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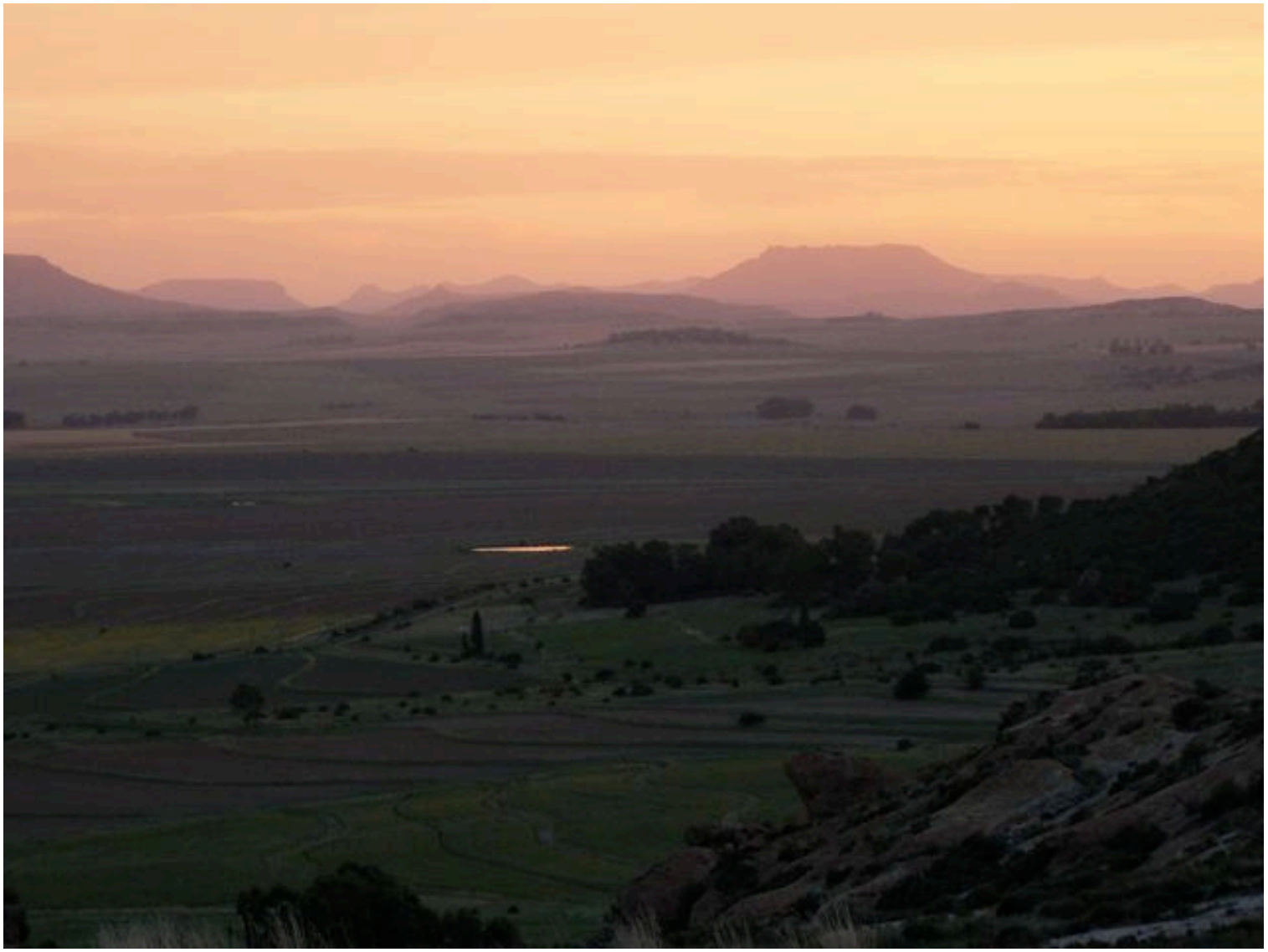

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A u g u s t

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Instructions for Authors

The Lesotho Medical Association Journal accepts editorials, original research papers, review papers, case discussions, clinical guidelines, letters and Lesotho medical news reviews.

The author should submit both an electronic and hard copy of the manuscript to the address below:

Lesotho Medical Association Journal
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From the President's Pen

Dear colleagues,

I take this opportunity to greet you once again in the name of God Almighty, the Creator of the heavens and the earth and of us to whom He gave dominion over the works of His mighty hands.

In His infinite wisdom God created us in His own image. He created us as one body, an assembly of different parts that play uniquely different roles but in a unified and integrated manner as to be able to serve that one body in living up to its purpose of creation. Albeit the unique differences in their divinely assigned roles, colleagues, let us remember that all parts of the body are dependent on each other to be able to function properly.

This interdependence, as ordinary as it may seem, is the great secret behind the body's survival and its success in doing what it is created for. The medical fraternity, like every other unit of our societal structure, is a body composed on individual parts, which are none other than you and I. It has a responsibility to mankind and society, the responsibility of looking after and taking care of the body of man as God created it.

As parts of this body, we have individual roles to play, roles that are determined by our individual talents, specialties and abilities as bestowed on us as flag bearers of this noble profession to which we belong. Like the parts of the body, our success in playing these roles use fully depends on how successful we are in integrating our individual talents and abilities through interdependent coexistence built on mutual respect and trust.

Colleagues, at this point and as I had similarly done on some occasions in the past, I preach to you unity. Its goes without saying that "united we stand, divided we fall". Surely, whatever gains we make in advancing the course of our Association in meeting its objectives and that of the fraternity at large depends on how united we are. Disunity blinds our understanding and ability to make critical and objective assessment of situations. It deprives us of love and indeed serves as a harbinger to strife.

I would like to report here and congratulate the Executive committee for the significant strides the LMA made in its current efforts to help build a sustainable human resource capacity for the nation's health sector. These efforts, it must be noted, are in keeping with the Association's strategies towards achieving its primary goal of bringing health to the doorsteps of our people. Together with the Ministry of Health and also LEBOHA, the Nursing Council, the Nursing Association and the Lesotho Medical, Dental and Pharmacy Council, we have put in place a working machinery that currently is working arduously towards a successful implementation of this project.

We have good support from the international community in these drives. In this aspect, I am

glad to mention that an international coordinator has been assigned to the project and funds have also been made available for it's financing. We extend our very sincere gratitude to our donors.

Talking about bringing health to the doorsteps of our people let me also mention a real challenge we face today as healthcare providers in the service of our dear country – the challenge of combating antimicrobial resistance to common anti-infective agents at our disposal in treating infectious diseases. This was the main theme of this year's well-patronized World Health Day Commemorations, which was held in Berea.

