Vascular disease in HIV/AIDS

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The well-documented relationship between vascular disease and HIV infection has evolved from infectious and inflammatory vasculitides to:

• premature atherosclerosis
• its related contributing conditions (metabolic syndrome, dyslipidaemia, insulin resistance syndrome) and
• complications (acute coronary and cerebrovascular syndromes).

Triple antiretroviral therapy ensures adequate viral suppression, which is paramount to the successful clinical management of HIV-infected patients. The associated metabolic effects as well as the increased longevity associated with these drugs increase the cardiovascular manifestations, which should be prevented and treated.

Perioperative risk assessment and reduction

The American Heart Association (AHA) ‘Initiative to Decrease Cardiovascular Risk and Increase Quality of Care for Patients Living with HIV/AIDS’ concluded that metabolic and anthropometric abnormalities contribute to cardiovascular disease risk. These abnormalities may be due to HIV or its therapy and may be affected by environmental and immunological factors with the net effect of being pro-atherogenic.

The Pavia consensus statement brought the following important aspects to attention:

• HIV infection may directly cause cardiovascular disease
• HIV therapy may cause cardiovascular disease
• HIV therapies may interact with drugs used for cardiovascular complications
• Primary prevention of coronary heart disease in patients receiving antiretroviral therapy includes exercise, diet and treatment of hyperlipidaemia, hypertension and glucose intolerance.

Peripheral arterial occlusive disease (PAOD) in HIV-infected patients
Van Marle reported the classic risk factors for PAOD to be less prevalent in their studied HIV population. Patients were also of younger age than expected at presentation. Three clinical syndromes may present:

Atherosclerotic disease
Contributing risk factors are:

• disease specific (metabolic syndrome, dyslipidaemia, insulin resistance syndrome, antiretroviral therapy, i.e. protease inhibitors) or
• due to a high prevalence of the traditional risk factors for atherosclerosis in this population.

Future research has to determine whether HIV infection and antiretroviral therapy contribute independently to increased cardiovascular disease.

Non-atherosclerotic disease

• Infectious arteritis (CMV, varicella zoster virus, mycobacterium and fungal infections)
• inflammatory vasculitis (leucocytoclastic vasculitis), polyarteritis nodosa (PAN), Henoch-Schönlein purpura, drug-related hypersensitivity vasculitis, temporal arteritis/polymyalgia rheumatic and nonspecific unclassified vasculitis) and
• microvascular changes (thrombocytic thrombotic purpura (TPP) and haemolytic uraemic syndrome).

Prothrombotic state

An increased prothrombotic state is observed in HIV patients with active opportunistic infections or malignancy and in patients with AIDS. The potential mechanisms proposed are the presence of antiphospholipid antibodies, such as anticardiolipin antibodies and lupus anticoagulants, and also increased levels of von Willebrand factor, fibrinogen and D-dimers as well as deficiencies of protein C, protein S, antithrombin III, heparin cofactor II and increased platelet activation.

Thus, the multifactorial pathogenesis may explain the peculiar characteristics and features of HIV-associated occlusive vasculopathy (Table I).

Aneurysms in HIV-infected patients (Fig. 1)
The pathogenesis is uncertain. It is commonly associated with, but not exclusively due to, a leucocytoclastic vasculitis. This process results in obliteration of the vasa vasorum and transmural necrosis. Infective aetiologies are a sporadic rather than a consistent finding.

Unlike degenerative aneurysms, these are found in atypical sites (common carotid and superficial femoral artery) with a saccular/multi-loculated morphology and tend to be multiple. Low CD4 counts as well as hypoalbuminaemia are consistent findings with prognostic implications.

