NATIONAL CLINICAL GUIDELINES

THE DIAGNOSIS & MANAGEMENT OF SKIN & SOFT TISSUE INFECTION

Ministry of Public Health

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Abbreviations

The abbreviations used in this guideline are as follows:

ART	Antiretroviral Therapy
BIT	Burrow Ink Test
CA-MRSA	Community-Associated Methicillin-Resistant Staphylococcus aureus
СТ	Computed Tomography
EPSD	Epidermal Parasitic Skin Diseases
FGSI	Fournier's Gangrene Severity Index
HIV	Human Immunodeficiency Virus
HrCLM	Hookworm-Related Cutaneous Larva Migrans
LRINEC	Laboratory Risk Indicator for Necrotising Fasciitis
MMR	Measles-Mumps-Rubella
MCV	Molluscum Contagiosum Virus
MMRV	Measles-Mumps-Rubella-Varicella
MRI	Magnetic Resonance Imaging
MRSA	Methicillin-Resistant Staphylococcus aureus
MSSA	Methicillin-Sensitive Staphylococcus aureus
NSAID	Non-Steroidal Anti-Inflammatory Drug
S. aureus	Staphylococcus aureus
S. pyogenes	Streptococcus pyogenes
SSTIs	Skin and Soft-Tissue Infections
VZV	Varicella Zoster Virus

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1 Information about this Guideline

1.1 Objective and Purpose of the Guideline

The purpose of this guideline is to define the appropriate diagnosis and management of skin and soft tissue infections in adults and children. The objective is to guide the appropriate investigation, treatment and referral of patients presenting to provider organisations in Qatar. It is intended that the guideline will be used primarily by healthcare professionals in both primary care and specialist settings.

1.2 Scope of the Guideline

This guideline covers the following aspects of care:

- General recommendations on clinical presentation, assessment, and investigation of skin and soft tissue infections (SSTIs).
- General principles of care and antimicrobial prescribing for SSTIs.
- Criteria for emergency and non-emergency referral to specialist care.
- Specific features and management of abscesses, folliculitis, furunculosis, carbunculosis, impetigo, cellulitis and erysipelas, animal and human bites, diabetic foot infections, infected burns, necrotising SSTIs, and viral SSTIs.

1.3 Editorial Approach

This guideline document has been developed and issued by the Ministry of Public Health of Qatar (MOPH), through a process which aligns with international best practice in guideline development and localisation. The guideline will be reviewed on a regular basis and updated to incorporate comments and feedback from stakeholders across Qatar.

The editorial methodology, used to develop this guideline, has involved the following critical steps:

- Extensive literature search for well-reputed, published evidence relating to the topic.
- Critical appraisal of the literature.
- Development of a draft summary guideline.
- Review of the summary guideline with a Guideline Development Group, comprised of practising healthcare professionals, subject matter experts and patient representatives, from across Qatar.
- Independent review of the guideline by the National Clinical Guidelines & Pathways Committee, appointed by the MOPH, from amongst stakeholder organisations across Qatar.

Whilst the MOPH has sponsored the development of the guideline, the MOPH has not influenced the specific recommendations made within it.

1.4 Sources of Evidence

The professional literature published in the English language has been systematically queried using specially developed, customised, and tested search strings. Search strategies are developed to allow efficient yet comprehensive analysis of relevant publications for a given topic and to maximise retrieval of articles with certain desired characteristics pertinent to a guideline.

For each guideline, all retrieved publications have been individually reviewed by a clinical editor and assessed in terms of quality, utility, and relevance. Preference is given to publications that:

- 1. Are designed with rigorous scientific methodology.
- 2. Are published in higher-quality journals (i.e. journals that are read and cited most often within their field).
- 3. Address an aspect of specific importance to the guideline in question.

Further information about the literature search and appraisal process is included in the appendix.

1.5 Evidence Grading and Recommendations

Recommendations made within this guideline are supported by evidence from the medical literature and where possible the most authoritative sources have been used in the development of this guideline. In order to provide insight into the evidence basis for each recommendation, the following evidence hierarchy has been used to grade the level of authoritativeness of the evidence used, where recommendations have been made within this guideline.

Where the recommendations of international guidelines have been adopted, the evidence grading is assigned to the underlying evidence used by the international guideline. Where more than one source has been cited, the evidence grading relates to the highest level of evidence cited:

- Level 1 (L1):
 - Meta-analyses.
 - Randomised controlled trials with meta-analysis.
 - Randomised controlled trials.
 - Systematic reviews.
- Level 2 (L2):
 - Observational studies, examples include:
 - Cohort studies with statistical adjustment for potential confounders.
 - Cohort studies without adjustment.
 - Case series with historical or literature controls.
 - Uncontrolled case series.
 - Statements in published articles or textbooks.
- Level 3 (L3):
 - Expert opinion.
 - Unpublished data, examples include:
 - Large database analyses.
 - Written protocols or outcomes reports from large practices.

In order to give additional insight into the reasoning underlying certain recommendations and the strength of recommendation, the following recommendation grading has been used, where recommendations are made:

- Recommendation Grade A (RGA): Evidence demonstrates at least moderate certainty of at least moderate net benefit.
- **Recommendation Grade B (RGB):** Evidence is insufficient, conflicting, or poor and demonstrates an incomplete assessment of net benefit vs harm; additional research is recommended.
- Recommendation Grade C (RGC): Evidence demonstrates potential harm that outweighs benefit; additional research is recommended.
- **Recommendation of the GDG (R-GDG):** Recommended best practice based on the clinical experience of the Guideline Development Group members.

1.6 Guideline Development Group Members

The following table lists members of the Guideline Development Group (GDG) nominated by their respective organisations and the Clinical Governance Group. The GDG members have reviewed and provided feedback on the draft guideline relating to the topic. Each member has completed a declaration of conflicts of interest, which has been reviewed and retained by the MOPH.

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1.7 National Clinical Guidelines & Pathways Committee members

The following table lists members of the National Clinical Guidelines & Pathways Committee (NCGPC), appointed by the MOPH. The NCGPC members have reviewed and provided their feedback and approval of the guideline document. Each member has completed a declaration of conflicts of interest, which has been reviewed and retained by the MOPH.

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1.8 Responsibilities of Healthcare Professionals

This guideline has been issued by the MOPH to define how care should be provided in Qatar. It is based upon a comprehensive assessment of the evidence as well as its applicability to the national context of Qatar. Healthcare professionals are expected to take this guidance into account when exercising their clinical judgement in the care of patients presenting to them.

The guidance does not override individual professional responsibility to take decisions which are appropriate to the circumstances of the patient concerned. Such decisions should be made in consultation with the patient, their guardians, or carers and should consider the individual risks and benefits of any intervention that is contemplated in the patient's care.

2 Skin & Soft Tissue Infection - Pathway

Click on a box below to see the relevant page of the Pathway.



3 Key Recommendations of the Guideline

The key recommendations of this guideline are:

Examination (Section 5.3):

- All patients suspected for SSTI should be examined for¹:
 - Sepsis syndrome.
 - Systemic signs of infection (e.g. fever, raised white blood cell count).
 - Systemic toxaemia.
 - Life-threatening conditions.
 - Other medical emergency conditions (see *Section 7.3*).

Investigation (Section 5.4):

- Laboratory investigations are not always required for the diagnosis but they may be ordered in case of^{1,5,10} [L1, RGA]:
 - Unclear symptoms.
 - Atypical presentations.
 - Damage estimation.
 - Possibility of an alternative diagnosis.
 - o Differentiation between similar infections that require different management.
- Consider the following options:
 - Blood tests for^{2,4,13–16}:
 - Complete blood count.
 - Inflammatory markers.
 - Glucose levels.
 - HBA_{1C} levels.
 - Microbiological examination^{3,4,9,10,12–15}.
 - Urine sample^{12,13,17}.
 - Histological tests (e.g. if osteomyelitis is suspected)³.
 - Diagnostic imaging studies^{1,3,4,10,13,14,16}.
 - Biopsy (e.g. if leishmaniasis, mycobacterial infection, eosinophilic folliculitis or noninfective dermatosis is suspected)^{10,11}.
 - Tests for the presence of a specific virus 3,12,17 :
 - \circ Surgical exploration of the wound (for selected cases)^{5,10}.

General Principles of Management (Section 7):

- Prompt recognition and selection of appropriate treatment regimen are key factors limiting the morbidity and mortality associated with SSTIs⁵.
- Mild purulent SSTIs usually don't require any specific treatment as they often drain naturally^{3,10,13,15,18} [L1, RGA].
- Moderate purulent infections should be treated with incision and drainage^{3,10,13,18} [L1, RGA].
- Postoperative wound management and adequate nutritional support are vital for the good outcome [**R-GDG**].
- Antibiotic therapy should be initiated as soon as their necessity is recognised^{19,20} [L1]:
 - Empiric broad-spectrum therapy to cover the most likely pathogens is recommended as the initial step^{2,19,21} [L1, RGA].
 - Empiric antimicrobial therapy should be narrowed when culture tests and susceptibility results become available^{2,3,10,19,21} [L1, RGA].
- The duration of antibiotic therapy will depend upon the microorganism identified on culture and the clinical course of the patient^{1,3,10,24} [L1, RGA].

Emergency Referral (Section 7.3):

- The following conditions should be considered a medical emergency^{2,3,5,9,14,16,25,26}:
 - Rapidly progressing infection despite treatment.
 - SSTI with systemic signs of infection.
 - Signs of systemic sepsis.
 - Diagnosis of necrotising SSTIs or identification of a necrotic area.
 - $\circ~$ Infected burn wounds with the high concentrations of bacteria (>10 5 colony-forming units).
 - Other life-threatening or limb-threatening conditions (e.g. ulceration with limb ischaemia).
- Prompt surgical consultation is recommended for patients with aggressive SSTIs (e.g. necrotising fasciitis or gas gangrene)¹⁰ [L1, RGA].

Treatment of Specific Skin and Soft Tissue Infections:

- Refer to the sections below for further information on the diagnosis and management of specific SSTIs:
 - Abscesses (Section 8).
 - Folliculitis, Furunculosis and Carbunculosis (Section 9).
 - Impetigo (Section 10).
 - Cellulitis and Erysipelas (Section 11).
 - Bites (Section 12).
 - Diabetic Foot Infections (Section 13).
 - See also MOPH National Clinical Guideline on the Chronic Complications of Diabetes Mellitus.
 - Infected Burns (Section 14).
 - Necrotising Skin and Soft Tissue Infections (Section 15).
 - Viral Skin and Soft Tissue Infections, including (Section 16):
 - Measles.
 - Rubella.
 - Infectious Mononucleosis.
 - Herpes Zoster.
 - Molluscum Contagiosum.
 - Parasitic Skin Infections, including (Section 17):
 - Scabies.

0

- Pediculosis.
- Tungiasis.
- Hookworm-Related Cutaneous Larva Migration.
- Leishmaniasis.
- Loiasis.

4 Background Information

4.1 Definition

Skin and soft-tissue infections (SSTIs) include a variety of pathological conditions that result from microbial invasion of the skin and its supporting structures and involve the skin, underlying subcutaneous tissue, fascia, or muscles^{1,2}.

4.2 Classification

SSTIs are categorised by:

- Origin³:
 - Primary:

Microorganisms invade otherwise healthy skin.

- Secondary:
 - Microorganisms infect skin damaged due to underlying disease or trauma.
- Presence of purulent inflammation³:
 - Purulent infections (e.g. furuncles, carbuncles, abscesses).
 - Non-purulent infections (e.g. erysipelas, cellulitis, necrotising fasciitis).
- Severity of clinical presentation³:
 - o Mild.
 - o Moderate.
 - o Severe.
- Anatomical extent⁴:
 - o Focal.
 - Diffuse.
- Prognosis¹:
 - Uncomplicated:
 - Superficial infections (e.g. cellulitis, simple abscesses, impetigo, and furuncles).
 - Low risk for life- or limb-threatening SSTIs unless they are improperly treated.
 - Complicated:
 - Deep soft-tissue infections (e.g. necrotising infections, infected ulcers, infected burns, and major abscesses)
 - High risk for life-threatening conditions.
 - Further classified as non-necrotising and necrotising.

4.3 Prevalence

The true prevalence of SSTIs remains largely unknown as mild infections are typically self-limiting and usually do not require medical attention³.