The Honourable Minister of Health's reaction to the main presentation of the day which brought home to the audience research findings on the state of the problem was that of a plea soliciting our concerted involvement in finding solutions to the problem. The dangers of antimicrobial resistance cannot be over emphasized. It is a major health risk that undermines and compromises our efforts to defeat ill health in our societies. The LMA has taken a serious note of the situation and I would want at this point to assure the Honourable Minister of our total commitment to finding solutions to the problem.

The AGM of the LMA convenes at the end of July to elect a new leadership. I take this opportunity to invite all members of the fraternity to this all-important meeting.

Thank you and God be with you.

Dr C.K. Hoedoafia (President – LMA, 2011)

Wake Up Call

M MOKETE, MD

A time for recollection; a time for reflection; a time for resolution; a time for redoubling efforts for action because, as we noted in the last editorial, "time and tide wait for no man."

The Chinese aptly put it that if you want to invest in a nation: train women because they effectively pass on the information to the children they conceive, bear, and spend more time bringing them up than the fathers.

When educated women are empowered; they have the capacity to acquire information and hence to be able to beat poverty and hunger – a number one millennium goal. Educated and empowered women can face HIV/AIDS with sufficient determination and armamentarium to prevent infection, survive, teach and support all those who or may be vulnerable. Thus successfully tackling Millennium Goal 6 (HIV/AIDS).

The Chinese, further, say that if you want to invest for one year: plant corn; if you want to invest for ten years: plant trees; but if you want to invest for a hundred years or more, train people which tallies very well with Millennium goal 2 (universal education).

Concerted and redoubled efforts for training with objectives in mind can succeed to achieve all the millennium goals including care of the environment (Goal 7) and worthwhile partnerships (Goal 8) with the 1st world initiatives and maximum use of all human capital.

Again, governments and all international organizations are urged not to shift the 2015 goal posts one more time but wake up and throw in all efforts to achieve millennium goals which affect all of us in one way or another. We will not achieve the goals if we bury our heads in the sand ostrich-style and stay within our various comfort zones.

Ultrasound Evaluation of Uterine Fibroid

Dr M Rahman, MBBS, DMRD

Introduction

Fibroid or Leiomyoma are the most common tumours in 20% to 30% of women over 30 years of age and in 75% of hysterectomy specimen. It arises from overgrowth of smooth muscles and connective tissue in the uterus. A clonal aberration commonly in chromosome 7 and 12 in the origin of tumours.

Estrogen and Progesterone receptors play an important role in the growth of such tumours. Elevated estrogen levels may cause fibroid enlargement during the first trimester of pregnancy. It is shown that 15% to 30% of fibroids may enlarge then shrink in puerperium. Fibroids also shrink after menopause. Sometimes re-growth may occur with hormonal therapy. The tumours are responsible for at least one third of all gynecologic admissions to hospitals and are found in the general population in about one in four women in active reproductive life. Uterine fibroid are rare before the age of 20 years of age but can be found. The symptoms are more common between 35 to 45 year olds. Fibroids even when extensive may be asymptomatic. The most important symptoms produced by submucosal fibroids are abnormal uterine bleeding. Compression on the urinary bladder may produce urinary frequency, sudden pain, disruption of blood supply and infertility. Myomas in pregnant women increases the frequency of spontaneous abortion, fetal malpresentation, uterine inertia and postpartum haemorrhage. Malignant transformation (Leiomyosarcoma) is extremely rare. Ultrasound has rapidly become established as a very important modality for tomographic imaging of soft tissue. The soft tissue images are obtained without the need for contrast agents and as far as is known, ultrasound used at diagnostic intensity level does not cause damage to tissue. The resolution capabilities of ultrasounds provide excellent image differentiation of the uterus and the ovaries.

The preferred imaging modality for the evaluation of uterine fibroids is both a transabdominal and transvaginal ultrasound. It is an efficient technique for an objective assessment of the size, location and consistency of abdominal and pelvic findings. This combined information form the bimanual pelvic examination, and allows for a more complete assessment of the patient. Transabdominal ultrasonography provides a panoramic view and easily understood orientation of relative structures. Most Leiomyomas occur in fundus and body of the uterus and only 5% in the cervix. They may be solitary, and multiple in 98% of cases or they may be diffuse. Most fibroids (95%) are intramural, they are located in the middle of the myometrium. Submucosal or exophytic fibroids are located in the sub serosal layer and tend to cause a focal bulge in the exterior surface of the uterus. They can become pedunculated. Submucosal or subendometrial fibroids are the least common.

Fibroid of the uterus generally cause some uterine enlargement unless they persist in an atrophic postmenopausal uterus, in which case the overall size may be within normal limits. The uterine enlargement produced by fibroid may be generalized, resulting in a smooth globular control; however a uterus with fibroid will have a lobulated contour. They may be some distortion or displacement of the endometrial echocomplex. The most frequent pera abdominal

USG appearance of fibroid is concentric, solid, hyperechoic mass. These solid masses absorb sound waves and therefore, cause a variable amount of acoustic shadowing. Fibroids may vary in three degrees of echogenicity, they can be either homogenous or hyperechoic, depending on the amount of fibrous tissue and calcification. Fibroids may have anechoic components resulting from necrosis. The aim of this study was to see the pattern of fibroid uteri in our population.

Patients and Methods

The prospective study was carried out on 100 patients, consecutively and cross-sectionally aged 21-60 years, which appeared to have a suspicious case of uterine fibroid. Patients were collected from the outpatient department and the Department of Obstetrics and Gynaecology of the Queen Elizabeth II hospital as well as from the district hospitals and private practices from January 2010 to April 2011.

All the information and records were kept confidentially in our register book. Chief complaints and findings of Physical examination were recorded. Consecutively cross-selected cases were diagnosed clinically as well as by ultrasound, then operation. Biopsy was done in Pathcare of the Free State. However biopsy reports were not available in our department due to the unavailability of time from across the border.

Results

One hundred patients who were diagnosed as uterine fibroid between the ages of 22 to 60 years and maximum patients were between 31 to 45 years old. The most common presentation was bleeding found in 44 (44%) cases; pain was present in 30 (30%). Both bleeding and pain was present in 20 (20%) and anaemia in 14 (14%), primary infertility in 6 (6%) and secondary infertility in 15 (15%) patients. In this study out of 100 patients, 8 (8%) were nulliparous, 14 (14%) were primiparous and 78 (78%) were multiparous.