Table I. Characteristics and features of HIV-associated occlusive vasculopathy

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young age at onset of disease (mean age 40 yrs)</td>
<td>Most patients present with advanced vascular disease (&gt;50% critical limb ischaemia)</td>
</tr>
<tr>
<td>Smoking is a common traditional risk factor</td>
<td>CD4+ T cell count &lt;200 cells/µl</td>
</tr>
<tr>
<td>Hypo-albuminaemia is a reliable predictor of increased perioperative complications and poor long-term survival</td>
<td>Infra-inguinal disease more often than aorto-iliac occlusive disease</td>
</tr>
<tr>
<td>Macroscopic findings</td>
<td>Normal arteries proximal, and the occlusion is of a fibro-obliterative nature with cords/strings of fibrous tissue which can be removed from the arterial lumen</td>
</tr>
<tr>
<td>Arteriographic findings</td>
<td>Pristine proximal arteries with extensive disease extending into the small and medium-sized arteries with poor run-off</td>
</tr>
<tr>
<td>Duplex ultrasonographic findings</td>
<td>Hyperechoic ‘spotting’ in the arterial wall, the ‘string of pearls sign’</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Limb salvage rate 27%</td>
</tr>
<tr>
<td>Wound sepsis</td>
<td>Graft sepsis</td>
</tr>
<tr>
<td>Perioperative mortality 6.95%</td>
<td>Late mortality 28.75%</td>
</tr>
</tbody>
</table>

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The 2008 American College of Chest Physicians guidelines make no reference to the HIV-infected patient suffering from VTE. Veller and Eyal recommend prophylaxis along the same guidelines as those for a patient suffering from cancer and standard treatment protocols for patients with proven VTE.14

References available at www.cmej.org.za

**Table I. Anal disorders in HIV-positive patients (most often found in combination)**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Average incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proctitis/skin involvement</td>
<td>40</td>
</tr>
<tr>
<td>Condylomata acuminata</td>
<td>43</td>
</tr>
<tr>
<td>Fissure-in-ano</td>
<td>30</td>
</tr>
<tr>
<td>Anal ulcer</td>
<td>29</td>
</tr>
<tr>
<td>Haemorrhoids</td>
<td>15</td>
</tr>
<tr>
<td>Chlamydia/gonorrhoea</td>
<td>2</td>
</tr>
<tr>
<td>Kaposi's sarcoma</td>
<td>2</td>
</tr>
<tr>
<td>Squamous cell carcinoma</td>
<td>1</td>
</tr>
</tbody>
</table>

Anal incontinence and soiling is a socially unacceptable and often devastating problem associated with anal disease in AIDS.

The higher prevalence of malignant disease in these patients should also be kept in mind, i.e. Kaposi's sarcoma of the anal canal skin, Hodgkin's lymphoma and possibly squamous cell carcinoma.4

**Clinical approach**

**History**

A careful history of the main complaint, all symptoms associated with the anal area, and function and anatomy of the anus should be obtained in an objective, formal yet sympathetic fashion. Patients are reluctant to divulge details and may have to be gently prompted to obtain an adequate picture of the pathology and altered function involved. Questions on sexual practices and particularly anal intercourse can sometimes be embarrassing to both patient and physician. Matter-of-fact queries with regard to pain during anal intercourse will afford the patient the opportunity to deny or admit to such practice; subsequent response to questioning may then follow spontaneously.

Keeping in mind the possible pathology, sensitively formulated leading questions will often clear doubts regarding the problem to be managed and its intensity.

**Local examination**

Most patients can be examined in a consulting room or clinic cubicle. Privacy, nursing/assistant support and good lighting are basic requirements, as well as anaesthetic jelly, swabs, lubricant, a proctoscope, malleable probes and suctioning equipment. Facilities for needle, forceps and open biopsies should be available and a bi-valve speculum for vaginal examination in female patients should always be at hand. Positioning is very important: in addition to the usual Sims' position (left lateral knee flexed), the knee-chest (jack-knife) position can be practical.5

In a limited number of patients the above approach will not be possible because of age, non-compliance and pain. An examination under general anaesthesia should then be
done. Look for pathology that should be expected, i.e. skin lesions, condylomata, abscesses and sepsis, proctitis, small peri-anal fistulas, fissures, infected thrombosed haemorrhoids and sphincter tone. Anal canal ulcers and multiple fissures may be missed, and associated rectal disease should be identified. In advanced destructive anal disease of fully fledged AIDS the findings may be confusing because of the presence of multiple pathology, and a descriptive clinical assessment should be made (Figs 1-6).