4.4 Risk Factors

Risk factors for SSTIs include^{2,3}:

- Age (children or older adults).
- Asplenia.
- Presence of other diseases:
 - Cardiopulmonary disease.
 - o Diabetes mellitus.
 - Hepatorenal disease.

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- Obesity.
- Peripheral neuropathy.
- Lymphoedema or lymphatic insufficiency.
- Pre-existing skin diseases.
- Peripheral arteriovenous insufficiency.
- Trauma, including:
 - o Cuts.
 - Animal or human bites.
 - \circ Surgery.
- Immunosuppression:
 - Human immunodeficiency virus (HIV) infection.
 - Chemotherapy.
 - Antiretroviral therapy.
 - Disease-modifying antirheumatic drugs.
- Debility.
- Prolonged hospitalisation and long-term care.
- Long-term intravascular access.
- Dialysis (peritoneal, haemodialysis).
- Crowded living conditions.
- Poor hygiene (including sharing of personal items).
- Poor nutrition.
- Alcohol and substance abuse.
- Physical contact (e.g. sports activities).
- Occupation:
 - Health care professionals.
 - o Military personnel.

4.5 Prognosis

Necrotising SSTIs are associated with poor clinical prognosis¹. Any delay in diagnosis or treatment of these conditions correlates with a poor outcome and possible death^{1,5}. *Fournier's Gangrene Severity Index* (FGSI) may be used as a standard score to predict the prognosis of the patients^{1,6}.

In Qatar, the highest mortality among patients with necrotising fasciitis was reported among those with the infection in the chest, axilla and gluteal regions⁷. Patients with necrotising fasciitis of the perineum and genitalia had the lowest mortality.

Assessment of the complicating factors (e.g. underlying diseases) and appropriate use of antibiotics are essential to optimise outcomes of patients presenting with SSTIs^{3,4}. Among the signs that predict a poor outcome are^{5,8,9}:

- Diabetes mellitus.
- Obesity.
- Heart failure.
- Leukopenia.
- Thrombocytopenia.
- Haemolysis.
- Severe renal failure.
- Myoglobinuria.
- Immunosuppression.
- Systemic illness.
- Sepsis.
- Other.

5 Clinical Assessment

5.1 Clinical Presentation

SSTIs are diverse in their clinical presentations³. Clinical features of common SSTIs are listed in each of the sections that relate to them.

In general:

- Mild infections present with local symptoms only, such as^{2,3,5}:
 - o Warmth.
 - o Erythema.
 - o Oedema.
 - \circ Tenderness.
 - o Pain.
- Moderate to severe infections are associated with systemic signs of infection^{3,5}:
 - Fever >38ºC.
 - Tachycardia (heart rate >100 beats/min).
 - Tachypnoea (respiratory rate >20 breaths/min).
 - Hypotension.
 - \circ White blood cells >12*10³ cells/mm³.
 - Fatigue.
 - Nausea or vomiting.

5.2 History

Important points to elicit include the following^{3,5,10–14}:

- Age of the patient.
- Presence of long term conditions (e.g. diabetes mellitus).
- History of pre-existing skin diseases.
- Recent trauma (e.g. wounds and bites).
- Recent surgery.
- Previous antimicrobial therapy.
- Immunisation status.
- Allergies if known.
- Recurrent hospital admissions.
- Lifestyle and hobbies.

Additional points in the history to elicit relating to specific infections are provided in the sections relating to those infections.

5.3 Examination

All patients suspected for SSTI should be examined for¹:

- Sepsis syndrome.
- Systemic signs of infection (e.g. fever, raised white blood cell count).
- Systemic toxaemia.
- Life-threatening conditions.
- Other medical emergency conditions (see Section 7.3).

If none of the above are detected, a physical examination should be performed, including^{1,3,10} [L1]:

• Examination of the wound.

- Screening for the local signs of infection such as:
 - Heat.
 - o Pain.
 - \circ Swelling.
 - \circ Erythema.
 - \circ Tenderness.
 - \circ $\,$ Delayed healing.
 - Discharge from the wound site.
 - Presence of an abnormal smell (e.g. foul smell in Fournier's gangrene).
- Presence of damage to surrounding or underlying structures.
- Testing for pain or loss of sensation.
- Presence of discoloration.
- Presence of crepitus.
- Examination of lymph nodes.
- Screening for Nikolsky sign (i.e., epithelial desquamation resulting from slight pressure or rubbing the skin or oral mucosa).
- Screening for insect bites, abrasions, surgical wounds.
- Screening for obesity or malnutrition.

Particular points in the examination of specific infections, are provided in the sections relating to those infections.

6 Investigation

Laboratory investigations are not always required for the diagnosis but they may be ordered in case of^{1,5,10} [**L1, RGA**]:

- Unclear symptoms.
- Atypical presentations.
- Damage estimation.
- Possibility of an alternative diagnosis.
- Differentiation between similar infections that require different management.

Consider the following options:

- Blood tests for^{2,4,13–16}:
 - $\circ \quad \text{Complete blood count.}$
 - o Inflammatory markers.
 - o Glucose levels.
 - HBA_{1C} levels.
- Microbiological examination^{3,4,9,10,12–15}:
 - Open wound tissue specimens.
 - Purulent wounds pus culture.
 - Ulcerated skin tissue biopsy or curettage.
 - Non-open wounds needle aspiration.
 - Sepsis syndrome or severe infection blood culture.
 - o Gram stain.
 - Throat swabs.
 - Tzanck smear (if herpes infection is suspected).
- Urine sample^{12,13,17}.
- Histological tests (e.g. if osteomyelitis is suspected)³.
- Diagnostic imaging studies^{1,3,4,10,13,14,16}:
 - o Ultrasound (e.g. to differentiate simple cellulitis from necrotising fasciitis).
 - Doppler vascular examination.
 - X-ray (e.g. in septic arthritis or osteomyelitis).
 - Magnetic resonance imaging (MRI) (e.g. in necrotising infections).
 - Computed tomography (CT) (e.g. in necrotising infections).
- Biopsy (e.g. if leishmaniasis, mycobacterial infection, eosinophilic folliculitis or non-infective dermatosis is suspected)^{10,11}.
- Tests for the presence of a virus^{3,12,17}:
 - Virus specific antibodies in serum (e.g. in measles).
 - Polymerase chain reaction (e.g. in varicella zoster virus, VZV).
 - o Real-time polymerase chain reaction (e.g. in measles).
 - Fluorescent antibody staining of infected cells.
 - Molecular genotyping of virus.
 - Serologic tests.
- Surgical exploration of the wound (for selected cases)^{5,10}.

7 General Principles of Management

7.1 Principles of Care

Prompt recognition and selection of appropriate treatment regimen are key factors limiting the morbidity and mortality associated with SSTIs⁵.

Mild purulent SSTIs usually don't require any specific treatment as they often drain naturally^{3,10,13,15,18} [L1, RGA]. Moderate purulent infections should be treated with incision and drainage^{3,10,13,18} [L1, RGA]. In children, minimally invasive techniques (e.g. stab incision, haemostatic rupture of septations, in-dwelling drain placement) are preferred² [L1, RGA].

Topical and/or oral antibiotic administration is usually required in purulent cases that don't resolve naturally within several days and in mild to moderate non-purulent infections^{1,3,10} [L1, RGA]. If patients do not improve (or worsen) after 48h of treatment, they should receive antibiotics against methicillin-resistant *Staphylococcus aureus* (MRSA)² [L1, RGA].

Postoperative wound management and adequate nutritional support are vital for the good outcome [**R-GDG**].

7.2 Principles of Antimicrobial Treatment

Should antibiotic treatment be or is likely to become an issue, an early microbiology culture and sensitivity of relevant specimens is essential (see *Section 6*) [**R-GDG**].

Antibiotic therapy should be initiated as soon as their necessity is recognised^{19,20} [L1]:

- Empiric broad-spectrum therapy to cover the most likely pathogens is recommended as the initial step^{2,19,21} [L1, RGA].
- Patients unresponsive to antimicrobial therapy should be re-evaluated clinically and microbiologically whenever possible ^{1,21} [L2, RGA].
- Empiric antimicrobial therapy should be narrowed when culture tests and susceptibility results become available^{2,3,10,19,21} [L1, RGA].

When selecting antibiotics for empirical antibiotic therapy, consider the following^{3,19,21,22} [L1, RGA]:

- Most likely microorganism(s) and known susceptibility patterns in Qatar.
- Localisation of infection.
- Clinical severity.
- Relevant drug interactions.
- Recent antibiotic use (past 90 days).
- Individual health conditions of the patient (e.g. allergy).
- Characteristics of the medication:
 - Antimicrobial infusion time.
 - Pharmacokinetics of the antimicrobial.
 - Potential adverse effects.

If there is uncertainty on the appropriate patient-specific antimicrobial therapy, consult an infectious disease specialist and/or clinical pharmacist for advice and guidance²³ [L1].

The duration of antibiotic therapy will depend upon the microorganism identified on culture and the clinical course of the patient^{1,3,10,24} [**L1, RGA**]. A typical duration of treatment ranges from 5 to 10 days⁸.

Therapy with intravenous antibiotics is usually required for 7-14 days for hospitalised patients² but may be extended depending on the patient's condition^{2,10} [**L1**, **RGA**]. Intravenous antimicrobials may also be continued at home under close supervision after initiation in the hospital or emergency department² [**L1**, **RGA**].

Intravenous antibiotics should be discontinued when^{2,14} [L1]:

- Drainage or debridement is completed.
- The clinical picture improves.
- The patient can tolerate oral intake.

Blood levels of aminoglycosides and vancomycin should be monitored, particularly in elderly patients ²¹ [L2, RGA].

7.3 Emergency Referral

The following conditions should be considered a medical emergency^{2,3,5,9,14,16,25,26}:

- Rapidly progressing infection despite treatment.
- SSTI with systemic signs of infection.
- Signs of systemic sepsis.
- Diagnosis of necrotising SSTIs or identification of a necrotic area.
- Infected burn wounds with the high concentrations of bacteria (>10⁵ colony-forming units).
- Other life-threatening or limb-threatening conditions (e.g. ulceration with limb ischaemia).

Prompt surgical consultation is recommended for patients with aggressive SSTIs (e.g. necrotising fasciitis or gas gangrene)¹⁰ [L1, RGA].

7.4 Referral to Specialist Care

Hospitalisation and specialist care are required if at least one of the following is present^{1,2,16,27–29}:

- Severe or complicated SSTIs.
- Bullous impetigo, particularly in infants ≤1 year of age.
- Infection near the eyes or nose (including periorbital cellulitis).
- Failed outpatient treatment.
- Patient is immunocompromised or has unstable comorbid illnesses.
- High risk of complications.
- Inability to tolerate oral medications.
- Problems with feeding.
- Infection overlying or near an indwelling medical device.
- Recurrent SSTI.
- Uncontrolled infection despite adequate outpatient antimicrobial therapy.
- MRSA colonisation.
- Uncommon pathogens are suspected.
- Need for surgical intervention under anaesthesia (including surgical debridement).

Consider a consultation with an infectious disease specialist, surgeon, dermatologist or microbiologist when required^{1,10} [L1, RGA].

Consider referral to a diabetologist if a patient is suspected for diabetes that has not been previously diagnosed³⁰ [L1, RGA].

7.5 Follow-Up

Close follow-up is necessary during and after treatment. Before discharge, patients should receive a plan for follow-up care in the outpatient setting^{31,32} [L1, RGA].

Following discharge from inpatient care, primary healthcare professionals should focus on⁹:

- Reviewing and adjusting long-term medication.
- Identifying new physical, mental, and cognitive problems.
- Educating patients and caregivers about:
 - Good personal hygiene.
 - Self-isolation (if required).
 - \circ Vaccination.

8 Abscesses

8.1 Classification and Aetiology

There are two main types of abscesses¹³ [L1]:

- Cutaneous abscess:
 - Develops within the epidermis and dermis (e.g. boils).
 - Cutaneous abscess is diagnosed if¹ [L1]:
 - Induration and erythema are limited only to a defined area of the abscess and do not extend beyond its borders; and
 - The abscess does not extend into deeper tissues; and
 - The abscess does not have a multiloculated extension.
- Deep abscess:
 - Develops inside an organ or in the spaces between organs. Examples include¹³:
 - Anorectal abscess.
 - Bartholin's cyst.
 - Dental abscess.
 - Peritonsillar abscess.
- The diagnosis of a deep abscess requires more complex analysis. Laboratory investigations should be performed in addition to symptom assessment¹³ [L1, RGA].