Table-I

Clinical presentation (n=100)

Presentation	Number	Percentage
Bleeding	44	44%
Pain	30	30%
Bleeding and Pain	20	20%
Anaemia	14	14%
Infertility (primary)	6	6%
Infertility (secondary)	15	15%

Ultrasonographical appearance of fibroid described as hypo, hyper and mixed echopattern--- hypoechoic found in 67 (67%) cases, hyperechoic in 18 (18%) cases and mixed echotexure in 15 (15%) cases. Single fibroid was found in 42 (42%) cases and multiple fibroids were found in 58 (58%) cases. In this study 90 (90%) cases were intra-mural, 7 (7%) cases are subserous and 3 (3%) cases were detected as submucosal fibroid.

Table-II

Types of fibroid detected by transabdominal ultrasonography according to their various sites (n=100)

Types	Number	Percentage
Intramural	90	90%
Sub serous	7	7%
Sub mucosal	3	3%

Table-III
Trans-abdominal ultrasound appearance of fibroid uteri (n=100)

USG Echopattern	Number	Percentage
Hypoechoic	67	67%
Hyperechoic	18	18%
Mixed	15	15%

Discussion

In the study the highest incidence of uterine fibroid was found between the ages of 31 to 45 years. Tindal (1994) found uterine fibroid between the ages of 35 to 45 years. Heather (1994) found peak incidence of fibroid after the age of 35 years. These two authors findings were close to the study's observation. In Bangladesh, Khan (1997) found in a study of fifty cases peak incidence of uterine fibroid between the age of 31 to 40 years. In the same Institute of Post Graduate Medicine and Research, Dhaka, Noor (1995) observed symptomatic uterine fibroid between the ages of 35 to 45 years. S.N. Mostafa (2005) in IPGM& R Dhaka observed peak incidence of fibroids in women between 30 to 44 years of age in her 46 case study. These three studies are also close to my findings.

In this study uterine fibroid was found 78% in multiparous, 14% in primiparous and 8% in nulliparous. These findings are almost similar to the other studies in Bangladesh, Noor (1995) studied 50 cases of fibroid in IPGM&R Dhaka, among them 10% nulliparous, 8% primiparous and 82% multiparous. Khan (1997) in a similar study of 50 cases of uterine fibroid in IPGM& R Dhaka, observed 14% nulliparous, 10% primiparous and 70% multiparous.

My study showing multiple uterine fibroids was in 58 cases (58%) and single in 42 cases (42%). Fibroids are usually multiple but up to 20% of small fibroids may not be demonstrated by ultrasound. Gross et al. (1983) found that ultrasounds fail to detect 22% of uterine fibroid, particularly those smaller than 2mm. Failure to detect smaller fibroids by transabdominal ultrasnography could be the most logical explanation in early time, however recent high resolution transvaginal ultrasound reduces the error in ultrasound study in the detection of multiple fibroids.

The majority of uterine fibroids are intramural or subserosal. Only 5% are submucosal. Callen (1983) found intramural fibroids are the most common. Tindal (1994) also observed intramural fibroid are most common. In my study intramural fibroid were found in 90% of cases, subserous in 7% and submucosal in 3% of cases. S.N. Mostafa (2005) found in her 46 case study- Intramural 92.9%, Subserosal 4.8% and Sub mucosal 2.4%. My findings are similar to their observation.

Alteration of texture depends on the presence or absence of degenerative/necrotic changes and the type of degeneration. Necrosis in the fibroid undergoes cystic change. Fatty degeneration and calcification increases the echogenicity and calcification can cause acoustic shadows. In my study of 100 cases – 67 cases (67%) were hypoechoic, 18 (18%) were hyperechoic and 15 (15%) were mixed in echopattern. Persaud V and Arjoor PD (1997) studied echopattern in uterine fibroid are found 72.32% hypoechoic, 16.92% hyperechoic and 10-76% of mixed echopattern, which is closer to my study. Heather (1994) found most of the fibroids were hypoechoic ultrasonographically.

Conclusion

In spite of newer diagnostic modalities, ultrasonography remains the basic imaging modality for the evaluation of uterine fibroid, as it is a safe, non-invasive, easily available, cost effective technique. This can provide valuable information regarding uterine fibroid and can act as an excellent diagnostic tool.

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Approaching Cutaneous Adverse Drug Reactions

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Introduction

Adverse drug reactions (ADR) are common and pose a major health problem to patients and drain significant resources. They are divided into 2 types. Type A ADR, which are predictable, dose dependent and are the most common; accounting for 80% of all cases. Type B ADR are unpredictable, dose independent and comprise 15–20% of all adverse drug reaction. These include immunologically mediated drug hypersensitivity or non-immune mediated idiosyncratic reactions(1). A cutaneous adverse drug reaction (CADR) is defined as any undesirable change in the structure or function of the skin, its appendages or mucous membranes as a result of drug use and it encompass all adverse events related to drug eruption, regardless of the aetiology. The reaction can either be confined to the skin only or be part of a multisystem disorder. In this review the focus will be on type B or idiosyncratic cutaneous reactions.

It is estimated that drug hypersensitivity reactions are ~ 100 times more common in HIV-infected persons(2). This makes CADR an increasing management problem for clinicians particularly in high HIV-prevalence settings like ours. The true incidence of cutaneous adverse drug reactions (CADR) is unknown due to variable presentation, differences in populations, variations in prescribing patterns, inaccurate reporting and limitations in case definition. The associated morbidity and mortality also remain poorly defined but it can be significant.

General Measures in Managing CADR

It has been well established that early recognition and withdrawal of the offending drugs improves outcomes in the management of severe cutaneous adverse drug reactions(3). Thus, it is important for the diagnosis to be made timeously and causality be assigned so that the offending drug(s) can be removed from the treatment regimen, particularly in situations where there are limited effective drugs e.g. antituberculous drugs. It has also been shown that early referral to a specialized unit improves outcomes in severe CADR(4).

To make a correct diagnosis of CADR, there are important points to be emphasized. A good clinical history is crucial in establishing the diagnosis of a possible CADR. The history should include all prescribed medications, including self- prescriptions, over the counter preparations, homeopathic preparations, herbal products and traditional preparations. The history taking should also aim to establish a temporal relationship between the initiation of the drug(s) and the development of CADR as well as treatment interruptions, responses to drug withdrawal and rechallenge. This information together with the known side-effect profile of the drug(s) will help in identifying the offending drug(5).

A well-directed and thorough clinical examination will frequently provide an accurate diagnosis. It is crucial to remember that the skin reacts in a limited number of ways to different insults including infections and inflammatory conditions. It is thus important to consider other aetiologies for the eruption when making

a diagnosis. The clinician should describe the morphology and distribution of the eruption and a complete evaluation of the skin and its appendages, including all the mucous membranes. This will help in identifying the type of CADR and formulating an appropriate management plan.