It may be necessary to perform proctosigmoidoscopy, either of the rigid or preferably of the flexible variety, to exclude or confirm the presence of associated rectosigmoid disease.

Appropriate special investigations such as MCS, histology and ultrasonography of the anal canal should be available to come to a final diagnosis.

The presenting symptoms of anal disease in HIV-positive patients in order of prevalence are the following:

- pain
- soiling
- lump/tumour
- blood/pus in stool
- diarrhoea/tenesmus.

Fig. 1. Perineal abscess.

Fig. 2. Hypertrophied anal papillae.

Fig. 3. Condylomata acuminata.

Fig. 4. Acute proctitis.

Fig. 5. Anal canal tissue breakdown.

Fig. 6. Squamous carcinoma.

Be careful not to hurt the patient or to cause further harm. Every clinical finding during the physical examination should be fully investigated and adequately documented in the patient’s file.

Management

Protection of the clinician and all nursing and other staff, including students, should be a priority. Gloves, masks and/or a visor must be used.

Surgical intervention in the treatment of anal disease, e.g. haemorrhoidectomy, should be carefully considered in the HIV-positive patient because of delayed wound healing and even non-healing. According to some, the use of surgery should be limited to the drainage of pus. Disease in this area is usually more aggressive and persistent, and the recurrence rate of resected condylomata has been reported to be as high as 50% within 3 months. Peri-anal necrosis and necrotising fasciitis after surgery is a dire and often fatal complication.

In spite of the above, recent reports have been more positive regarding healing and cure rates after surgery. Acceptable results are reported with most interventions and an ‘appropriately aggressive approach’ seems justified.

It is however very important that surgical intervention be restricted to conditions that require surgery as a matter of urgency and in which conservative management is deemed inadequate. Therefore, a haemorrhoidectomy on uncomplicated haemorrhoids would be contraindicated in the HIV-positive patient, while abscesses, active infected fistulas, anal ulcers, complicated haemorrhoids and extensive condylomata are important when considering surgery. Patients on antiretroviral therapy generally respond well. Satisfactory wound healing and eventual cure usually follow meticulous surgical technique and follow-up in more than 80% of cases. Patients scheduled for surgical intervention must be on effective antiretroviral therapy; a low CD4 count (<200µl) and a high viral load may lead to poor or no healing of surgical wounds.

Careful selection of patients for surgery, gentle tissue-saving techniques and meticulous postoperative follow-up should result in a positive outcome.

Every patient’s needs should be individualised and potential life-threatening complications of a conservative approach should be weighed against the 20% chance of poor results and non-healing after surgery.

References available at www.cmej.org.za
Soft-tissue tumours and HIV/AIDS

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Soft-tissue sarcomas are a diverse group of malignancies, with the majority originating from the mesoderm. The mesoderm accounts for approximately 75% of the body’s mass but only for about 1% of tumours in the adult population. Approximately 43% arise from the extremities and the remainder from various other anatomical sites.1

With the advent of increasingly available antiretroviral drugs, patient life expectancy is increasing and so is the incidence of previously rare conditions, such as Kaposi’s sarcoma and lymphoma.

Risk factors

There are certain well-defined risk factors, including ionising radiation, chromosomal abnormalities, chemical exposure and lymphoedema. Some sarcomas have an increased incidence in the HIV population, notably Kaposi’s sarcoma and various lymphomas.