Abscesses are usually caused by gram-positive cocci, e.g. *Staphylococcus aureus* (*S. aureus*)^{3,14}. They may also be polymicrobial, including anaerobes.

8.2 Clinical Presentation and Assessment

The features of an abscess include^{1–3,5,13}:

- Signs and symptoms of inflammation (pain, redness, heat and swelling).
- Collection of pus with surrounding granulation.
- Openings that drain pus.
- Swollen lymph nodes.
- Possible overlying skin necrosis.
- Features attenuated in cold abscess.
- Axilla, groin, and/or perineal abscess can occur in hidradenitis suppurativa.
- Fever.

In addition to general history taking and examination, described in *Sections 5.2 and 5.3*, enquire about the following^{3,10,13}:

- Presence of underlying inflammatory condition (e.g., hidradenitis suppurativa).
- History of staphylococcal infections.
- Recent injury in the area of abscess.

8.3 Management

Small skin abscesses don't require any specific treatment^{13,18} [**L1**, **RGA**]. Incision, surgical drainage, and local wound care are sufficient treatment for large cutaneous abscesses^{1,3,10} [**L1**, **RGA**]. Consider applying warm moist compresses to facilitate pus elimination and reduce swelling^{3,13} [**L1**, **RGA**].

Systemic antibiotic therapy is not required in the absence of systemic inflammatory response syndrome^{1–}^{3,10} [L1, RGA].

For complicated abscesses, antimicrobial therapy should be based on results of wound cultures^{1,5} [L1, RGA] and administered when clinically indicated^{2,3,5,33} [L1, RGA]. Clinical indications for antimicrobial therapy include^{2,3,5,33}:

- Patient is of an extreme age (children and older adults).
- Patient has associated comorbidities or immunosuppression.
- The disease is severe or extensive.
- If the abscess is deep (e.g. intra-abdominal).
- There is associated septic phlebitis.
- There are signs and symptoms of systemic illness.
- Recurrent abscesses formation.
- Abscess is difficult to drain completely.
- Incision and drainage alone do not improve the condition.

Empirical antimicrobial regimens are provided in *Table 8.3* below. See also the notes in *Section 7.2* on general principles of antimicrobial treatment of SSTIs.

Type of Abscess	Empirical Antimicrobials	Additional Notes
Moderately Severe	Oral flucloxacillin or cephalexin.	If methicillin-sensitive <i>S. aureus</i> (MSSA) is isolated. Typical duration is 5-10 days for outpatients and 7-14 days for inpatients.
Abscess	Trimethoprim/sulfamethoxazole or doxycycline, or clindamycin.	If community-associated MRSA (CA-MRSA) is confirmed.
Severe	Intravenous cloxacillin or cefazolin, or clindamycin.	If MSSA is suspected or cultured.
Abscess	Intravenous vancomycin or daptomycin, or linezolid.	If MRSA is suspected or cultured.

Table 8.3: Empirical Antimicrobials for the Treatment of Complicated Abscesses^{10,14}.

Antimicrobials are usually ineffective without drainage^{13,14}. Consider the following options to drain deep abscesses¹³ [**L1**, **RGA**]:

- Percutaneous abscess drainage:
 - If the internal abscess is small.
- Surgical drainage:
 - If the abscess is too large to be drained with a needle.
 - If a needle is not possible to use safely in the area of the abscess.
 - The needle drainage has not been effective in removing all of the pus.

Other treatment approaches to decolonise bacteria from the body may also be considered^{3,10,13} [L1, RGA]:

- Nasal decolonisation with mupirocin (twice daily for 5 days).
- Daily washes with chlorhexidine or antiseptic soap.
- Decontamination of personal items.

If recurrent abscess occurs at a site of previous infection, the site should be inspected for local causes (e.g. pilonidal cyst, hidradenitis suppurativa, or foreign material)¹⁰ [**L1**, **RGA**]. Adult patients with recurrent abscesses that began in early childhood should be evaluated for neutrophil disorders¹⁰ [**L1**, **RGA**].

9 Folliculitis, Furunculosis, and Carbunculosis

9.1 Aetiology

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Causes of folliculitis, furunculosis and carbunculosis, include^{3,5,11,15}:

- Immunocompetent patients:
 - S. aureus.
 - \circ Candida.
 - o Dermatophytes.
 - Pseudomonas aeruginosa.
 - Malassezia furfur.
 - Immunosuppressed patients:
 - S. aureus.
 - Klebsiella.
 - Enterobacter spp.
 - Proteus spp.

9.2 Clinical Presentation and Assessment

Table 9.2(1) outlines the differentiating features of folliculitis, furunculosis, and carbunculosis.

Type of Infection	Specific Features		
Folliculitis	 Usually occur in areas with increased sweating but may affect any area of the body except the palms and soles. Presents as follicular-based papules or pustules. May be associated with pruritic skin conditions, acne, or steroid use. Pruritus, painful or painless papules or pustule. Swelling. 		
Furuncle	 Furuncles are deeper than folliculitis. Painful swollen boils. Systemic features of infection. 		
Carbuncle	 Carbuncles are larger that furuncles. Carbuncles involve skin and subcutaneous tissue. Usually occur over thicker skin of neck, back, and lateral thighs. Drain through multiple pores. Systemic features of infection. 		

Table 9.2(1): Differentiating Features of Folliculitis, Furunculosis and Carbunculosis^{1–3,5,15,18}.

In addition to general history taking and examination, described in *Sections 5.2 and 5.3*, enquire about the following¹¹:

- Prolonged use of oral antibiotics.
- Use of topical corticosteroids.
- Frequent shaving.
- Increased sweating.
- Hot tub and/or swimming pool exposure.
- Medications that increase risk of folliculitis (e.g. lithium and cyclosporine).

Consider alternative diagnoses as listed in *Table 9.2(2)*.

Folliculitis	Furunculosis and Carbunculosis	
Acne vulgaris.	Cystic acne.	
Papulopustular rosacea.	Hidradenitis suppurativa.	
 Drug-induced folliculitis. 	Cellulitis.	
Hidradenitis suppurativa.	Osteomyelitis.	
Scabies.	• Orf.	
Pseudofolliculitis barbae.	Anthrax.	
Keratosis pilaris.	Arthropod bite.	
Acne keloidalis nuchae.		

 Table 9.2(2): Differential Diagnoses for Patients with Suspected Folliculitis, Furunculosis or Carbunculosis^{11,15}.

9.3 Investigation

Histopathology is usually not required for the diagnosis¹¹ [L2]. Potassium hydroxide preparation may be used to diagnose demodex folliculitis or pityrosporum folliculitis¹¹ [L2].

If folliculitis is suspected, specify the type of infection¹¹ [L2] i.e.:

- Superficial bacterial folliculitis.
- Pityrosporum folliculitis.
- Viral folliculitis.
- Demodex folliculitis.
- Eosinophilic folliculitis.

9.4 Management

9.4.1 Folliculitis

Simple cases of folliculitis resolve spontaneously within a few days^{3,11}. In more extensive cases, incision and drainage may be considered along with administration of antibiotics^{3,11} [**L1**, **RGA**].

Demodex folliculitis should be treated with anti-parasitic agents¹¹ [L2, RGA]:

- First-choice medication is topical permethrin 5% cream.
- Second-choice treatment is antibiotic therapy (see *Table 9.4.1*).

Viral folliculitis secondary to molluscum contagiosum infection may be treated with¹¹ [L2, RGA]:

- Curettage.
- Cryotherapy.
- Topical cantharidin.

Type of SSTI	Empirical Antimicrobials	Additional Notes	
Staphylococcal folliculitis	Topical mupirocin or clindamycin.	Not recommended for children younger than 2 months.	
Gram-negative folliculitis	Ampicillin, trimethoprim- sulfamethoxazole, or ciprofloxacin.	Oral antibiotic treatment that cover pseudomonas are recommended.	
Pityrosporum folliculitis	Itraconazole or fluconazole.	Systemic therapy with oral antifungal agents is recommended.	
Viral folliculitis	Oral acyclovir, valacyclovir, or famciclovir.	If caused by infection with VZV or herpesvirus infections.	
Demodex folliculitis	Oral ivermectin or metronidazole.	Consider dual therapy ivermectin and metronidazole.	

Table 9.4.1: Empirical Antimicrobial Regimens for Treatment of Folliculitis^{2,11}.

Consider the following treatment options in patients with eosinophilic folliculitis¹¹ [L2, RGA]:

- First-choice is the antiretroviral therapy (ART) to treat the patient's underlying HIV.
 - Complimentary treatment after ART initiation may include:
 - Topical corticosteroids.
 - Antihistamines.
 - Phototherapy.
 - Itraconazole or isotretinoin.

9.4.2 Furunculosis and Carbunculosis

Small furuncles don't require any specific treatment^{3,10,15,18} [L1, RGA]. If they do not improve after 2-3 days of moist heat, a topical antimicrobial and drainage are required³ [L1, RGA]. Large furuncles and all carbuncles should be treated with incision and drainage^{3,10,15,18} [L1, RGA].

Systemic antimicrobials are not recommended for routine use^{3,10,15,18} [L1, RGB] but may be considered in patients with carbuncles¹⁸ [L1, RGA] or with evidence of systemic infection¹⁰ [L1, RGA].

Type of SSTI	Empirical Antimicrobials	Additional Notes	
Moderate to Severe cases of Furunculosis or	Oral cephalosporins (e.g. cephalexin) or flucloxacillin.		
	Clindamycin, tetracyclines, trimethoprim-sulphamethoxazole, linezolid, or glycopeptide.	If MRSA is suspected or cultured.	
Carbunculosis	Topical clindamycin or mupirocin.	Topical antibiotics may be used as adjunctive therapy. Mupirocin should be applied twice daily to the inner nares for 12 to 30 days.	

Table 9.4.2: Empirical Antimicrobial Regimens for Treatment of Furunculosis and Carbunculosis^{11,15}.

If patients have recurrent carbuncles, the following treatments may also be considered¹⁵ [L2, RGA]:

- Bathing with a benzoyl peroxide wash or antibacterial soap.
- Decolonisation of the patient's nares with mupirocin.

To prevent spread of infection, the patient should be advised on prophylactic measures¹⁸ [L1, RGA], including:

- Washing hands after touching affected areas.
- Using a separate face cloth and towel.
- Washing underwear, bed linen, and towels at a high temperature.
- Appropriate wound dressing until the wound healed completely.

10 Impetigo

10.1 Aetiology

Impetigo is typically caused by gram-positive cocci (e.g. *S. aureus*), group A Streptococcus, or a mix of staphylococci and streptococci^{2,3,5,10}.

10.2 Clinical Presentation and Assessment

The presenting features of impetigo are 1-3,5:

- Erythematous, vesicular, and pruritic lesions.
- Affects skin of the face and limbs.
- Can occur at any age but most commonly affects children.
- Associated with mild soreness and redness.
- May cause glomerulonephritis.
- Vesicles may enlarge (bullae).
- May spread to lymph nodes, bones, joints, or lungs.

It is important to differentiate between the two main types of impetigo^{3,5} [L1, RGA]:

- Non-bullous:
 - Small fluid-filled vesicles that develop into pustules, that then rupture to leave goldenyellow crusts.
- Bullous:
 - Vesicles that develop into yellow fluid-filled bullae, that then rupture to leave brown crusts.

10.3 Management

Treatment options for patients with impetigo are^{1,3,5,10,29} [L1, RGA]:

- Localised non-bullous impetigo:
 - First-choice: Hydrogen peroxide 1% cream applied 2-3 times/day for 5 days²⁹.
 - Second-choice: A short course of a topical fusidic acid 2%, mupirocin 2% or retapamulin applied 2-3 times/day for 5 days^{2,3,29}.
- Widespread non-bullous impetigo:
 - First-choice: A short course of a topical fusidic acid 2%, mupirocin 2% or retapamulin applied 2-3 times/day for 5 days^{2,3,29}. Along with topical, systemic regimen is necessary: consider cephalexin, or flucloxacillin [**R-GDG**].
 - Second-choice: Empirical antimicrobial therapy directed against MSSA (oral cloxacillin or cephalexin)^{3,5,10,29}.
 - Antimicrobial therapy using oral penicillin if the cultures reveal only streptococci¹⁰.
- Bullous impetigo:
 - Empirical antimicrobial therapy based on oral antibiotics against Gram-positive bacteria^{1,29}.
 - Consider using mesh gauze sponges or brushes and antibacterial soap⁵.
 - Soaking in soapy warm water may be used to facilitates crust removal³.
- If MRSA is suspected or cultured from swabs:
 - Use oral trimethoprim-sulfamethoxazole, doxycycline, or clindamycin^{5,10}. Check for history of sulphonamides drug allergy and contraindications (e.g., pregnancy and age restrictions) before prescribing [**R-GDG**].

