar to Sjögren’s syndrome, with sicca symptoms and often marked salivary gland enlargement. Inflammatory infiltrates are also more often seen although typical Sjögren’s autoantibodies are absent. Salivary gland histology shows characteristic cystic changes with lymphocyte infiltration. Since DILS responds very well to ART, it is now much less
common. However, DILS remains a major differential diagnosis for ‘seronegative’ Sjögren’s syndrome.

IRIS (immune reconstitution inflammatory syndrome, or immune restoration disease)

Treatment of HIV with ART aims to restore and maintain near-normal immune function. Rapid restoration of immune function can be associated with a marked inflammatory response – IRIS. This is an exaggerated or deregulated immune response, usually to opportunistic infections, but reactivation of pre-existing autoimmune disorders can occur, and new autoimmune syndromes can also develop. Many syndromes have been described in this context, including SLE, RA, and particularly autoimmune thyroiditis and sarcoidosis. Most arise de novo, although about 20% are pre-existing disease made quiescent by the immunosuppression of advanced HIV. The time of onset of IRIS is usually around 4–8 weeks after initiation of ART, although autoimmune conditions can develop much later: a median 21 months for Graves’ disease, and up to 3 years for sarcoidosis. Risk factors for developing IRIS include severe immunosuppression (low nadir CD4 count), first exposure to ART and recent opportunistic infections. Most symptoms resolve spontaneously although severe IRIS may require treatment with corticosteroids. ART can usually be continued alongside steroid treatment, with particular caution required in eye or central nervous system disease.

Bone disease in HIV

Osteopaenia and osteoporosis

Osteoporosis is a major emerging problem in chronic HIV infection. Bone mineral density (BMD) can be reduced by HIV itself, antiretroviral therapy and vitamin D deficiency as well as traditional risk factors unrelated to HIV. With an ageing HIV-positive population, co-morbidities such as osteoporosis need to be managed collaboratively with HIV physicians, rheumatologists and primary care. Use of the antiretroviral agent tenofovir, a nucleotide reverse transcriptase inhibitor, and the protease inhibitors have been particularly associated with low BMD compared with other agents, although this does not appear to result in a higher proportion of patients experiencing fractures. Reductions in BMD and increased markers of bone turnover were seen in the first 6–12 months of antiretroviral therapy, but not thereafter. Of course, timely treatment of HIV infection remains essential, as the inflammatory state and lower CD4 counts associated with untreated disease are associated with worse effects on BMD and other co-morbidities, including cardiovascular disease and malignancies.

Low vitamin D levels are common among HIV-positive individuals in the UK and repletion should be considered. Current BHIVA guidance is that routine DEXA scanning is not recommended for all HIV-positive individuals, but should be done routinely in women aged 65 years or more and men aged 70 years or more. In addition, DEXA scanning should be considered in the over-50s and where there are other risk factors for bone loss, such as a low nadir CD4 (the lowest CD4 in the course of the illness, usually pre-treatment) or high baseline viral load, or where a high or intermediate risk is identified by FRAX® score.

Avascular necrosis (AVN)

AVN is seen more frequently among HIV-positive than HIV-negative individuals. It most commonly affects the femoral head, although other joints, or multiple joints, can be involved. Corticosteroid use, hyperlipidaemia, smoking and HIV-related factors such as low nadir CD4 count and previous AIDS-defining illness have been associated with AVN in observational studies. The contribution of ART to AVN remains uncertain. The development of severe and persistent non-traumatic bone pain at any site in the context of HIV infection should prompt consideration of avascular necrosis (Figure 1). Conversely, HIV infection should be considered as a cause of AVN, especially when multifocal.