A drug can cause different types of CADR, although some drugs are more likely to cause a particular type of a reaction. A good example is a drug hypersensitivity syndrome (DHS) caused by anticonvulsants, which was previously called anticonvulsant hypersensitivity syndrome. However, anticonvulsants cause numerous types of CADR including Stevens Johnson syndrome and morbilliform eruptions(6). Thus, one should be careful in attributing causality on the basis of the association of a drug with a type of CADR and vice-versa.

The current gold standard for ascribing causality is a rechallenge with the offending drug, however this may be associated with additional and potentially fatal CADR and is best done in a specialized center under close monitoring. Rechallenge is only indicated if an essential drug, for which there is no alternative, is thought to be responsible for the reaction(5).

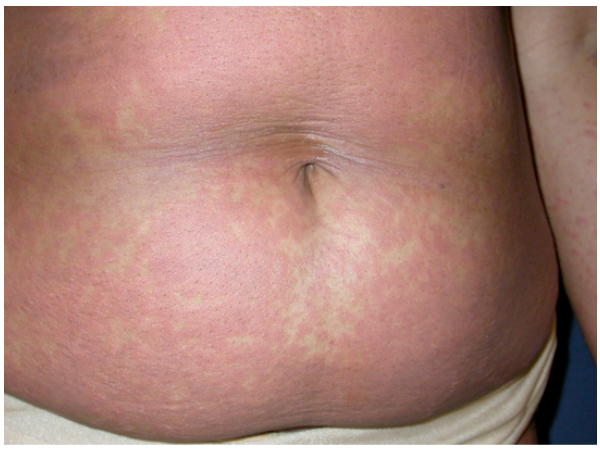
Types of CADR and Their Management Strategies

Morbilliform Drug Eruption

Morbilliform eruption or maculopapular exanthems are the most common presentation of CADR, accounting for 95% of all cases(7). The rash usually presents 7-14 days after initial exposure to the offending drug, with erythematous macules and papules spreading centrifugally. **(Figure 1)** The eruption can become confluent leading to erythroderma. Morbilliform drug eruptions are frequently self-limiting with no serious sequelae. In cases involving essential drugs it is possible to treat through the exanthem. However, maculopapular exanthem can be the initial presentation of more serious reactions such as Stevens Johnson and drug hypersensitivity syndromes so close monitoring is recommended(5). Administration of topical corticosteroid and systemic antipruritic agents is usually adequate to control the disease.

There are a wide variety of drugs that can cause maculo-papular eruption including penicillins, cephalosporin's, antiepileptic therapy, sulfonamide antibiotics and allopurinol(8).

FIGURE 1



Urticaria and Angioedema

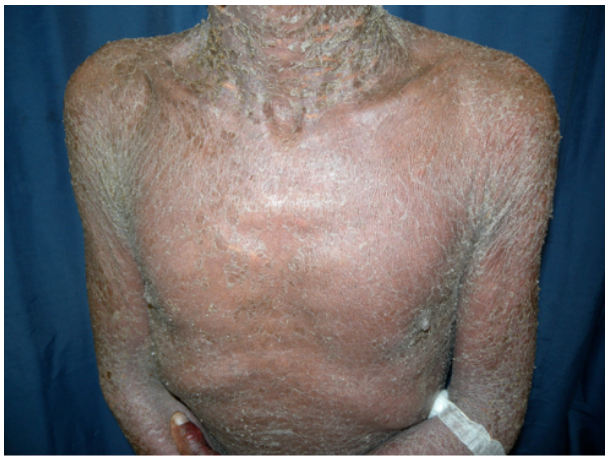
Urticaria presents as itchy erythematous wheals that develop within a few minutes after ingesting the offending drug. The lesions are migratory and transient, resolving within 24 hours. The trunk and limbs are frequently involved. The involvement of deeper dermal and subcutaneous tissues is known as angioedema. This consists of pale swelling of the face including the lips, eyelids and ears. The buccal mucosa, tongue, larynx and pharynx can be affected leading to respiratory failure. A wide variety of drugs and vaccines are incriminated with antibiotics and NSAIDs being the most common(9). Another form of angioedema to keep in mind is

ACE-inhibitor associated which can present, in addition to above features, with abdominal pain, vomiting, diarrhea, leucocytosis and ascites. ACE-inhibitor associated angioedema can present years after initiation of the drug. These patients are often extensively investigated for an acute abdomen. The symptoms resolve with withdrawal of the drug(10). Urticaria is managed with oral antihistamines. Management of angioedema is beyond the scope of this review bar to say it is a medical emergency.

Drug Hypersensitivity Syndrome

Also known as drug rash with eosinophilia and systemic symptoms (DRESS), DHS is a severe disease associated with a mortality of up to 10%. It has a latency period of more than 3 weeks on first exposure to the drug and presents with fever, oedema, lymphadenopathy, leukocyte abnormalities (particularly eosinophilia) and hepatitis. Less frequently nephritis, pancreatitis, pneumonitis and myocarditis have been described. The eruption can be urticarial or maculopapular, but vesicles, pustules, cheilitis, conjunctivitis and purpura have been described. The severity of the rash is not necessarily proportional to the extent of systemic involvement. The presence of a fever and facial oedema should increase suspicion of systemic disease. If the eruption is extensive and prolonged it is characterised by extensive scaling referred to as exfoliative dermatitis. **(Figure 2)** The clinical symptoms can persist for up to 2 weeks after withdrawal of the offending drug(11). Potent topical steroids form the first-line of management. In severe and persistent disease, particularly with associated significant hepatitis, systemic steroids are indicated.