Combination treatment with topical and oral antibiotics is not recommended²⁹ [L1, RGB].

Patients with impetigo, and their parents or carers if appropriate, should be advised about:

- Good hygiene measures to reduce the spread of impetigo to other areas of the body and to other people²⁹ [L1, RGA].
- Disinfection of towels and bedding⁵ [L2].
- Seeking medical help if²⁹ [L1, RGA]:
 - Symptoms worsen rapidly or significantly.
 - Infection has not improved after completing a course of treatment.

If the infection is worsening, has not improved, or has recurred after completing a course of appropriate treatment, consider the following^{5,29} [L1, RGA]:

- Sending a skin swab for microbiological testing.
- Adjusting antibiotics appropriately after receiving culture results.
- Taking a nasal swab and starting treatment for decolonisation.

11 Cellulitis and Erysipelas

11.1 Aetiology

Cellulitis is an infection of the dermis and the subcutaneous tissue¹. Erysipelas is usually referred to as a type of superficial cellulitis involving the face¹⁰.

Typical microorganisms in cellulitis infection are group A *b*-hemolytic *Streptococcus* species and *S*. *aureus*^{1,5,14}. Cellulitis and erysipelas in patients without penetrating trauma and no evidence of MRSA are typically caused by gram-positive *Streptococcus pyogenes* (*S. pyogenes*)^{1,3}.

11.2 Clinical Presentation and Assessment

The diagnosis must be supported by the clinical presentation and at least two of the four criteria should be present diagnose cellulitis¹⁶ [L2]:

- Warmth.
- Erythema.
- Oedema.
- Tenderness.

Differentiate between the two types of cellulitis^{3,16} [L1, RGA]:

- Non-purulent:
 - Without purulent drainage or exudate or associated abscess.
- Purulent:
 - Associated with purulent drainage or exudate in the absence of a drainable abscess.

Consider alternative diagnoses^{16,28}:

- Chronic venous stasis dermatitis.
- Chronic venous insufficiency.
- Necrotising fasciitis (see *Section 15*).
- Septic arthritis.
- Deep vein thrombosis.
- Inflammatory reaction to an immunisation or an insect bite.

The differentiating features of cellulitis and erysipelas are provided in the table below:

Type of Infection	Specific Features
Cellulitis (purulent, non- purulent)	 Usually present in the lower extremities. Localised over areas of skin breakdown (e.g. insect bites, abrasions, surgical wounds). Begins as a hot, red, oedematous, sharply defined eruption. Non-elevated and poorly defined margins. Signs and symptoms of infection. Regional lymphadenopathy is common. Leucocytosis may develop. Can be purulent or non-purulent. Can progress to adjacent tissue leading to an abscess, septic arthritis, or osteomyelitis.

Type of Infection	Specific Features
Erysipelas	 Bright red, tender plaque with well-demarcated margin. Distinctly raised inflamed skin. Usually occur over face, ears, or lower legs. Often preceded by flu-like symptoms. Lymphangitis or lymphadenitis. Leucocytosis. Burning pain.

 Table 11.2: Differentiating Features of Cellulitis and Erysipelas^{1-3,5}.

11.3 Management

Cellulitis and erysipelas should be managed with antimicrobial therapy as per the table below^{1,3,28} [L1, RGA]. Consider antibiotic prophylaxis in patients with recurrent cellulitis or erysipelas²⁸ [L1, RGA].

Type of infection or Prophylaxis	Empirical Antimicrobials	Additional Notes
Cellulitie er	Oral penicillin V.	First-choice antibiotics should be directed primarily against Gram-positive streptococci. Antibiotic therapy for 5 days is usually sufficient but may be extended if no improvement observed.
Erysipelas	Cloxacillin, cephalexin, or clindamycin.	If MSSA is suspected or cultured. Antibiotic therapy for 5 days is usually sufficient but may be extended if no improvement observed.
	Cefazolin or cloxacillin.	First-choice antibiotics for paediatric population. If fasciitis suspected add clindamycin.
Prophylaxis	Oral penicillin V or erythromycin. Intramuscular benzathine penicillin.	Prophylactic administration may be considered in patients with 3-4 episodes of cellulitis per year despite attempts to treat predisposing factors.

Table 11.3: Recommended Antimicrobials for Treatment and Prophylaxis of Cellulitis and Erysipelas^{3,10,16,21}.

Intravenous systemic antibiotics are recommended in patients with^{3,10} [L1, RGA]:

- Moderate cases of cellulitis with systemic signs of infection if:
 - Cellulitis associated with penetrating trauma.
 - Systemic inflammatory-response syndrome is present.
 - Infection is due to the injection drug use.
- MRSA is cultured.

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- Severe cases of cellulitis if:
 - Cellulitis is not responding to oral antibiotic therapy.
 - Patient is immunocompromised.
 - There are clinical signs of deeper infection.
 - Systemic inflammatory-response syndrome is present.

If required, consider adjuvant therapy when necessary, including:

• Non-steroidal anti-inflammatory drugs (NSAIDs) may be used to reduce inflammatory signs [R-GDG].

- Systemic corticosteroids are applicable only for patients who do not have diabetes ^{3,10} [L1, RGB]:
 Consider prednisone 40 mg daily for 7 days.
- Wound management for cellulitis with open wound.
- Compression stockings, decongestive therapy.
- Emollients may be used to keep skin hydrated.
- Immobilisation and elevation of the affected extremity may also be helpful in facilitating drainage³ [L1, RGA].

Additional investigations for underlying diseases that worsen the prognosis (e.g. diabetes mellitus or peripheral arterial occlusive disease) may be required⁴ [L2].

Treatment of predisposing factors should be provided to all patients^{3,10,28} [L1, RGA]. These include^{3,10,28}:

- Obesity.
- Oedema.
- Venous insufficiency.
- Eczema.
- Underlying cutaneous disorders.
- Fissuring, scaling, and maceration in the interdigital toe spaces.

Possible complications of cellulitis should be reviewed and evaluated³ [L1, RGA]. These include^{3,16}:

- Abscess.
- Septic arthritis.
- Osteomyelitis.
- Endocarditis.
- Bacteraemia.
- Sepsis.

12 Bites

12.1 Aetiology

Note that only 10–20% of bite wounds become infected, including¹:

- 30-50% of cat bites.
- 5-25% of dog bites.
- 20-25% of human bites.

Consider different types of bites³⁴:

- Large wild animal bites (e.g. wild dogs).
- Small wild animal bites (e.g. mice, rats).
- Large pet animal bites (e.g. dogs, cats, horses).
- Small indoor pet animal bites (e.g. hamsters, guinea pigs).
- Human bites (including a clenched fist injury).

The common pathogens associated with bite wound infections are SSTIs^{2–4,10,35}:

- Dog bites:
 - Aerobic organisms:
 - Pasteurella spp.
 - Streptococcus spp.
 - Staphylococcus spp.
 - Capnocytophaga canimorsus.
 - Anaerobic organisms:
 - Fusobacterium spp.
 - Porphyromonas spp.
 - Prevotella spp.
 - Bacteroides spp.
 - Propionobacterium spp.
 - *Peptostreptococcus* spp.
- Cat bites:
 - Aerobic organisms:
 - Pasteurella spp.
 - Streptococcus spp.
 - Staphylococcus spp.
 - Moraxella spp.
 - Anaerobic organisms:
 - Fusobacterium spp.
 - *Porphyromonas* spp.
 - Prevotella spp.
 - Bacteroides spp.
 - Propionobacterium spp.
- Human bites:
 - Aerobic organisms:
 - Fusobacterium spp.
 - *Peptostreptococcus* spp.
 - Veillonella spp.
 - Anaerobic organisms:
 - Fusobacterium spp.
 - Prevotella spp.
 - *Peptostreptococcus* spp.
 - Veillonella spp.

12.2 Clinical Presentation and Assessment

Features of an infection in a bite wound, include^{2,3,10,35}:

- Infection sets in 8-72 hours after animal bites.
- May involve tendons, tendon sheaths, bone, and joints.
- Lymphadenitis or lymphangitis.
- Fever.
- Leucocytosis.

In addition to general history taking and examination, described in *Sections 5.2 and 5.3*, enquire about the following^{3,10,35}:

- Animal contacts.
- How and when the bite occurred.
- Tetanus immunisation status.
- Immunisation history of the animal if available.
- Medical history of the human biter, if known (e.g. viral hepatitis, HIV, other transmissible diseases).

12.3 Management

Gentle but deep irrigation of the wound with sterile normal saline is recommended to remove foreign materials and pathogens^{1,3,34,35} [L1, RGA]. Irrigation under pressure should be avoided¹ [L1, RGC].

NB: Primary closure of wounds should be avoided unless the wound is on the face or neck^{3,10,35} [L1, RGC].

Universal antimicrobial prophylaxis is not recommended¹ [L1, RGB] but may be considered in some patients, e.g. in case of face or neck wounds with primary closure [R-GDG]. It is also not recommended if the patient presents \geq 24h after the bite with no clinical signs of infection¹ [L1, RGB].

Patients who are at increased risk of infection, or have immunocompromising conditions (e.g. asplenia, advanced liver disease), or implants (e.g. artificial heart valves), should receive antibiotic therapy for 3-5 days for^{1,3,10} [L1, RGB]:

- Fresh deep wounds.
- Joint, bone or tendon injuries.
- Wounds in critical bodily areas: hands, feet, areas near joints, face, genitals.

All children with bite wounds receiving antibiotic therapy should be re-evaluated within 24-48 hours to monitor for signs and symptoms of infection³⁵ [L2, RGA].

Empirical Antimicrobials	Additional Notes	
Amoxicillin/clavulanate.	First choice pre-emptive antibiotics. Antibiotic therapy for 7-10 days is usually sufficient.	
 Second-generation cephalosporins (cefuroxime, cefoxitin). 		
 Third-generation cephalosporins (ceftriaxone, cefotaxime). 		
 Fluoroquinolones (ciprofloxacin, levofloxacin, moxifloxacin). 	Alternative options of pre-emptive antibiotics. Antibiotic therapy for 7-10 days is usually sufficient.	
Carbapenems.		
Doxycycline.		
 Trimethoprim/sulfamethoxazole plus 		
clindamycin or metronidazole.		

Table 12.3(1): Recommended Antimicrobials for use in Infected Bites^{2-4,10,35}.

All patients should be considered for post-exposure prophylaxis to infectious organisms^{1–3} [L1, RGA]:

- With dog bites^{3,35}:
 - Rabies (rabies virus).
 - Tetanus (Clostridium tetani).
 - Leptospirosis (Leptospira spp.).
 - Tularaemia (Francisella tularensis).
- With cat bites^{3,35}:
 - Cat-scratch disease (Bartonella spp.).
 - Tularaemia (Francisella tularensis).
 - Sporotrichosis (*Sporothrix* spp.).
 - Rabies (rabies virus).
 - Tetanus (*Clostridium tetani*).
 - With rodent bites³⁵:
 - Rat-bite fever (*Streptobacillus moniliformis* or *Spirillium minus*).
 - Leptospirosis (Leptospira spp.)
 - Tularaemia (Francisella tularensis).
 - Tetanus (*Clostridium tetani*).
- With human bites^{1,2,35}:
 - Hepatitis B and C.
 - Herpes (herpes simplex virus).
 - Cytomegalovirus infection.
 - Syphilis (*Treponema pallidum*).
 - Tetanus (*Clostridium tetani*).
 - o HIV.

Tetanus toxoid should be administered to patients without a toxoid vaccination in the last 10 years (see *Table 12.3(2)*)¹⁰ [L1, RGA].

Possible complications should be reviewed and evaluated³⁵ [L2, RGA]. These include³⁵:

- Cellulitis (see Section 11).
- Osteomyelitis.
- Tenosynovitis.
- Tendinitis.
- Orbital cellulitis.
- Brain abscesses.