Epstein-Barr virus is associated with smooth-muscle tumours in patients with HIV.2

The most commonly used staging system is based on the AJCC (American Joint Committee on Cancer) guidelines. The AJCC guidelines combine various factors, i.e. tumour grade, tumour size, depth of invasion, degree of nodal involvement and presence or absence of metastatic disease.2

Examination

Examination often reveals very few abnormalities, but the following factors indicate an increased risk of malignancy and should be actively sought and noted in patient records:4
• pain
• function loss
• neurovascular bundle invasion
• >5 cm in size
• located deep to the fascia
• systemic symptoms
• metastasis
• recurrence
• increased vascularity, i.e. warm.

Biopsy

A mass that has been present for more than 4 weeks or is greater than 5 cm in diameter is an indication for a biopsy. Core needle biopsy (e.g. Tru-cut needle) has been shown to be accurate with regard to sensitivity and specificity. Fine needle aspiration should be avoided, as this does not allow grading or histological typing. Incision biopsies have a potential to seed cancerous cells and should be done by the surgeon who takes responsibility for the final surgical management of the tumour, if possible.

Therapy

In the past amputation was considered to be the only option for treating soft-tissue tumours of the extremities. Currently, excision with a margin of at least 1 - 2 cm is considered adequate. Compartment excisions have shown to be of no greater benefit than wide local excisions. Excision with clear margins yields recurrence rates of between 12% and 31%.

Kaposi’s sarcoma

This particular disease represents the most common tumour in the HIV population. As stated above, with an increasing life expectancy the prevalence of Kaposi’s sarcoma is increasing.

Four clinical variants are described:5
• classic/sporadic/Mediterranean
• endemic African
• organ transplant associated
• epidemic (AIDS related).

There exists a theory of multiple infectious events. Kaposi’s disease has been shown to be related to Kaposi’s sarcoma-related herpesvirus (KSHV) or herpesvirus type 8.6

The diagnosis is primarily clinical, with characteristic lesions that are not painful and usually located on the extremities. Lymphoedema occurs secondarily to the obstruction of the lymphatics. A Zimbabwean study revealed that most patients present in the late stages of AIDS (stages 3 and 4).

A histological diagnosis should be made before initiating therapy, but it bleeds. However, it should be borne in mind that the disease is not curable and one should aim to:
• alleviate symptoms
• reduce the tumour mass
• reduce the oedema
• prevent disease progression.

Treatment can be broadly grouped into two groups:

Treatment targeting lymphoedema:
• elevation of the limb
• exercise
• multilayer bandages
• compression stockings
• manual lymphatic drainage.

Treatment targeting the disease:
• radiation
• antiretroviral therapy
• chemotherapy – this may suppress an already depressed immune system.
transmission is more prevalent than vice versa. In anal intercourse the insertive partner is less likely to contract HIV than the receptive partner.

Male genital manifestations of HIV infection
HIV can present in different forms in the male urogenital tract. The different manifestations are summarised in Table I.

Sexually transmitted infections
Genital herpes simplex virus (HSV)
HSV types 1 and 2 are very common in HIV-infected men. The course of the infection may be prolonged and intravenous acyclovir may be necessary to cure the lesions. Patients with acyclovir-resistant HSV have been described and need treatment with foscarnet or topical cidofovir gel.

Human papillomavirus (HPV)
Warts (condylomata acuminata) are found on the penis, urethra, scrotum and perineum. Other clinical presentations of HPV include bowenoid papulosis and epidermodysplasia verruciformis. Men with extensive penile warts should be screened for HIV.

Syphilis
There is a high prevalence of syphilis in HIV-infected populations, especially homosexual men. It progresses faster from secondary to tertiary syphilis in HIV-infected men, who, with early syphilis, have a high risk for the development of neurological complications and treatment failure of standard regimens.

Chancroid
Chancroid, caused by Haemophilus ducreyi, is a co-factor for HIV transmission. Chancroid in HIV-infected individuals may be resistant to standard regimens. Ulcers heal more slowly and prolonged courses of treatment may be needed.