FIGURE 2



Stevens Johnson Syndrome and Toxic Epidermal Necrolysis

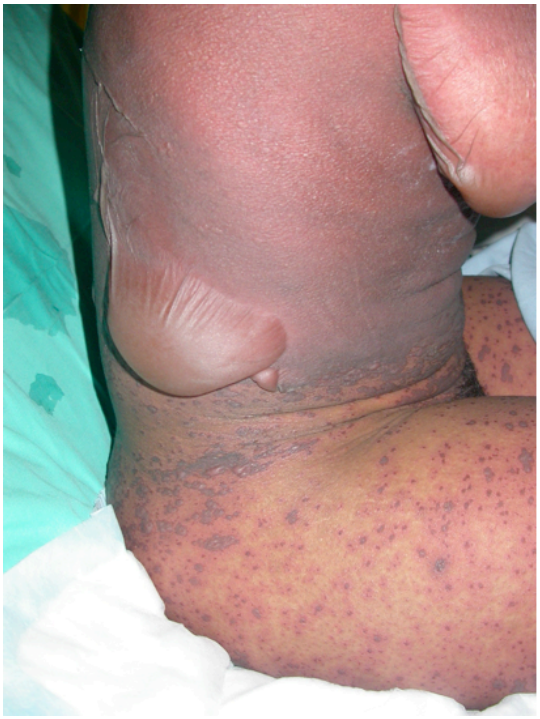
Stevens Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are considered as a spectrum of the same disease. In SJS there is <10% of epidermal detachment and in TEN there is >30%. SJS/TEN overlap lies between these two extremes(12). The early symptoms of fever, malaise, cough, stinging eyes and a sore throat are often confused with an upper respiratory tract infection. This rapidly progresses to erythematous macules and targetoid lesions, epidermal detachment and mucositis. **(Figure 3)** Early painful erythema and blisters of the palms and soles are a hallmark of SJS and TEN. TEN can be associated with mortality of up to 50%(13). Nevirapine, cotrimoxazole, antituberculous drugs and anticonvulsants are commonly implicated in our setting.

Management of SJS/TEN requires a multidisciplinary team approach including dermatologists, burns unit specialists, ICU specialists, nutritionists, ophthalmologists, microbiologists, infectious disease specialists, general physicians and a pain management team centered around a good core of experienced nurses. Adequate nutrition, fluid balance, temperature control, pain relief, eye care, prevention of mucosal adhesions and meticulous monitoring for sepsis

are crucial in preventing both short term and long-term sequelae. Debridement is discouraged as the intact necrotic skin provides a good sterile barrier to the regenerating epidermis. The use of systemic steroids is controversial(14).

Septicaemia is the most common cause of death and early use of parenteral antibiotics is recommended on signs of systemic infection. Prophylactic antibiotics are not recommended and the choice of antibiotics should be based on skin cultures until blood cultures are available to direct appropriate therapy(15).

FIGURE 3



Fixed Drug Eruption

Fixed drug eruption (FDE) presents as solitary or numerous itchy, round, well-circumscribed, erythematous macules that evolve into oedematous plaques on the skin or mucosae and resolve with persistent hyperpigmentation. **(Figure 4)** They tend to recur in exactly the same sites on re-exposure to the offending drug, with new lesions erupting elsewhere. The trunk, lips, palms, soles, glans penis, and groin are favoured sites of FDE. Occasionally the lesions can be extensive and bullous resembling Stevens Johnson syndrome and toxic epidermal necrolysis(8). **(Figure 5)** The withdrawal of the offending drug is adequate in most cases. For extensive bullous FDE the management is similar to that of SJS/TEN although extensive conjunctival and oral involvement is infrequent.

FIGURE 4

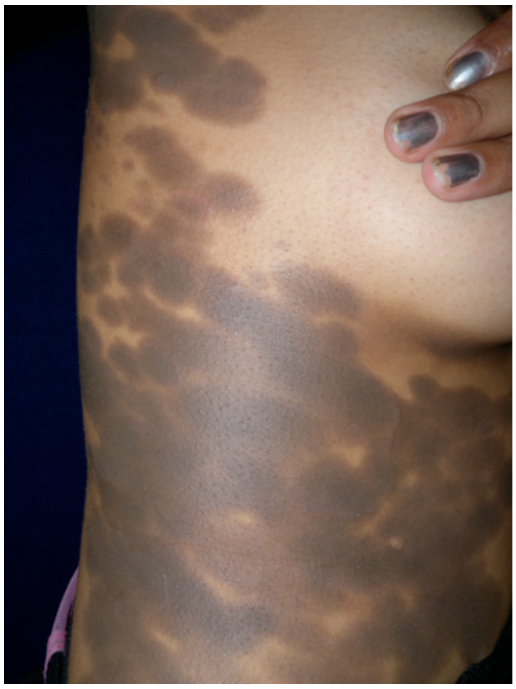
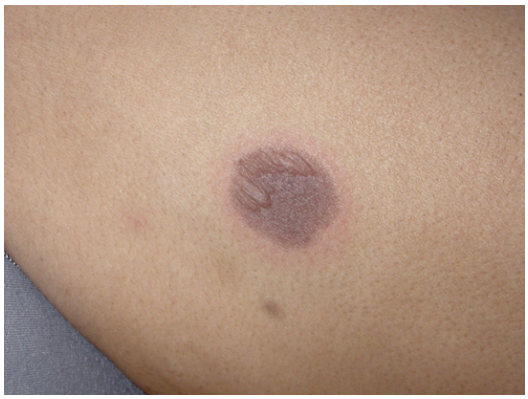


FIGURE 5



Lichenoid Drug Eruption

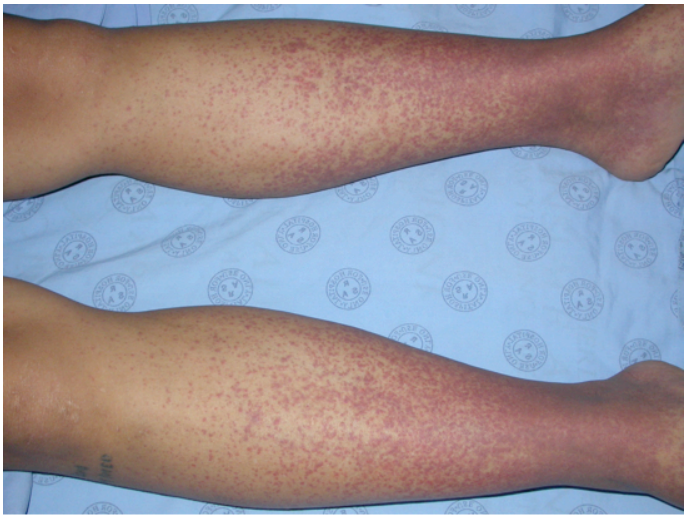
Lichenoid drug eruption (LDE) presents as itchy pink macules that gradually progress to become purplish, flat-topped, polygonal and scaly papules. Sometimes the lesions persist as

macules and increase in size with continuing exposure to the offending drug. Buccal and genital mucosae are favoured sites characterised by a white lace pattern called Wickham's striae. The latency period between initiation of the drug and the development of the lesions ranges from days to several years, with most cases occurring within a few months. The lesions usually resolve spontaneously with withdrawal of the offending drug often with post-inflammatory hyperpigmentation(8).