Age	Vaccination History	Clean, Minor Wounds	All Other Wounds
0-6 years	Unknown or not up-to-date on DTaP series based on age	DTaP (If DTaP is not available, OK to give Tdap)	DTaP TIG
	Up-to-date on DTaP series based on age	No indication	No indication
7-10 years	Unknown or incomplete DTaP series	Tdap and recommend catch-up vaccination	Tdap and recommend catch- up vaccination TIG
	Completed DTaP series AND <5 years since last dose	No indication	No indication
	Completed DTaP series AND ≥5 years since last dose	No indication	Td, but Tdap preferred if child is 10 years of age
11 years and older	Unknown or <3 doses of tetanus toxoid containing vaccine	Tdap and recommend catch-up vaccination	Tdap and recommend catch- up vaccination TIG
	3 or more doses of tetanus toxoid containing vaccine AND <5 years since last dose	No indication	No indication
	3 or more doses of tetanus toxoid containing vaccine AND 5-10 years since last dose	No indication	Tdap preferred (if not yet received) or Td
	3 or more doses of tetanus toxoid containing vaccine AND >10 years since last dose	Tdap preferred (if not yet received) or Td	Tdap preferred (if not yet received) or Td

 Table 12.3(2): Tetanus Vaccination and TIG for Wound Management ³⁶.

DTaP – Diphtheria and Tetanus toxoids and acellular pertussis vaccine.

Tdap – tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis.

TIG – Tetanus immune globulin.

Td – tetanus and diphtheria toxoids.

13 Diabetic Foot Infections

13.1 Aetiology

The pathogens commonly encountered in diabetic foot infections include^{3,14,30}:

Type of Infection	Most Common Pathogens	
Mild to moderate cases	• Staphylococcus spp.	
Moderate to severe cases and wounds, longstanding ulcers, or those previously treated with antibiotics.	 S. aureus. Streptococcus agalactiae. Gram-negative bacilli. Gram-negative rods. Anaerobic bacteria. 	
Infections of the ischemic foot	Anaerobic pathogens	

 Table 13.1: Common Pathogens Causing Bacterial SSTIs in the Feet of Diabetic Patients^{3,14,30}.

13.2 Clinical Presentation and Assessment

Diabetic foot infection (DFI) is typically a deep abscess, cellulitis of the dorsum, or a mal perforans ulcer³. Localised signs of infection may be present but pain and systemic signs of infection are usually absent³ [L1].

Laboratory studies may be required to determine the appropriate management³ [L1, RGA]:

- Imaging.
- Cultures obtained from deep tissue samples post-debridement.

If a DFI is suspected, enquire about³⁰:

- History of diabetes.
- History of penetrating trauma.
- Possibility of extension of local infection.

13.3 Management

All patients with DFI should be offered the multidisciplinary foot care service^{14,26} [L1, RGA] that has access to 26 [L1]:

- Rehabilitation services.
- Plastic surgery.
- Psychological services.
- Nutritional services.
- General and vascular surgery.
- Podiatry.
- Endocrinology.
- Occupational therapy.
- Tissue viability team.
- Other services listed in NCG on *Chronic Complications of Diabetes Mellitus* by MOPH ³⁷.

Treatment of a DFI is based on the extent and severity of the infection^{26,30} [L1, RGA]:

- Mild infections:
 - Should be treated with oral antibiotics.
 - Treatment in the outpatient setting is sufficient. Hospitalisation is not required.
- Selected patients with moderate infections and all patients with severe infections:
 - Should be treated with parenteral antibiotics.
 - Require hospitalisation, surgical consultation, and additional evaluation.
 - $\circ~$ Surgical debridement and drainage of deep tissue abscesses and infections may be required $^{30}.$
 - For more details, refer to the MOPH National Clinical Guideline on *Chronic Complications* of *Diabetes Mellitus*³⁷.

As most DFIs are polymicrobial, no single antibiotic regimen is clearly superior to another³⁰ [L1, RGB]. Consider regimens listed in *Table 13.3*.

DFIs are frequently complicated with osteomyelitis^{3,30}.

Type of Infection	Empirical Antimicrobials	Additional Notes	
Ulcer with Superficial Inflammation <2 cm	Cefuroxime axetil or Cloxacillin.	Duration of treatment is two weeks.	
Ulcer with Inflammation (>2 cm)	Co-amoxiclav or ampicillin/sulbactam.	First-choice medications.	
and Extension into Fascia	Moxifloxacin or ertapenem.	Second-choice medications.	
All Other Cases	Linezolid or clindamycin.	If MRSA is suspected or cultured.	

Table 13.3: Selection of Empirical Antibiotics for Treatment of DFI²¹.

14 Infected Burns

14.1 Aetiology

Infections of burn injuries can be caused by^{1,25}:

- Gram-positive bacteria:
 - S. aureus.
 - S. pyogenes.
 - Streptococcus agalactiae.
 - Enterococcus spp.
- Gram-negative bacteria:
 - Pseudomonas aeruginosa.
 - Acinetobacter spp.
 - o Escherichia coli.
 - o Klebsiella.
 - Enterobacter.
 - o Serratia.
 - o Proteus.
 - Anaerobic organisms:
 - Bacteroides.
 - Fusobacterium spp.
- Fungi/yeasts:
 - Candida tropicalis.
 - Candida krusei.
 - Candida albicans.
 - $\circ \quad \text{Non-albicans Candida spp.}$
 - Aspergillus.
 - Fusarium.
 - o Rhizopus.
 - o Mucor.

14.2 Clinical Presentation and Assessment

Burn wound colonisation should be diagnosed when²⁵ [L2]:

- Bacteria are present at low concentrations (<10⁵ colony-forming units) on the wound's surface.
- Surrounding erythema or cellulitis is not evident; but
- Deterioration of the wound surface can be observed.

Burn infection should be diagnosed when²⁵ [L2]:

- Bacteria are present at high concentrations (>10⁵ colony-forming units) in the burn wound and scab.
- Cellulitis is evident.
- Warmth within the area, pain or tenderness, advancing swelling, or induration are present.

14.3 Management

Irrigation of the wound and debridement of necrotic tissue and the eschar, should be performed to decrease the incidence of invasive burn wound infection^{1,25} [L1, RGB].

Broad-spectrum antimicrobial agents effective against both aerobic and anaerobic organisms and antifungals may be considered in the patients with^{1,25} [L1, RGA]:

- Systemic signs of infection.
- Compromised immune status.
- Severe comorbidities.
- Associated severe cellulitis.
- Severe and deep wounds.

Antibiotic prophylaxis in burn injuries is not generally recommended¹ [L1, RGB].

15 Necrotising Skin and Soft Tissue Infections

15.1 Aetiology

The causative organisms responsible for necrotising skin and soft tissue infections, include^{3,5,10,14}:

- S. pyogenes.
- S. aureus.
- Clostridium perfringens.
- Clostridium septicum.
- Vibrio vulnificus.
- Aeromonas hydrophilia.
- Enterobacteriaceae.
- Bacteroides spp.
- Clostridium spp.
- *Peptostreptococcus* spp.

15.2 Clinical Presentation and Assessment

The differentiating features of the different types of necrotising SSTIs are provided in the table below.

Type of Infection	Specific Features
Necrotising Fasciitis	 Usually affects the genitalia, perineum, or lower extremities. Starts with erythema, tenderness, warmth, swelling, and pain. Spread of infection involves any or all layers of the soft tissue. Signs and symptoms of infection are present. Overlying redness and cutaneous anaesthesia. Tense overlying oedema, bullae, and induration of apparently uninvolved tissues. Skin discoloration and crepitus are present. Pain is disproportionate to the physical findings. Rapid progression despite antibiotics.
Clostridial Myonecrosis (Gas Gangrene)	 Usually occur after abdominal surgery on the gastrointestinal tract. Starts as sudden onset of pain at the site of trauma or surgical wound. Severe pain at site followed by skin changes (e.g. pale, bronze, purplish red). Tenderness, induration, blistering, and tissue crepitus. Haemorrhagic bullae are common. Gas production. Muscle necrosis. Diaphoresis. Systemic signs of infection and toxaemia are present.
Fournier's Gangrene	 Develops in genital area and/or perineum but may extend up to the abdominal wall, or down into the thigh areas, or into the perirectal and gluteal spaces, and into the retroperitoneum. Testicular involvement is rare. Cellulitis, patches of gangrene, foul smell. Signs and symptoms of infection. Suppuration and necrosis of overlying skin.

Table 15.2: Clinical Presentations of Necrotising SSTIs^{1-3,5}.

In addition to general history taking and examination, described in *Sections 5.2 and 5.3*, enquire about the following^{1–3,5}:

- Recent history of penetrating or blunt trauma.
- Presence of perianal abscesses or decubitus ulcers or recent surgery.
- Abdominal operations on the gastrointestinal tract that were complicated by superficial wound infections.
- Spread from a genital site such as Bartholin abscess, episiotomy wound, or a minor vulvovaginal infection.
- Urological manipulation.
- History of obesity or malnutrition.
- Intravenous drug abuse.

15.3 Investigation and Diagnosis

Necrotising fasciitis is an aggressive SSTI involving the superficial fascia, which comprises all the tissue between the skin and underlying muscles^{5,10}:

- Polymicrobial necrotising fasciitis spreads in 3-5 days³.
- Streptococcal necrotising fasciitis can progress within 1-2 days³.
- "Idiopathic" or "spontaneous" necrotising fasciitis should be considered in the absence of trauma or another cause for the infection⁵ [L2, RGA].

Imaging studies may provide useful information when the diagnosis is uncertain¹ [L1, RGB], but they should not delay surgical consultation and further interventions¹ [L1, RGC].

Laboratory investigations and surgical exploration may be required to make the definitive diagnosis^{3,5,10} [**L1, RGA**]:

- The LRINEC Scoring systems for indicating a necrotising SSTI are provided in *Table 15.3* below.
 - Scores >6 are indicative, scores >8 are strongly predictive.
- Surgical findings include:
 - Swollen and dull-grey fascia.
 - Wooden-hard induration of the subcutaneous tissues.
 - Absence of true pus even after deep dissection.
 - Tissue planes can be readily dissected with a gloved finger or a blunt instrument.

Test	Laboratory Values		Score
	>13.5 g/dL	(>135 g/L)	0
Haemoglobin	11 - 13.5 g/dL	(110-135 g/L)	1
	<11 g/dL	(<110 g/L)	2
	<15 000 cells/mm ³	(<15.0*10 ⁹ cells/L)	0
White Cell Count	15 000 - 25 000 cells/mm ³	(15.0-25.0 x 10 ⁹ cells/L)	1
	>25 000 cells/mm ³	(>25.0 x 10 ⁹ cells/L))	2
CRD	<150 mg/L	(<1 430 nmol/L)	0
CRP	≥150 mg/L	(≥1 430 nmol/L)	4
Cadium	≥135 mEq/L	(≥135 mmol/L)	0
Soulum	<135 mEq/L	(<135 mmol/L)	2
Creatining	≤1.6 mg/dL	(≤141 µmol/L)	0
Creatinine	>1.6 mg/dL	(>141 μmol/L)	2
Pland Clusters	≤180 mg/dL	(≤10 mmol/L)	0
Bioda Giucose	>180 mg/dL	(>10 mmol/L)	1

Table 15.3: The LRINEC (Laboratory Risk Indicator for Necrotising Fasciitis) Scoring System^{2,38}.

15.3.1 Fournier's Gangrene

Fournier's gangrene is a necrotising fasciitis of the perineal, genital (the scrotum and penis or vulva) and perianal region^{10,39} [**L1**, **RGA**].

15.3.2 Clostridial Myonecrosis

Clostridial myonecrosis (gas gangrene) is an acute infection by clostridium or bacillus of healthy living tissue characterised by gas production and muscle necrosis^{1,3}. Spontaneous gangrene may develop in the absence of trauma. It usually occurs in patients with neutropenia or gastrointestinal malignancy¹⁰ [L1].

Recurrent gas gangrene may occur several decades after the primary infection⁵ [L2].

Laboratory investigations and surgical exploration may be required to make the definitive diagnosis^{1,3,5,10} [**L1, RGA**]:

- Laboratory abnormalities include:
 - Common blood test abnormalities (see *Table 15.3*).
 - Positive blood culture results.
- Surgical findings include:
 - At early stages, muscles are pale, oedematous and unresponsive to stimulation.
 - At later stages, muscles become frankly gangrenous, black, and extremely friable.