Molluscum contagiosum
It is caused by a sexually transmitted pox virus and is found in 10 - 20% of AIDS patients, most often on the face and genital areas. The lesions can become very large and widespread in AIDS patients. HIV-infected patients with molluscum contagiosum usually have a CD4 count of less than 250 cells/µl. Molluscum contagiosum lesions may simulate more serious infections, such as cutaneous pneumocytosis, histoplasmosis and cryptococcosis, and should be confirmed by biopsy.
HIV-related genito-urinary tract infections

Prostatitis
It is found in up to 8% of HIV patients and presents as acute prostatitis. It can be associated with superimposed urinary tract infections.

Epididymitis and orchitis
In autopsy studies 39% of AIDS patients have signs of opportunistic tests infections. Infections usually cause atrophy of the testes with spermatogenetic arrest and depletion of Leydig's cells. Immuno compromised patients can also develop epididymitis caused by atypical organisms such as Candida and cytomegalovirus.

Necrotising fasciitis (Fournier's gangrene)
It may be the presenting condition in previously undiagnosed AIDS patients. All patients with this life-threatening infection should be screened for HIV.

Neoplasms
Kaposi's sarcoma (KS)
Two types of KS are described, i.e. the classic type, which occurs in patients with lymphoma or immune deficiencies, and the epidemic type, which is associated with AIDS. KS can affect any skin area, including the male genitalia. A new herpesvirus, human herpesvirus 8, is associated with all cases of KS. An experienced observer can easily diagnose the typical purple indurated plaques, but the diagnosis needs to be confirmed by biopsy.

Non-Hodgkin's lymphoma (NHL)
Patients with NHL usually have widespread disease at presentation and genito-urinary sites may be involved primarily or secondarily. Since the introduction of antiretroviral therapy the incidence of KS has decreased and therefore NHL is the most common AIDS-associated malignancy in patients on therapy. The common head and neck problems in HIV-positive patients, Johlene Du Plessis, MB ChB, MMed (Chir)
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Approximately 41 - 68% of all HIV-infected patients will present with pathological conditions of the head and neck at some point in the disease. Common head and neck problems in HIV-positive patients are briefly discussed under the following headings:

Ulcers, plaques and gum disease
• ulcers, plaques and gum disease
• neoplastic growths in and around the mouth
• lumps and bumps.

Ulcers
Recurrent oral ulcerations classify the patient as having stage II disease according to the WHO staging of HIV/AIDS. Causes for these ulcers include viruses (herpes simplex virus (HSV), varicella zoster (VZ) and cytomegalovirus (CMV)), immunological causes (aphthous ulcers) and fungal infections (candida, histoplasmosis).

HSV and VZ (shingles) most often involve the perioral skin, but can also involve the oral mucosa. The typical prodrome precedes the vesicular eruption. It is very important to remember that if the tip of the nose is involved in shingles, the patient must be evaluated and monitored for possible involvement of the eye (corneal/conjunctival ulcerations) as this can lead to serious problems if left undiagnosed. CMV and aphthous ulcers can look very similar to VZ, but lack the typical prodrome. Leukoplakia and erythroplakia
Oral hairy leukoplakia (Fig. 1) presents as a painless, white, slightly elevated plaque that is not easily removable (as is the case with candida). It has a hairy appearance and is most commonly located on the lateral border of the tongue. It can also involve the ventral tongue and in rare cases the buccal mucosa. Patients are usually asymptomatic and lesions can only be observed. If a lesion should show change, however, a biopsy should be done.

Candida albicans
Candida can present in 4 different forms:
• pseudomembranous candidiasis/thrush (most common form)
• erythromatous candidiasis (red lesions on palate/dorsum of tongue)
• angular cheilitis (red, flaking lesions at corners of mouth)
• hyperplastic candidiasis (thick white plaques on mucosa) (Fig. 3).
mg daily PO for 14 days is used if oesophageal candida is diagnosed.