Drug-Induced Cutaneous Vasculitis

Drug-induced cutaneous vasculitis is characterised by palpable purpura, most frequently on the lower limbs. Depending on severity of the reaction, the purpura can progress to become haemorrhagic blisters and ulcers. An important variant is urticarial vasculitis, which presents like urticaria initially but the lesions are non-migratory, last for more than 24 hours and resolve with post-inflammatory hyperpigmentation. As with all cutaneous vasculitides, internal organ involvement should be excluded as these can be life threatening(16). General measures used to care for open wounds are usually adequate to promote healing of the skin lesions. (**Figure 6)**

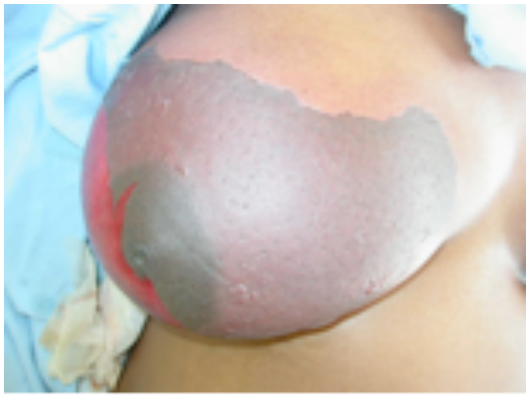
FIGURE 6



Anticoagulant-Associated Skin Necrosis

Anticoagulant-associated skin necrosis occurs in 1 in 10 000 patients exposed to warfarin. The lesions typically start 3–5 days after initiation of therapy as red, painful plaques evolving to necrosis, haemorrhagic blisters and ulcers. This is due to occlusion of skin and subcutaneous vessels by thrombus. The condition is associated with genetic deficiency of protein C leading to a transient hypercoagulable state after initiating warfarin therapy. The management is withdrawal of warfarin and replacement with heparin followed by administration of vitamin K(8). **(Figure 7)**

FIGURE 7



Acute Generalized Exanthematous Pustulosis

Acute generalized exanthematous pustulosis (AGEP) is characterised by a fever of >38°C, neutrophilia and small mostly non-follicular pustules on a background of oedematous erythema. These pustules are found in the main body folds (neck, axillae, groins), trunk and upper extremities. The eruption is associated with a burning or itching sensation. Sometimes oedema of the face and hands, purpura, and vesicles are found. The interval between drug administration and the onset of the rash is usually less than 2 days. The eruption lasts for 1–2 weeks and is followed by a superficial desquamation. Penicillins and antimalarials are the most commonly implicated drugs. With-

drawal of the offending, topical corticosteroids and oral antipruritic agents are the mainstay of management(17).

Conclusion

Severe CADR poses a significant challenge to clinicians. This review provides a basic approach to diagnose CADR, clinical features of the most common and important variants as well as a concise management approach to each type.

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Malignant Melanoma

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Introduction

Malignant Melanoma is an increasingly common invasive malignant epidermal tumour of Melanocytes with significant metastatic potential.

In 2005: an estimated 60,000 new cases of melanoma was diagnosed and almost 8,000 people died of the disease. It is the 5th most common malignancy for men, and 6th for women. It represents < 5% of skin cancers but accounts for 65% of deaths. The median age of diagnosis is 48.

10/100,000 people in the UK , and 42/100,0000 people in Australia have malignant melanoma. Malignant Melanoma is mostly cutaneous but it also occurs in the mucous membrane of the nose, mouth, anus, conjunctiva, choroid and pigmented layer of the retina. It is rare in the coloured race, but increasing among white people.

Lesotho suffers from some of lowest rates of Malignant melanoma but it is important for doctors to have an aptitude to discover Malignant Melanoma from other skin malignancies.

Clinical Types

There are several different clinical types of malignant melanoma, these include:

1. Superficial spreading melanoma
2. Nodular melanoma
3. Acral lentiginous melanoma
4. Lentigo malign melanoma

Phases of Growth

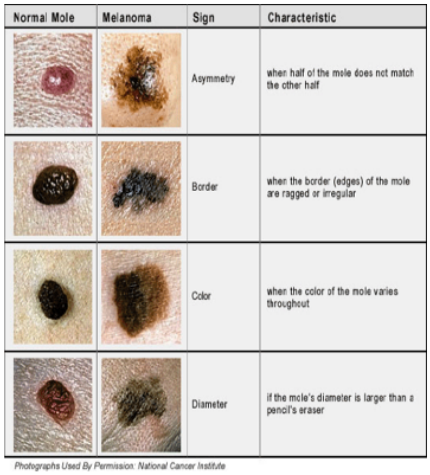
The phases of growth include:

1. Radial Growth Phase: tumour cells proliferate at the dermal-epidermal junction. Tumour expands radially
 – Lesions are confined to epidermis, may have superficial involvement of dermis
2. Vertical Growth Phase- lesion invades deeper into dermis, development of palpable nodule
 – *Nodular type of melanoma has only VGP

Typical Presentation

Malignant melanoma presents itself as a pigmented skin lesion that has recently changed. The lesion usually enlarges radially, then becomes raised. It also has irregular borders and variegated colour (pink- blue- black). Advanced lesions present themselves with ulceration or itching/bleeding including invasion into the cutaneous nerve plexus/superficial capillary bed.

Work-up of Suspicious



Photographs Used By Permission: National Cancer Institute

Pigmented Lesion

1. Biopsy- full-thickness excisional biopsy with 1-3 mm margins. Perform a full-thick incisional or punch biopsy for palm, face, subungual, etc.
2. Orient biopsy with definitive treatment in mind
3. Pathology: Breslow thickness, ulceration, Clark level, margin status (deep and peripheral), satellite lesions
4. After melanoma diagnosis is confirmed perform an H&P, lymph node exam, then complete a skin exam.
5. Family history of skin cancer, melanoma.

Clark's Levels for Melanoma

I: Epidermis basement membrane intact
II: Papillary dermis thru basement membrane
III: Functional dermis between papillary and reticular dermis
IV: Reticular dermis
V: Fat

Metastasis

Malignant melanoma can metastasize to the lungs (hematogenous spread), liver, brain, bone, GI, and adrenals. Melanoma most commonly metastasizes to the small bowel. If the tumor is >1.0 mm thick (Stage IB) perform a SLN biopsy. If the tumor is <1.0 mm thick with Clark's level IV or V, ulcerated or demonstrates regression perform a physical examination and staging evaluation.

Treatment:

Treatment of malignant melanoma includes excision of margin depending on the maximum tumour thickness.