15.4 Management

Surgical exploration with aggressive, total debridement of all devitalised and necrotic tissue is essential in all cases of necrotising SSTIs^{2–5,14} [L1, RGA]:

- Patients may require repeated surgeries to debride all affected tissue^{2,10} [L1, RGA].
- When extremities are involved, amputation may be required⁵ [L2, RGA].
- Diverting colostomy should be avoided when other methods available to avoid wound contamination¹ [L1, RGA]. In cases with faecal contamination, faecal diversion should be considered^{1,5} [L1, RGA]. The following options are available¹:
 - Colostomy.
 - Faecal tube system.
- Frequent operations and dressing changes should be provided when necessary⁵ [L2, RGA].

Negative pressure wound therapy for wound care after complete removal of necrosis in necrotizing infections may be considered¹ [L1, RGB].

Antibiotic treatment of necrotising infections should also be prompt and aggressive¹ [L1, RGA]:

- The infection must be immediately treated with high-dose parenteral broad-spectrum antibiotics directed against the polymicrobial aerobic and anaerobic microorganisms (see *Table 15.4*)^{1-3,5} [L1, RGA].
- The antibiotic spectrum can be narrowed once culture tests become available^{1,2,5} [L1, RGA].
- Cultures of the superficial wound may not reflect organisms in the deep tissue infection¹⁰ [L1, RGC].

Empirical Antimicrobials	Additional Notes	
 Ampicillin/sulbactam plus clindamycin. Pipracillin/tazobactam plus clindamycin 	First-choice antibiotics for patients with polymicrobial infections. Antibiotic therapy is usually required for 2-3 weeks.	
Add vancomycin	If MRSA is suspected or cultured. Vancomycin treatment should be avoided in patients with renal impairment.	
Parenteral aqueous penicillin G plus clindamycin.	Group A streptococcal and clostridial infections. Antibiotic therapy is usually required for 2-3 weeks.	
Doxycycline plus ciprofloxacin or ceftriaxone.	If Aeromonas hydrophila or Vibrio vulnificus is suspected or cultured.	

 Table 15.4: Selection of Antimicrobials for Treatment of Necrotising SSTIs^{2,3,10,21}.

All patients should receive supportive care to maintain oxygenation and tissue perfusion, including fluid resuscitation, organ support and nutritional support¹⁴ [L2, RGA]. Hyperbaric oxygen therapy is not recommended in necrotising fasciitis^{3,10} [L1, RGB].

16 Viral Skin and Soft Tissue Infections

16.1 Aetiology

Viral SSTIs are commonly caused by^{3,10,12,17,40}:

- Herpes virus.
- VZV.
- Measles morbillivirus.
- Rubella virus.
- Molluscum contagiosum virus (MCV).
- Epstein-Barr virus.

16.2 Clinical Presentation and Assessment

Differentiating features of different viral skin and soft tissue infections are provided in the table below.

Type of Infection	Specific Features
Herpes Zoster	 Unilateral rash, not crossing the body's midline. Starts as an erythematous, maculopapular rash. Disseminated rash (in immunocompromised patients). Vesicles transform into pustules that crust over and heal in 2-4 weeks. Episodic or continuous symptoms of pain. Itching and/or tingling. Postherpetic neuralgia as a complication.
Measles (Rubeola)	 Fever ≥38°C. Malaise. Cough, coryza, and conjunctivitis. Pathognomonic enanthem. Maculopapular rash spreading from the head to the trunk and to the lower extremities. Photophobia. Stomatitis.
Rubella (German measles)	 Slight fever. Mild, maculopapular rash starting on the face and spreading all over the body. Lymphadenopathy of posterior auricular or suboccipital lymph nodes. Nausea and mild conjunctivitis.
Infectious Mononucleosis	 Malaise. Extreme fatigue. Fever. Pharyngitis. Head and body aches. Adenopathy of lymph nodes in the neck and armpits. swollen liver or spleen or both. Rash.

Type of Infection	Specific Features	
Molluscum Contagiosum	 In children: lesions on the face, neck, trunk and arms. In adults: lesions on the genitals, pubic region, lower abdomen, upper thighs, and/or buttocks. Dome-shaped, smooth-surfaced, pearly, firm, skin-coloured, pink, yellow or white papules, 2-5 mm in diameter with central umbilication. Mechanical evacuation of content reveals a cheesy material containing degenerated keratinocytes and viral particles. Local pruritus or discomfort may be present. Eczema in surrounding skin. Infection on eyelid margins can cause chronic conjunctivitis. 	

 Table 16.2: Clinical Presentations of Viral SSTIs^{3,10,12,17,40-42}.

In addition to general history taking and examination, described in *Sections 5.2 and 5.3*, enquire about the following^{12,17,27}:

- Travel history.
- Expose to a person with febrile rash illness.
- Chemotherapy treatment in the past year.
- Hematopoietic stem cell transplantation.
- Solid organ transplant.

16.3 Investigation and Management of Specific Viral SSTIs

The investigation and management of each of the common viral SSTIs is provided in the following subsections.

16.3.1 Measles

16.3.1.1 Measles Diagnosis

Sometimes immunocompromised patients do not develop the rash¹². Laboratory investigations should be used to objectively confirm the suspected diagnosis¹² [L1, RGA]:

- Measles-specific IgM antibody in serum.
- Measles RNA in the throat swab.
- Urine samples.

Severity should be assessed to define proper management and treatment²⁷ [L1, RGA]:

- Uncomplicated measles:
 - No manifestation other than skin rash.
- Complicated measles:
 - Increased breath rate without chest indrawing:
 - ≥40 breaths/minute if aged >1 year.
 - ≥50 breaths/minute if aged <1 year.
 - Some dehydration.
 - Stridor only when the child is upset or crying.
 - Mouth ulcers not affecting intake of food or fluids.
 - Pus draining from the eyes.
 - Acute otitis media, duration <14 days.
 - Severe complicated measles:

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- Not able to drink or breastfeed.
- $\circ \quad \text{Convulsions.}$
- Lethargic or unconscious.
- \circ Deep or extensive mouth ulcers.
- Chest indrawing and rapid breathing.
- Stridor in a calm child.
- \circ ~ Corneal clouding or ulcers, or vision affected.
- o Mastoiditis.
- Severe malnutrition and dehydration.

All suspected measles cases should be reported local health departments within 24 hours¹² [L1].

16.3.1.2 Measles Prevention

Measles infection can be prevented with measles-containing vaccine¹² [L1, RGA]:

- It is usually administered as either the combination measles-mumps-rubella (MMR) vaccine or measles-mumps-rubella-varicella (MMRV) vaccine¹².
- Routine childhood immunisation is recommended¹² [L1, RGA]:
 - MMR vaccine:
 - The first dose should be applied at 12-15 months of age.
 - The second dose should be applied at 4-6 years of age or at least 28 days after the first dose.
 - MMRV vaccine:
 - The minimum interval between doses is three months.
- Infants 6-11 months old traveling abroad should receive one dose of MMR vaccine¹² [L1, RGA]. They should also receive 2 more doses according to the routinely recommended schedule (see above)¹² [L1, RGA].

16.3.1.3 Measles Management

There is no specific treatment for measles^{12,27} [L1]. The condition usually improves within 7-10 days.

General principles of management directed at^{12,27} [L1, RGA]:

- Relieving common symptoms²⁷.
- Providing nutritional support and promoting breastfeeding.
- Providing vitamin A.
- Assessment and treatment of common complications if they arise^{12,27}. Multiple complications should be treated at the same time²⁷ [L1, RGA].
 - Otitis media.
 - o Bronchopneumonia.
 - Laryngotracheobronchitis.
 - o Diarrhoea.
 - o Pneumonia.
 - o Croup.
 - Malnutrition.
 - Mouth ulcers.
 - Eye complications.

If measles is diagnosed, prompt measures should be taken to stop the spread of infection¹² [L1, RGA].

- Patients are considered to be contagious from 4 days before to 4 days after the rash appears.
- Up to 9 out of 10 susceptible persons with close contact to a measles patient will develop measles.

- The virus is transmitted by:
 - Direct contact with infectious droplets.
 - Airborne spread when an infected person breathes, coughs, or sneezes.
- The virus remains infectious in the air for up to 2h after an infected person leaves an area.

16.3.2 Rubella

16.3.2.1 Rubella Diagnosis

Some cases may be difficult to recognise as the rash resembles other rash illnesses¹⁷. Other cases may be subclinical or unapparent. Laboratory testing is required to confirm the diagnosis^{17,43} [L1, RGA]:

- Blood samples for serologic testing.
- Throat and nasal swab.
- Urine samples.

16.3.2.2 Rubella Prevention

Rubella can be prevented with rubella-containing vaccines (MMR or MMRV)^{12,17,43} [L1, RGA]:

• Refer to Section 16.3.1.1 for types of vaccine and immunisation schedule for children.

16.3.2.3 Rubella Management

There is no specific antiviral therapy for rubella¹⁷ [L1]. Symptoms are usually mild and do not require any specific treatment. Children with rubella usually recover within 1 week, for adults it may take longer.

If rubella is diagnosed, control measures should be taken to prevent spreading of infection¹⁷ [L1, RGA]:

- Patients are contagious from 7 days before to 7 days after the rash appears. They are most contagious on days 1-5 after the appearance of the rash, when it is erupting.
- All patients with rubella should be isolated for at least 7 days after they develop rash.
- The virus is transmitted by:
 - Direct contact with an infected person.
 - Contact with droplets of nasopharyngeal secretions.

Possible complications of rubella include^{17,43}:

- Arthralgia.
- Arthritis.
- Thrombocytopenic purpura.
- Encephalitis.
- Congenital rubella syndrome.
- Miscarriage, foetal death, stillbirth.

16.3.3 Infectious Mononucleosis

16.3.3.1 Infectious Mononucleosis Diagnosis

Laboratory investigations are required to make the definitive diagnosis⁴⁰ [L1]. Typical abnormalities include:

- Specific antibodies in serum.
- Atypical lymphocytosis.
- Neutropenia.
- Abnormal liver function.

Consider alternative diagnoses⁴⁰:

- Streptococcal pharyngitis.
- Viral pharyngitis.
- Acute cytomegalovirus.
- Toxoplasmosis.

16.3.3.2 Infectious Mononucleosis Management

Vaccination for infectious mononucleosis is not available⁴⁴ [L1]. There is also no specific antiviral therapy for infectious mononucleosis ¹⁷ [L1]. Most people recover within 2-4 weeks⁴⁴. Some patients may feel fatigued for longer. Rarely, symptoms of infectious mononucleosis can last >6months⁴⁴.

Advise patients to:

- Drink fluids and hydrated^{40,44} [L1, RGA].
- Get rest⁴⁴ [**L1, RGA**].
- Take over-the-counter medications for pain relief and fever^{40,44}[L1, RGA]:
 - Nonsteroidal anti-inflammatory drugs.
 - Acetaminophen.
 - Throat lozenges, sprays or gargling with a 2 percent lidocaine.
 - Avoid the following medications^{40,44} [L1, RGB]:
 - Penicillin antibiotics (e.g. ampicillin or amoxicillin).
 - Corticosteroids (e.g. prednisone).
 - \circ Acyclovir.
 - o Ranitidine.
 - Antihistamines.
- Self-isolate to stop transmitting virus via⁴⁴:
 - Bodily fluids, especially saliva.
 - Blood (including blood transfusions and organ transplantations).
 - Semen during sexual contact.

Possible complications of infectious mononucleosis should be reviewed and evaluated⁴⁰:

- Splenomegaly.
- Hepatomegaly.
- Splenic rupture.
- Acute interstitial nephritis.
- Haemolytic anaemia.
- Myocarditis and cardiac conduction abnormalities
- Neurologic abnormalities.
- Cranial nerve palsies
- Encephalitis.
- Meningitis.

- Mononeuropathies.
- Retrobulbar neuritis.
- Thrombocytopenia.
- Upper airway obstruction.

16.3.4 Herpes Zoster

Diagnostic testing is not usually required³ [L1, RGB], but may be considered in patients with atypical presentations or with suspected herpes simplex virus³ [L1, RGA].

Consider alternative diagnoses¹⁰:

- Atypical varicella.
- Herpes simplex virus.

Antiviral therapy is the main treatment option for patents with VZV¹⁰ [L1, RGA]. Consider the following medications¹⁰ [L1, RGA]:

- Acyclovir.
- Famciclovir.
- Valacyclovir.

Oral administration is recommended in otherwise healthy patients or in immunocompromised patients with mild cases of VZV¹⁰ [L1, RGA]. Immunocompromised patients with more extensive cases should receive intravenous administration¹⁰ [L1, RGA].