**Acute necrotising ulcerative gingivitis/periodontitis**

This is an acute opportunistic infection of the gingiva that can spread to the underlying alveolar bone. Causative organisms include *Treponema* spp, *Selenomonas* spp, *Prevotella* intermedia, *Borrelia* spp, beta-haemolytic group B streptococci and *Candida albicans*. Patients present with painful, bleeding gums with varying amounts of necrotic tissue. Treatment consists of meticulous oral hygiene, debridement of necrotic tissue and local and systemic antibiotics. Oral antiseptics such as Glycothymol mouth wash can be used as adjunct therapy.

**Neoplastic growths in the mouth**

**Human papillomavirus infection**

These lesions present as condylomata acuminata, warts or focal epithelial hyperplasia. Treatment consists of simple surgical excision; biopsies can be done first if there is doubt about the diagnosis.

**Kaposi's sarcoma**

Kaposi's sarcoma usually presents on the skin, but the hard palate, gingiva, buccal mucosa and the dorsum of the tongue can also be involved (Fig. 4). Lesions present as red to purple plaques which can also ulcerate and cause pain and bleeding. Most of the time, mucosal lesions will accompany cutaneous lesions. Treatment consists of triple antiretroviral therapy and external beam radiation in collaboration with an oncologist.

**Lymphoma**

Lymphoma can present in the oral cavity as a swelling or mass, but is extremely rare and usually associated with a poor prognosis.

**Squamous cell carcinoma**

Squamous cell carcinoma will present as in HIV-negative patients and the treatment approach will be the same.

**Molluscum contagiosum**

These lesions are characterised by flesh-coloured, dome-shaped, smooth or umbilicated papules and are caused by a DNA poxvirus. They are found commonly on the lips, buccal mucosa and palate (Fig. 5). Treatment of these lesions consists of cryotherapy or excision.

**Lumps and bumps**

**Lymphadenopathy**

HIV-related lymphadenopathy is discussed by WJ Jacobs.

**Parotid enlargement**

Unexplained persistent parotid enlargement is also a stage II-defining disease according to WHO staging of HIV/AIDS. Patients complain of mildly tender, soft parotid swelling that can be unilateral or bilateral (Fig. 6). The pathophysiology behind this phenomenon is a diffuse lymphoid infiltrate, hence the name diffuse infiltrative lymphocytosis syndrome or DILS.

### Table I. Diagnosis and treatment of HIV-associated head and neck lesions

<table>
<thead>
<tr>
<th>Disease</th>
<th>Clinical appearance</th>
<th>Diagnosis</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candida</td>
<td>See text for different clinical types</td>
<td>Clinical picture +/- culture/biopsy</td>
<td>• Topical antifungal treatment</td>
</tr>
<tr>
<td>Periodontal disease</td>
<td>Halitosis, bleeding gums, severe pain in gums</td>
<td>Clinical picture</td>
<td>• Systemic anti-fungal treatment for oesophageal candida</td>
</tr>
<tr>
<td>Oral hairy leukoplakia</td>
<td>White lesion lateral aspect of tongue, non-removable</td>
<td>Clinical picture +/- tissue biopsy</td>
<td>• Aggressive plaque removal and debridement by dentist</td>
</tr>
<tr>
<td>Herpes virus ulcers</td>
<td>Painful solitary/multiple vesicular lesions, may erode/coalcsce</td>
<td>Clinical picture +/- smears +/- viral culture +/- biopsy</td>
<td>• Topical or systemic antibiotics</td>
</tr>
<tr>
<td>Recurrent aphthous ulcers</td>
<td>Painful, well-circumscribed, shallow ulcers</td>
<td>Clinical picture +/-biopsy</td>
<td>• Good oral hygiene</td>
</tr>
<tr>
<td>Kaposi's sarcoma</td>
<td>Painless red, bluish/purple maculae, papules/nodules</td>
<td>Clinical picture +/-biopsy</td>
<td>• Observation!</td>
</tr>
<tr>
<td>Non-Hodgkin's lymphoma</td>
<td>Firm, painless focal swelling or poorly defined alveolar mass</td>
<td>Biopsy</td>
<td>• Biopsy indicated if there is change in lesion's appearance</td>
</tr>
<tr>
<td>DILS</td>
<td>Painless uni/bilateral parotid swelling</td>
<td>Sonar, FNA</td>
<td>• In severe cases consider oral aciclovir</td>
</tr>
<tr>
<td>Molluscum contagiosum</td>
<td>Flesh-coloured, dome-shaped, smooth or umbilicated papules</td>
<td>Clinical picture</td>
<td>• Oral aciclovir</td>
</tr>
</tbody>
</table>