Carcinoma in situ	5mm margin
0.1 -1.5 mm thick (pT1-2)	10mm margin
1.6 -4 mm thick (pT3)	10-20 mm margin
> 4mm thick (pT4)	20-30 mm margin

Histological very wide excision of margins (up to 5cm)

If SLN is positive (micro-metastatic melanoma), perform a completion lymphadenectomy of the regional node (10-30% have additional positive nodes)

Prognosis:

This relates most closely with Breslow thickness, nodal involvement and metastasis

Tumour Thickness	Approx 10 years survival
<0.76	>95%
1.5 - 2.5	70%
4 - 7.99	50%
>8	30%

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The Management of Benign Prostatic Hyperplasia

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Clinical Scenario

A 64 year-old man complains of 12 months of reduced urinary flow and nocturia, which is disrupting his sleep. He is worried about prostate cancer. Apart from hypertension he is fit and well without any other significant medical history.

Clinical Evaluation

History

A focused urological history is mandatory. This can be divided into voiding symptoms (degree of hesitancy, flow, straining and post micturition dribbling) and storage symptoms (urgency, urge incontinence and frequency and nocturia). In addition to this it is important to enquire about whether the patient has any history of haematuria or dysuria and also about any previous urological interventions or instrumentation.

A commonly used and useful tool for the assessment of BPH is the American Association of Urology/International Prostate Symptom Score (AUA/IPSS). This is a validated, reproducible and simple method of helping to classify the patient's symptoms into mild, moderate and severe groups[1]. It can be repeated at intervals to allow monitoring of the patients symptoms over time. Figure 1 shows the form that the patient has to fill in. The scores are then added together. The patient's symptoms can then be classified into mild, moderate or severe as shown at the bottom of the chart.

In addition to the score, there is a quality of life question at the end which is useful in the assessment of the impact of the symptoms on the patient. (See Figure 1).

Clinical Examination and Tests

A general medical examination should be carried out including evaluation of the bladder, kidneys and external genitalia. Rectal examination allows the assessment of the prostate consistency (smooth, firm, craggy) and an estimate of the size of the prostate.

Ideally every patient should have an evaluation of the flow rate by voiding into a flow meter and an ultrasound evaluation of the urine residual following voiding. Peak urinary flow >15 mls per second means the patient is unlikely to have significant outflow obstruction, 10-15ml/sec is in the equivocal range and if the peak flow is consistently below 10 ml/sec the patient is likely to have significant outflow obstruction. Two or more flow tests of greater than 150mls are required to make this assessment.

Ultrasound residual measurement of greater than 150mls on at least two occasions is predictive of less favourable outcomes of treatment.

Laboratory Investigations

Urinalysis is mandatory to exclude abnormalities such as infection, haematuria, and glycosuria. An assessment of Urea and Creatinine should also be made.

The Role of PSA

Many of the men such as the patient in the scenario who present with lower urinary tract symptoms are worried about prostate cancer and as a result part of the evaluation of a man with suspected BPH almost always includes assessment for prostate cancer first by clinical means but also with the help of Prostate Specific Antigen.

PSA is a protein produced exclusively by the prostate, which can be detected in the blood stream. It can be raised in a number of conditions including prostate cancer, urinary infection, and following instrumentation such as cystoscopy and prostate biopsy. A "normal" rectal examination without undue manipulation for the prostate is not associated with an increase in the prostate specific antigen. PSA is used to screen for Prostate cancer but is not itself diagnostic. It has specificity of 93.2% and a sensitivity of 72.1% and a positive predictive value of 25.1%. [2]

In recent years age-specific PSA has become widely available in an attempt to increase the accuracy of the test.[3] An example of the reference ranges are shown below.

The reference ranges may differ slightly depending on the laboratory doing the test.

TABLE 1

Age Range	PSA
50-59	<3ng / ml
60-69	<4ng / ml
>70	<5ng / ml

Before the PSA test is undertaken the patient must be appraised of its limitations. Perhaps the most important of these is that an elevated PSA below 10ng/ml is still only associated with cancer in about one in five men. The higher the PSA, however, the higher the likelihood of cancer being found.

Medical Management of Prostate Cancer

Watchful Waiting

Many men seek consultation in order to make sure nothing serious is going on. In our case the man was worried about cancer of the prostate. If the patient's symptoms are tolerable without recourse to intervention and there are no worrying signs such as a high residual urine volume, it is safe to simply reassure the patient and monitor his progress over time. Ball et al followed up 107 men with symptoms of prostatic obstruction in whom there was no clinical indication for prostatectomy and found that after 5 years only 2 had developed retention and 10 had needed prostatectomy. 97 had not required treatment.[4]
Another study, comparing surgery with watchful waiting, showed that if the patient does not wish to have surgery, a conservative approach could be safely adopted.[5]

Alpha Blockers

In the 1970's phenoxybenzamine was the first alpha-blocker that gained wide use in the treatment of BPH.[6] It was shown for the first time that Alpha-blockers could be used to improve the flow rate and thus bladder outflow symptoms. Phenoxybenzamine had both alpha 1 and alpha 2 activity and as a result had significant cardiovascular side effects.

Selective Alpha 1 blockers such as doxazosin, tamsulosin and alfuzosin were then developed and are the mainstay of the management of bladder outflow obstruction today. These

drugs work by the blockade of alpha-receptors in the smooth muscle elements of the bladder neck and prostate thus reducing intra-urethral pressure and improving urine flow. They have been shown to be generally safe although they can still have significant but reversible side effects[7, 8]. The main side effects are dizziness due to hypotension and ejaculatory dysfunction (mainly in the form of retrograde ejaculation). Other side effects include syncope, headache, asthenia and peripheral oedema. Overall, Alpha 1 blockers produce an improvement in symptoms and flow rate in most patients[7].

5 Alpha Reductase Inhibitors

Men castrated before puberty do not develop BPH. This is because BPH is mediated in part by the action of testosterone. The most important form of testosterone in relation to BPH is dihydrotestosterone, which is made by the action of the enzyme 5-alpha reductase on testosterone.[9]

5 alpha reductase inhibitors such as Finasteride and Dutasteride block the enzyme and thus reduce dihydrotestosterone levels. This promotes the reduction of prostate size of up to 24% and thus helps to reduce outflow resistance and improve urine flow. These drugs have their greatest effect on moderately enlarged and large prostate glands. They have also been shown to reduce the incidence of acute urinary retention and decrease the number of men who end up needing surgery.[9, 10] The two drugs have been shown in a randomised double blind study to be equivalent in their efficacy.[11]

Their main disadvantage is the fact that unlike alpha-blockers, it takes up to 12 weeks to realise the maximal effects of both finasteride and dutasteride. They also cause a reduction in libido in up to 5.4% of men and impotence in up to 15% although these effects are reversible upon stopping the medication.[12]

Combination Therapy

The combination of alpha-blockers and 5 alpha reductase inhibitors has been shown to have additional benefits in the treatment of BPH. The Medical therapy of Prostatic Symptoms research group (MTOPS) have shown that combination therapy with doxazosin and finasteride reduced the risk of progression of the disease than either drug alone.[13] Similar results have been shown with other combinations.[10, 14]

Surgical Management of BPH

In men who have been unable to tolerate or failed medical treatment (for example due to unacceptable side effects) or those who have experienced the complications of BPH such as urinary retention, surgical treatment remains a good option. There are numerous options all with varying merits.