16.3.5 Molluscum Contagiosum

16.3.5.1 Molluscum Contagiosum Diagnosis

Molluscum contagiosum (MCV) has two main subtypes mainly transmitted by direct physical contact, including auto-inoculation ^{41,42}.

The diagnosis is usually done on clinical and dermoscopic grounds ⁴¹ [L1]. Consider in vivo confocal microscopy to distinguish the condition from other skin infections ⁴¹ [L1].

Laboratory investigations are not usually required. The following tests may be considered in unclear cases:

- Histological examination.
- Polymerase chain reaction.
- Electron microscopy.

Differential diagnoses include ⁴¹:

- Genital warts (condyloma acuminata).
- Flat warts.
- Lichen planus or nitidus.
- Secondary syphilis condylomata lata.
- Pyogenic granuloma.
- Ectopic sebaceous glands.
- Dermal cyst.
- Vulvar lymphangioma circumscriptum.
- Keratoacanthoma.

- Basal cell carcinoma.
- Amelanotic melanoma.

16.3.5.2 Molluscum Contagiosum Management

Molluscum contagiosum is a benign and usually and self-limiting viral infection of the skin ⁴¹. In most immunocompetent patients, the signs and symptoms resolve in 3 months to 3 years when an immune response develops ⁴²:

- Most children do not require treatment ⁴² [L2, RGA].
- Adults patients with sexually transmitted MCV usually request treatment. Active therapy is also indicated in patients with ⁴¹ [L1, RGA]:
 - Extensive involvement.
 - Disease persistence.
 - Cosmetic reasons.
 - Fear of disease spread and scarring.
 - Complications (e.g. pruritus, inflammation, and secondary infection).

Treatment	Additional Notes	
Cautery	Immediate results. Local anaesthetics may be required.	
Curettage	Less suitable for genital skin because of pain.	
Liquid nitrogen cryotherapy	Should be applied with caution.	
Podophyllotoxin*	May be patient-applied.	
Imiquimod*	Not generally recommended but may be considered if other	
Squeezing/piercing lesions	treatments are not suitable. Should be applied with caution.	
 Topical Medications: Salicylic, lactic, glycolic and trichloroacetic acids. Benzoyl peroxide 5%. Aluminium acetate solution (Burrow's solution 1:30). Hydrogen peroxide. Iodine. Potassium hydroxide. Silver nitrate. Nitric oxide. Cantharidin. Lemon myrtle oil. Tea tree oil. Tretinoin. Adapalene. 	Not generally recommended but may be considered if other treatments are not suitable. Varying evidence for efficacy has been reported for non-genital molluscum. No recommendation can be given regarding the use for genital infections. Topical treatments should be avoided in pregnancy and breastfeeding.	

 Table 16.3.5.2: Selection of Medication for Treatment of Molluscum Contagiosum ^{41,42}.

Sharing towels, sponges or bathing together should be discouraged to reduce the spread of infection to healthy individuals ^{41,42} [L2, RGA].

17 Parasitic Skin Infections

17.1 Aetiology

Most common parasites causing skin infections include ^{45,46}:

- Epidermal parasitic skin diseases (EPSD):
 - Scabies:
 - Sarcoptes scabiei.
 - Pediculosis:
 - Pediculus humanus var. capitis.
 - Pediculus humanus var. corporis.
 - Phthirus pubis.
 - Tungiasis consider in immigrants and travellers from the Caribbean, sub-Saharan Africa and South America:
 - Female sand flea *Tunga penetrans*.
 - Hookworm-related cutaneous larva migrans (HrCLM) consider in immigrants and travellers from developing countries:
 - Ancylostoma caninum.
 - Ancylostoma braziliense.
 - Uncinaria stenocephala.
- Cutaneous leishmaniasis *Leishmania* parasites (>20 different species).
- Loiasis tissue nematode Loa loa.

17.2 Clinical Presentation and Assessment

Differentiating features of different parasitic skin infections are provided in the table below.

Type of Infection	Specific Features	
Scabies ^{45–48}	 Symptoms may not appear for up to 2 months after being infested. In re-infested individuals, symptoms appear 1-4 days after exposure. Itching and a skin rash (pruritus). Severe itching at night. Itching and rash may affect much of the body or be limited to common sites (e.g., between the fingers, armpits, waist). The head, face, neck, palms, and soles often are involved in infants and very young children. Tiny intra-epidermal burrows sometimes can be seen on the skin. Skin sores contaminated with secondary bacterial infection (e.g. group A Streptococci) may be present. Cellulitis, boils, and pyomyositis may develop if untreated. Lymphangitis and generalised lymphadenopathy. Crusted scabies may develop in the elderly, immunocompromised or physically incapacitated individuals. It is characterised by vesicles and thick crusts over the skin that can contain many mites. 	
Pediculosis ^{46,49}	 On the first infestation, sensitisation commonly takes 4 to 6 weeks. Pruritus. Intense itching can lead to scratching and subsequent excoriations and secondary cellulitis. If untreated, the skin may become lichenified and hyperpigmented. 	

Type of Infection	Specific Features	
Tungiasis ^{45,50,51}	 Itching and local irritation. Pain and sensation of a foreign body. Lesions, 99% of which occur at the feet. Most lesions occur on the nail rim. Desquamation of the surrounding skin. Heavy inflammation often surrounds the lesions. Secondary bacterial infection is often present. Suppuration, ulceration and lymphangitis can develop. Gangrene or necrosis of surrounding tissue may occur. 	
Hookworm-Related	Serpiginous rash with pruritus.	
Cutaneous Larva	 Raised red lines on the feet or lower part of the legs. 	
Migration ^{52,53}	Scratching lines can lead to bacterial infections.	
Leishmaniasis 54,55	Cutaneous leishmaniasis:	
	• Painless and chronic skin lesions occurring at sites of infected sand fly bites.	
	Slow spontaneous healing.	
	May be associated with mucosal leishmaniasis.	
	Mucocutaneous leishmaniasis:	
	 Chronic unexplained congestion/secretions. 	
	 Partial or total destruction of mucous membranes of the nose, mouth and 	
	throat.	
	Visceral leishmaniasis:	
	Irregular bouts of fever.	
	Weight loss.	
	Enlargement of the spleen and liver	
	Anaemia.	
Loiasis 56	Presence of an eye worm.	
	Calabar swellings.	
	Unexplained peripheral eosinophilia.	

Table 17.2: Clinical Presentations of Parasitic SSTIs.

17.3 Diagnosis and Management

17.3.1 Scabies

17.3.1.1 Scabies Diagnosis

A suspected diagnosis of a scabies infestation can be made on the basis of clinical distribution and appearance of the skin lesions. A definite diagnosis of scabies should be made on microscopic identification of the mites, eggs or faecal pellets (scybala) from the scrapings of the skin burrows.

Consider applying other tests when required ⁴⁸ [L1, RGB]:

- Burrow ink test (BIT) to identify the burrows.
- Dermoscopy.
- Detecting parasitic DNA from cutaneous scales.
- Optical coherence tomography.

The differential diagnosis for scabies includes⁴⁸ [L1]:

- o Impetigo.
- Folliculitis.

- Papular urticarial.
- Atopic, contact or seborrhoeic dermatitis.
- Dermatitis herpetiformis.
- o Psoriasis.
- Pytiriasis rosea.
- Secondary syphilis.
- Lymphoma and pseudolymphoma (if scabies presents with nodules).

17.3.1.2 Scabies Management

Treatment should be applied simultaneously for household members and sexual contacts to prevent reinfestation ⁴⁷ [L1, RGA].

Several scabicides are available only by prescription to treat human scabies (see Table 17.3.1.2)^{47,48}.

Medications	Additional Notes	
	Two (or more) applications, each about a week apart, may be required	
Permethrin cream 5%	to eliminate all mites.	
	Applicable in children ≥2 months.	
Crotamiton lotion (or cream) 10%	May be used in children.	
	Frequent treatment failure has been reported.	
Malathion aqueous 0.5% liquid	May be used if permethrin cream is inappropriate.	
Sulphur ointment 5%-10%	Applicable in children <2 month of age.	
Benzyl benzoate 25% emulsion	Second-line treatment option. The use should be restricted to patients who have failed treatment with or cannot tolerate other medications.	
Lindane lotion 1%		
Oral ivermectin		

 Table 17.3.1.2: Selection of Medications for Treatment of Scabies ^{47,48}.

Bedding, clothing, and towels used by infested persons and their household, sexual, and close contacts anytime during the 4 days before treatment should be decontaminated by ⁴⁷ [L1, RGA]:

- Washing at high temperature (60°C) and drying in a hot dryer; or
- o Dry-cleaning; or
- Sealing in a plastic bag for at least 72 hours.

Oral antihistamines to control pruritus should be avoided during pregnancy when possible, especially during the first trimester ⁴⁸ [L1, RGC]. If required, consider chlorpheniramine ⁴⁸ [L1].

17.3.2 Pediculosis

17.3.2.1 Pediculosis Diagnosis

Lice infestation is diagnosed definitively by visual inspection ^{46,49} [L1]:

- At least one live louse must be observed on the hair (head or pubic lice) or in the seams of clothing (body lice).
- The presence of nits (louse eggs that may or may not be viable) on examination is not enough to diagnose pediculosis as they may remain on the hair for months after successful treatment.

Consider using bright light, magnifying lenses, and/or combing the hair with a "lice comb" for the hair inspection 46,57 [L1]. Search behind the ears and on the back of the neck. Patients with pubic lice should be evaluated for sexually transmitted infections 46 [L1].

17.3.2.2 Pediculosis Management

All family members of the patient with pediculosis should be examined and treated ⁴⁶ [L1, RGA].

Treatment options include topical insecticides and oral agents (see *Table 17.3.2.2*) ^{46,49} [**L1, RGA**]. Repeat treatment (two applications 7 to 10 days apart) is usually required for complete eradication ⁴⁶ [**L1**].

Medications	Additional Notes	
Permethrin 1%	First-line treatment option.	
Pyrethrins 1%	Applicable in children ≥2 months.	
Benzyl alcohol lotion 5%	Applicable in children ≥6 months.	
Pyrethrins 0.3%/piperonyl butoxide 4% shampoo or mousse	Should be avoided in patients with chrysanthemum allergy.	
Crotamiton lotion (or cream) 10%	May be used in children.	
Malathion lotion 0.5%		
Spinosad* 0.9%	May be considered if first-line treatment is inappropriate.	
Oral ivermectin		
Isopropyl myristate 50% and ST- cyclomethicone 50%	May be considered if first-line treatment is inappropriate. Not recommended for use on infants or children <4 years of age.	
Lindane lotion	Not generally recommended and should be avoided when possible. It should not be used in children, older persons, or adults weighing less than 50 kg.	
Carbaryl	Prohibited due to carcinogenic effects.	

 Table 17.3.2.2: Selection of medications for treatment of pediculosis 46,49,58.

* - Spinosad has not been evaluated for the treatment of pediculosis pubis.

Bedding, clothing, and towels used by infested persons and their household should be decontaminated by washing at high temperature (\geq 50°C) ⁴⁶ [L1, RGA].

Wet combing is not recommended as a primary treatment, but may considered as adjuvant treatment ^{46,49} [**L1, RGB**].

The following treatment measures are not recommended ^{46,49} [L1, RGB]:

- Sprays, carpet treatments, and other chemical environmental decontamination measures.
- Household products (such as mayonnaise, petroleum jelly, olive oil, tub margarine, etc.).
- Natural products (e.g., tea tree oil).
- Aromatherapy.
- Using flammable, toxic and dangerous substances (e.g., gasoline or kerosene) or using products intended for treating lice in animals is strictly prohibited ⁴⁹ [L1, RGC].

17.3.3 Tungiasis

17.3.3.1 Tungiasis Diagnosis

The diagnosis of tungiasis is made by clinical inspection and is based on the morphological characteristics of the different developmental stages c [L1].

A biopsy of lesions is not generally recommended ⁵¹ [**L2**, **RGB**] but may be considered in cases with atypical presentation (e.g. is lesions have a pseudoepitheliomatous appearance at the ectopic site) ⁵¹.

17.3.3.2 Tungiasis Management

Limited treatment options are available for patients with tungiasis, see table below.

Treatment	Additional Notes
Topical application of a two-component dimeticone of low viscosity.	First-line treatment.
Surgical extraction*	The use is discouraged unless first-line treatment is applicable.