...
There are numerous head and neck conditions that are commonly seen in HIV-positive patients, not all requiring surgical referral.

The diagnosis of DILS can be made by ultrasound/CT, which will show multiple cystic lesions throughout the superficial and deep lobes of the parotid gland. It has been described as having a Swiss cheese appearance on imaging. FNA of this lesion will reveal a benign lymphoepithelial cyst. Please do not do true-cut biopsies! You don't want the pathology report to come back reporting 'pieces of normal facial nerve!' Management of these lesions is not surgical and consists of antiretroviral therapy and/or external beam radiation.

The diagnosis and treatment of HIV-associated head and neck diseases are summarised in Table I.

Conclusion
There are numerous head and neck conditions that are commonly seen in HIV-positive patients, not all requiring surgical referral. It is very important that primary care physicians recognise these conditions and know when to refer them and when they can be managed at primary care level.

References available at www.cmej.org.za

The paediatric surgeon and HIV
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Children form a large proportion of the HIV-positive population. A portion of infected infants progress rapidly to full-blown AIDS and die within 1 year without treatment.likewise, Kaposi’s sarcoma is treated very successfully in children. One should remember that antiretroviral therapy is the first line of treatment, combined with other forms of ablation, such as cryotherapy and radiation, as well as different regimens of chemotherapy.

Other malignancies. Incidental findings of seropositivity and even AIDS are found among our patients with different forms of malignancy. These patients are put on antiretroviral therapy at the time that they start chemotherapy. Care should be taken, because these patients are known to develop immune reconstitution inflammatory syndrome (IRIS).

References available at www.cmej.org.za
The impact of HIV/AIDS on orthopaedic surgery

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As is the case with other surgical disciplines, orthopaedic surgery was dramatically influenced by the HIV pandemic. Early experience, reporting complication rates of 140% and mortality rates of 55 - 70%, led to a pessimistic approach to surgery in HIV-positive patients. The initial perception was that these patients were prone to:

• poor wound healing
• high postoperative complication rates
• a protracted postoperative period
• higher mortality rates.

These early studies were skewed by the fact that these procedures were usually performed as emergencies presenting as direct consequence of HIV infection. Later follow-up work changed this perspective dramatically. The success of antiretroviral therapy has further led to a change in the initial approach to the HIV positive.

The impact of HIV can be divided into orthopaedic diseases caused by HIV/AIDS and problems or potential complications with orthopaedic surgery and implants in HIV-positive patients.

HIV-related orthopaedic diseases

Osteonecrosis, osteopenia, and osteoporosis are conditions seen increasingly in patients with HIV. These conditions may be caused by the infection itself, antiretroviral therapy or lipodystrophy.

Osteopenia and osteoporosis

A change in bone turnover occurs in HIV-positive patients. This is probably because of deregulation between osteoclast and osteoblast function. Studies reported increased cytokine levels (IL, TNF) and increased osteoprotegerin levels, leading to deregulation of RANKL-RANK interaction.