Hypophosphataemic osteomalacia

Tenofovir can also cause renal tubular dysfunction, with increased renal loss of phosphate ions. Rarely, this can be severe enough to cause Fanconi syndrome, with hypophosphataemic osteomalacia. This usually presents with painful proximal myopathy, with radiological pseudofractures (Figure 2). Treatment is with withdrawal of tenofovir and phosphate replacement, although this may need to be at very high dose and

![FIGURE 1. Coronal PD fat-suppressed image of R knee showing osteonecrosis of the distal femur and a large joint effusion](image-url)
Infections

Although immunodeficiency is a risk factor for bone and joint infections, (perhaps surprisingly) musculoskeletal infection does not appear to be significantly increased in HIV-positive compared with HIV-negative patients. Some series report an increased incidence of septic arthritis. Staphylococcus aureus is the most common organism, as in non-HIV-associated sepsis. However, the CD4 count does influence the pathogen: pyogenic organisms predominate if CD4>250/µl whereas opportunistic organisms are observed when the CD4 counts are <100/µl (Mycobacterium haemophilum and M kansaii being the most common). Other severe musculoskeletal infections, such as pyomyositis and osteomyelitis are rare in the ART era.

Muscle

Muscle involvement in HIV-positive patients can manifest as weakness or myalgia and may be due to the HIV, antiviral treatment or reflect muscle disease unrelated to HIV. Muscle weakness in HIV may also be due to neuropathy or CNS diseases, or the ‘wasting syndrome’ seen in advanced HIV infection (now rare in the ART era).

Muscle pain is common and considered by people with HIV to be an unpleasant and intrusive symptom. It can be present as part of the primary HIV seroconversion or can occur due to medication. Fibromyalgia remains a common cause of muscular pain.

- High-dose zidovudine causes myopathy but other nucleoside reverse transcriptase inhibitors do not seem to cause this problem. CK levels are often normal but muscle biopsy shows ragged red fibres, suggesting mitochondrial dysfunction. Myopathy usually resolves within 1–2 months of stopping the drug. With the advent of ART high-dose zidovudine is now seldom used, and therefore its muscular effects are less common.
- Rhabdomyolysis may be associated with primary HIV infection and with very advanced HIV disease.
- Statins are widely used to treat dyslipidaemia associated with ART and can cause myalgia and, less commonly, rhabdomyolysis. Simvastatin has major drug interactions and should not be used in patients on ART. Pravastatin is probably least likely to cause muscle side-effects and has minimal drug–drug interaction with PIs.
- HIV-associated polymyositis was seen in 2–7% of patients in the pre-ART era, and has been described at every stage during the course of HIV infection. It is clinically and histologically indistinguishable from idiopathic polymyositis. However, auto-antibodies are often absent, EMG can be normal, and muscle biopsies may not show typical inflammatory changes. It generally has a good outcome with conventional immunosuppressive therapy and may even resolve spontaneously. It is hypothesised that muscle damage is caused by direct invasion of muscle cells by HIV.
- Dermatomyositis and inclusion body myositis seem rare in HIV-infected patients.
- Nemaline (rod) myopathy is a rare but well described cause of painless muscle weakness in HIV-positive individuals. CK may be mildly elevated or high, and muscle biopsy shows no inflammation with conventional histology but characteristic nemaline bodies on electron microscopy or rod structures in atrophic fibres. Despite the absence of muscle inflammation on biopsy, some patients may respond to prednisolone or plasmapheresis.
- Osteomalacia associated with tenofovir usually presents with painful myopathy, as discussed above.

Conclusion

HIV infection is not rare in the UK, and rheumatologists, like all physicians, need a good working knowledge of HIV medicine, and how this impinges upon their own specialist area of practice.

Rheumatologists will recognise some common themes between their specialty and management of HIV – for example, the importance of early diagnosis and treatment in improving outcome, and the importance of detecting and managing co-morbidities in chronic disease.

Rheumatologists need to be aware of altered differential diagnoses of musculoskeletal presentations in...
HIV infection and also of the potential for major drug interactions with antiretroviral therapy, particularly with triamcinolone and colchicine.

Management of chronic autoimmune disease in people with HIV requires a meticulous balancing of the risks and benefits of immunosuppression, although in practice the basic approach to management is not changed by HIV. Long-term follow-up data are required to determine whether additional risks emerge with long-term treatment with biologic drugs and other powerful immunosuppressants.

A close working relationship between rheumatologists and specialists in HIV medicine is, and will remain, crucial in managing HIV-associated rheumatic disease.

References


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ISSN 1759-7846. Published 3 times a year by Arthritis Research UK.