Trans-Urethral Resection of the Prostate (TURP) & Open Prostatectomy

TURP remains the "gold-standard" treatment for BPH when compared to other surgical methods. It does however have significant side effects and complications. These include bleeding, infection, incontinence, and sexual dysfunction (retrograde ejaculation, impotence). The patient is also usually required to stay in hospital for 2-3 days.

Open prostatectomy has largely fallen by the wayside in modern medical practice except in patients with very large prostates (in excess of 100 grams). It carries significantly higher risks of bleeding, infection and incontinence when compared to TURP. It also, being an open operation, results in a longer hospital stay post op

and a longer period of recovery following discharge.

Laser Treatment of BPH

In recent years Laser treatment of the prostate has become popular. There are two main methods - ablation and enucleation of the prostate.[15]

Laser ablation of the prostate involves the application of laser energy to the tissue of the prostate to vapourise it thus creating a channel in a similar way to TURP. The most commonly used laser for this method is the KTP or "Greenlight laser". The main advantages over TURP and other methods is a short hospital stay (often less than one day), and a reduction in bleeding.[15] It is however associated with greater costs although these are to some extent offset by the shorter hospital stay. Short-term results are good but long-term data is yet to emerge.

Another method is that of laser enucleation of the prostate, which again results in the formation of a cavity similar to that created by conventional TURP. The most commonly used laser for this is the Holmium-YAG laser. It also has the advantages of a shorter hospital stay and fewer problems with bleeding.[16] Unlike the KTP laser the Holmium laser allows the retrieval of a specimen for pathology. Both the KTP and Holmium lasers have no limitation of the size of prostate that can be operated on which is an advantage over conventional TURP.[15, 16]

Other methods

Apart form the above various methods of achieving the same result have been developed and include the use of prostatic stents, balloon dilation of the prostate and thermotherapy. The results of these treatments have not been sufficiently good for them get wide acceptance.

Conclusion

In the case of the patient mentioned at the beginning of this short paper, given that he is otherwise fit, any of the above methods of treatment could apply. It is important to guide the patient through the process of evaluation and treatment. The end-point could be as simple as reassurance that benign prostatic hyperplasia is part of the normal process of ageing or medication or even surgical intervention.

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FIGURE 1: International Prostate Symptom Score (IPSS)

Name: Date:

	Not at all	Less than 1 time in 5	Less than half the time	About half the time	More than half the time	Almost always	Your Score
Incomplete emptying Over the past month, how often have you had a sensation of not emptying your bladder completely after you finish urinating?	0	1	2	3	4	5	
Frequency Over the past month, how often have you had to urinate again less than two hours after you finished urinating?	0	1	2	3	4	5	
Intermittency Over the past month, how often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency Over the last month, how difficult have you found it to postpone urination?	0	1	2	3	4	5	
Weak stream Over the past month, how often have you had a weak urinary stream?	0	1	2	3	4	5	
Straining Over the past month, how often have you had to push or strain to begin urination?	0	1	2	3	4	5	
Total IPSS Score							

Quality of life due to urinary symptoms	**Delighted**	**Pleased**	**Mostly satisfied**	**Mixed about equally satisfied and dissatisfied**	**Mostly dissatisfied**	**Unhappy**	**Terrible**
If you were to spend the rest of your life with your urinary condition the way it is now, how would you feel about that?	0	1	2	3	4	5	6
Total score: 0-7 Mildly symptomatic; 8-19 moderately symptomatic; 20-35 severely symptomatic.							

DADD Summer School Lesotho

Dr. Raute Molise

DAAD Summer School 2010 was held in Lesotho at Mmelesi Lodge Thaba-Bosiu and was organized by the Lesotho Medical Association (LMA). Managing HIV in a primary Health Care Setting was the selected course, which was divided into different topics. The Summer School program started on 11th October 2010 and successfully ended on 23rd October 2010. Twenty-one participants from different countries attended this course, they were:

Anthony Enimil, Johannes Jochum, Raymond Yurika, Mbu Tabenyang, Peter Kishimbo, Yunus Mbage, Lucienn,
Nguelejack, Fuad al-Sabri,
Judith Minimeka, Lawrence Watket,
Melese Abede, Ludmilla Hatman,
Michall Schklyanka, Claudia Circher,
Chechenyeva Vera, Sr Diane Fortuine, Sr Irima Kruger, Sr Juanita Mc Laughlin, Dr Daniel Nemachema, Sr M. Mphana, and Dr Nyambuwa. Lecturers came from Lesotho, South Africa, Germany and the United Kingdom.

From Lesotho:

Dr Malikotsi Metsing,
Dr Lineo Thahane,
Dr Mathias Adorka,
Mr Sepetla Tlaitlai,
Dr Tlali Mpholo,
Dr Ravi Gupta,
Mr Ingo Seifert,
Dr A Tiam,
Dr Titi Mohapi,
Dr M. Mokete

From South Africa:

Prof. Jean Nachega,
Dr. Amy Slogrove,
Ms Nocawe Frans,
Dr Jantjie Taljard,
Prof. Rosenkranz,
Dr Steyn Petrus.

From Germany:

Dr Tessa Lennenemann,
Dr Christoph Konigs,
Dr Makase Nyaphisi,
and Dr Anette Heberi.

Selected Topics

HIV transmission, ART paediatric, choice of regimen, adherence and continuation of care, drug resistance and treatment failure, PMTCT, pharmacology, treatment update, HIV transmission in a hospital setting, disclosure in children, defaulters, HIV in hepatitis, adherence in children, Lesotho health referral system, alternative approaches to HIV management, Thaba-Tšoeu Clinic visit, Karabong Clinic visit, oral

issues, ophthalmic issues, surgical issues, HIV / TB co-Infection and case presentations.

Summer School 2010 was organised at a mammoth level in Lesotho. It will ever be a mile benchmark for LMA members. The Summer School provided the opportunity for professionals to share their views on the management of the above topics in different countries of varying cultural backgrounds.

All LMA members worked day and night to make it successful. Thanks to Dr Makase Nyaphisi who helped us and was physically present. Hopefully future courses will further improve our knowledge and skills.

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