Antiretroviral therapy (specifically protease inhibitors) and lipodystrophy caused by antiretrovirals have also been linked to bone loss, but conclusive evidence remains lacking.

The incidence of osteopenia is 14 - 84% and that of osteoporosis is up to 45% in HIV patients, but no difference in fracture rates could be demonstrated between patients receiving and not receiving a protease inhibitor regimen.

Approach to emergency and elective orthopaedic surgery

When dealing with an HIV-positive patient presenting with an orthopaedic complaint, one has to distinguish between the asymptomatic seropositive patient and the symptomatic AIDS patient. Staging the disease, using the WHO staging system based on CD4 counts and clinical information, led to a more aggressive surgical approach in these patients. Furthermore, the surgical approach is dictated by whether it is an emergency or an elective case.

Trauma

Polytrauma

HIV is reported as a significant prognostic indicator for an adverse outcome in acute lung injury and adult respiratory distress syndrome in ICU patients. It is also reasonable to expect higher secondary infection rates in HIV-positive patients.

Closed fracture

Initial unrefined studies reported infection rates as high as 24 - 40%, but recent studies showed infection rates of 3.5% in patients with CD4 counts as low as 200 cells/µl.

Open fracture

Depending on the level of contamination, infection rates as high as 42% can be expected compared with 11% in controls. Open tibia fractures are particularly prone to deep chronic infection and the use of external fixation seems well advised.

Fracture union

A higher incidence of HIV was found in patients presenting with delayed union or non-union, but this seems to respond to stable internal fixation and autologous bone grafting.

Late sepsis

An increased risk of late sepsis was found in trauma and arthroplasty patients and removal of instrumentation may be indicated as the disease advances. These late infections can be due to reactivation of latent bacteria or may be because of late haematogenous seeding.

The approach to trauma should be guided by the merits of the patient’s injury, and the established priorities of early and adequate debridement and fracture stabilisation still hold. It might be worthwhile to prefer external immobilisation when dealing with open fractures in HIV-positive patients, especially open tibia fractures.

Elective surgery

Arthroplasty

Most of the research in this regard was on HIV-positive haemophilic patients. A large retrospective multicentre study found an increased rate of deep sepsis – as high as 18.7%. These patients are also at risk for bleeding in and around their joints as well as bacteraemia associated with regular factor transfusions. Both of these factors may contribute to late sepsis. HIV-positive haemophilic patients may not be a true reflection of the infection risk associated with HIV-positive arthroplasty. HIV-positive non-haemophilic patients may also have a need for arthroplasty because of a higher incidence of avascular necrosis. The risk of sepsis seems to be lower in these patients, but literature remains lacking.

Spinal surgery

Young and others found no major complications on a small series of HIV positive patients undergoing spinal surgery. The mean CD4 count in these patients was 279 cells/µl.

Other implants and elective surgery

A prolonged course of prophylactic antibiotics (10 days of cefuroxime) was found to be beneficial in WHO group A patients. Otherwise elective surgery seems to be safe in HIV-positive patients.

Wound healing

Harrison et al. found that in the absence of preoperative wound contamination, there is no higher incidence of wound infection regardless of the CD4 count. Buehrer confirmed this in a similar HIV-positive haemophilic study. The same was found by Harrison et al., but they also found a more rapid progression to AIDS in patients who had a lower CD4 count.

Conclusion

The initial nihilistic surgical approach to HIV-positive patients seems to have been too cautious. With a few exceptions, most orthopaedic surgery can be undertaken safely in these patients, provided that one adheres to proper surgical principles.

References available at www.cmej.org.za
Vascular disease in HIV/AIDS

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References


HIV-related anal disease

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References


Soft-tissue tumours and HIV/AIDS

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References


Male genital disease in HIV and AIDS

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References


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References

1. Gurney TA, Murr AH. Otolaryngologic manifestations of human immunodeficiency
The paediatric surgeon and HIV

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References

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References