Table 17.3.3.2: Selection of medications for treatment of tungiasis ^{50,51,59}.

* Surgical extraction of burrowed sand fleas should be performed by sterile instruments in an appropriately equipped health facility ⁵⁰ [L1, RGA]. The wound should be dressed appropriately. The tetanus vaccination status needs to be verified and a booster vaccination given, if indicated.

The following medications are not recommended due to proven inefficiency or insufficient evidence of efficiency ^{50,51} [**L1**, **RGB**]:

- Topical metrifonate.
- Oral or topical thiabendazole.
- Oral or topical ivermectin.

17.3.4 Hookworm-Related Cutaneous Larva Migrans

17.3.4.1 Hookworm-Related Cutaneous Larva Migrans Diagnosis

Hookworm-Related Cutaneous Larva Migrans (HrCLM) is a clinical diagnosis based on the presence of the typical signs and symptoms, and exposure history to zoonotic hookworm ⁵³ [L1]. Blood tests and biopsy are usually not necessary for diagnosis ⁵² [L2, RGB].

Differential diagnoses include ^{52,53}:

- Hookworm.
- Gnathostomiasis.
- Strongyloidiasis.
- Cutaneous pili migrans.
- Myiasis.
- Loiasis.
- Scabies.
- Schistosomiasis.
- Tinea corporis.
- Contact dermatitis.

17.3.4.2 Hookworm-Related Cutaneous Larva Migrans Management

Hookworm-Related Cutaneous Larva Migrans (HrCLM) is usually a self-limiting infection. In most patients, the signs and symptoms resolve after 5-6 weeks without medical treatment. The larva will die in the skin and the itchiness and red lines will go away ⁵³.

For multiple lesions or severe infestation consider treatment with anti-worm medications see table below.

Medication	Additional Notes	
Oral albendazole	This drug is contraindicated in children younger than 2 years age.	
Oral ivermectin	One-time dose is usually required.	
Topical thiabendazole 10% solution	If infaction is local	
Topical thiabendazole 15% ointment		

 Table 17.3.4.2: Selection of Medications for Treatment of HrCLM ^{52,53}.

Adjuvant treatment to help control symptoms and to resolve secondary bacterial infections may be considered ⁵³.

The following treatment options are not recommended:

- Cryotherapy ^{52,53} [L1, RGB].
- Topical or oral steroids ⁵² [L2, RGB].
- Antibiotics ⁵² [L2, RGB].

17.3.5 Leishmaniasis

17.3.5.1 Leishmaniasis Diagnosis

There are three main forms of the disease ^{54,55,60}:

- Cutaneous leishmaniasis.
- Mucocutaneous leishmaniasis.
- Visceral leishmaniasis.

Cutaneous Leishmaniasis:

- Diagnosed by clinical manifestation with parasitological tests ⁵⁵ [L1].
- Tissue specimens should be collected from a lesion(s) ⁵⁴ [L1].
- Full-thickness skin biopsy may be considered ⁵⁴ [L1].
- Differential diagnoses include ⁶⁰:
 - Staphylococcal or streptococcal infection.
 - Mycobacterial ulcer.
 - o Leprosy.
 - Fungal infection.
 - Cancer.
 - $\circ \quad \text{Sarcoidosis.}$
 - Tropical ulcer.

Mucocutaneous Leishmaniasis:

- Diagnosed by clinical manifestation with parasitological tests ⁵⁵ [L1].
- Tissue specimens should be collected from a lesion(s) ⁵⁴ [L1].
- Biopsy specimens may be considered ⁵⁴ [L1].
- Differential diagnoses include ⁶⁰:
 - Allergic rhinitis.
 - Paracoccidioidomycosis and other deep mycoses.
 - Cancrum oris.
 - Lymphoma and other neoplasia.
 - o Leprosy.
 - Sarcoidosis.

Visceral Leishmaniasis:

- Diagnosed by combining clinical signs with parasitological, or serological tests ⁵⁵ [L1].
- Collection of tissue aspirates (bone marrow is preferred) or biopsy specimens is recommended ⁵⁴ [L1].
- Serum should be collected for antibody testing ⁵⁴ [L1].
- Blood sample should be obtained in immunocompromised patients ⁵⁴ [L1].
- Differential diagnoses include ⁶⁰:
 - $\circ \quad \text{Chronic malaria.}$
 - $\circ \quad \text{Schistosomiasis.}$
 - \circ Other systemic infections.

Multiple diagnostic approaches are available to detect leishmaniasis ⁵⁴ [L1]:

- Visualisation of the characteristic amastigote in smears.
- Histopathology.
- Parasite isolation by *in vitro* culture.
- Molecular detection of parasite DNA.

17.3.5.2 Leishmaniasis Management

Treatment should be given after confirmation of disease ⁶⁰ [L1]. Empiric treatment may be indicated on the basis of an individualised risk-benefit assessment ⁵⁴ [L1].

Immunocompetent patients with cutaneous leishmaniasis and low risk for mucocutaneous leishmaniasis may be observed without treatment until the lesion is healing spontaneously ⁵⁴ [L1, RGA].

All patients diagnosed as with visceral leishmaniasis require prompt and complete treatment ⁵⁵ [L1, RGA]. Available antileishmanial medicines are listed in *Table 17.3.5.2*.

Treatment	Additional Notes	
Old World Cutaneous Leishmaniasis		
Paromomycin ointments		
Intralesional pentavalent antimonials		
Thermotherapy	Applications of localised heat (50°C for 30 sec).	
Петнопетару	Local anaesthesia is required.	
Cryotherapy	Application of liquid nitrogen (-195°C) to the lesion.	
New World Cutaneous Leishmaniasis		
Pentavalent antimonials	Less effective in children under 5 years of age.	
Pentamidine		
Paromomycin sulfate		
Miltefosine		
Ketoconazole		
New World Mucocutaneous Leishmaniasis		
Pentavalent antimonials	Consider a combination with oral pentoxifylline.	
Amphotericin B deoxycholate		
Liposomal amphotericin B		
Pentamidine		
Miltefosine		
Visceral Leishmaniasis		
Pentavalent antimonials:		
Sodium stibogluconate	First-line treatment.	
Meglumine antimoniate		
Amphotericin B deoxycholate		
Lipid formulations of amphotericin B	Second-line treatment.	
Miltefosine	Compination treatment is preferred.	
Paromomycin (aminosidine)	wonotherapy should be inflited to liposofial amphotencin B.	

 Table 17.3.5.2: Selection of Medication for Treatment of Leishmaniasis ⁶⁰.

17.3.6 Loiasis

17.3.6.1 Loiasis Diagnosis

The standard diagnostic test for the diagnosis of loiasis is demonstration of microfilariae on a daytime (10AM to 2PM) Giemsa-stained thin or thick blood smear ⁵⁶ [L1]. The timing of the blood smear should be adjusted to reflect local time of the infection.

Consider concentration techniques in patients with low numbers of circulating microfilariae ⁵⁶ [L1]:

- Nuclepore[™] filtration.
- Knott's concentration.
- Saponin lysis.

17.3.6.2 Loiasis Management

All patients with loiasis must be assessed by a specialist as treatment is very complex and comprises high risks for encephalopathy and blindness ⁵⁶ [L1, RGA].

Surgical excision of migrating adult worms may be performed ⁵⁶ [**L1**, **RGA**]. Antiparasitic medications are still required after surgery ⁵⁶ [**L1**, **RGA**]. Available medicines are listed in the table below.

Medication	Indication*
Diethylcarbamazine	For patients with symptomatic loiasis and MF/mL <8,000. Contraindicated in patients with with onchocerciasis because of the risk of blindness and/or severe exacerbation of skin disease.
Albendazole	For patients with symptomatic loiasis and MF/mL <8,000 who have failed 2 rounds of diethylcarbamazine. For patients with symptomatic loiasis and MF/ml ≥8,000 to reduce level to <8,000 prior to treatment with diethylcarbamazine.
Apheresis followed by diethylcarbamazine	For patients with symptomatic loiasis, with MF/mL ≥8,000.

 Table 17.3.6.2: Selection of Medication for Treatment of Loiasis ⁵⁶.

* - The selection of treatment should be based on the number of microfilariae of Loa Loa per mL.

18 Key Considerations for Patient Preferences

Patient preferences refer to patient perspectives, beliefs, expectations, and goals for health and life, and to the steps employed by individuals in assessing the potential benefits, harms, costs, and limitations of the management options in relation to one another. Patients may have preferences when it comes to defining their problems, identifying the range of management options and selecting or ranking the outcomes used to compare these options.

It is important for healthcare professionals to develop an understanding of the patient as an individual and the unique way in which each person experiences a condition and its impact on their life.

The following recommendations are therefore made for physicians and other healthcare professionals regarding general principles of patient care in Qatar:

- **Respect Patients:** Treat patients with respect, kindness, dignity, courtesy and honesty. Ensure that the environment is conducive to discussion and that the patient's privacy is respected, particularly when discussing sensitive, personal issues. Ask the patient how they wish to be addressed and ensure that their choice is respected and used.
- Maintain Confidentiality: Respect the patient's right to confidentiality and avoid disclosing or sharing patients' information without their informed consent. In this context, students and anyone not directly involved in the delivery of care should first be introduced to the patient before starting consultations or meetings, and let the patient decide if they want them to stay.
- **Clarify Third-Party Involvement:** Clarify with the patient at the first point of contact whether and how they like their partner, family members or carers to be involved in key decisions about their care or management and review this regularly. If the patient agrees, share information with their partner, family members or carers.
- **Obtain Informed Consent:** Obtain and document informed consent from patients, in accordance with MOPH policy and guidance.
- Encourage Shared Decision Making: Ensure that patients are involved in decision making about their own care, or their dependent's care, and that factors that could impact the patient's participation in their own consultation and care including physical or learning disabilities, sight, speech or hearing impairments and problems with understanding, reading or speaking English are addressed.
- Disclose Medical Errors: Disclose errors when they occur and show empathy to patients.
- Ensure Effective Communication: Explore ways to improve communication including using pictures, symbols or involving an interpreter or family members. Avoid using medical jargon. Use words the patient will understand and confirm understanding by asking questions.
- **Ensure Continuity of Care:** Provide clear and timely sharing of patient information between healthcare professionals especially at the point of any transitions in care.

19 Performance Measures

A list of potential performance measures is given below in *Table 19.1*.

Number	Numerator	Denominator
SSTI01	The number in the denominator who received antibiotics as per guideline recommendations.	The number of patients who were diagnosed with an SSTI in the last 12 months and were prescribed antibiotic treatment.
SSTI02	The number in the denominator in whom antibiotics were de-escalated following the culture result.	The number of patients diagnosed with an SSTI requiring antibiotic therapy in the last 12 months with positive culture results.

 Table 19.1: Performance Measures.

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Appendix: Detailed Description of the Literature Search

A systematic search for existing literature on the low back pain was performed in the period June 7th – July 1st, 2019.

The search for clinical practice guidelines on the diagnosis and/or management of skin and soft tissue infections was performed in the *PubMed* database and websites of relevant organisations and societies including the *World Health Organization, American Academy of Family Physicians, American Academy of Dermatology Association,* and other. The present guideline is primarily based on practice guidelines by *Infectious Diseases Society of America* and *World Society of Emergency Surgery* and the *Surgical Infection Society Europe* and is supplemented with other relevant studies.

Peer-reviewed scientific publications were found in PubMed and via *Google Scholar* Internet search engine. Non-peer reviewed studies were identified in *bioRxiv*. Books were checked on PubMed. Information published on medical websites and drug prescribing information sheets were found via Google search engine.

The included publications were identified using the term "skin or soft tissue infection" and specified with the following terms in combinations:

Management, risk factors, aetiology, pathogens, prognosis, prevalence, presentation, symptoms, examination, imaging, differential/alternative diagnosis, screening, primary/secondary care, (non)complicated, abscess, folliculitis, furuncle, furunculosis, carbuncle, impetigo, human/animal bites, erysipelas, cellulitis, necrotizing, gangrene, herpes, measles, rubella, mononucleosis, diabetic foot, referral criteria, emergency, management, treatment, antibiotics, antimicrobials, antiseptic, debridement, wound, paediatric, children, recovery, outcome, follow-up, performance, quality measures/standards.

Figure A.1 on the next page demonstrates graphically the results of the search and application of exclusion criteria.



Fig A.1: Literature search results and application of exclusion criteria.

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