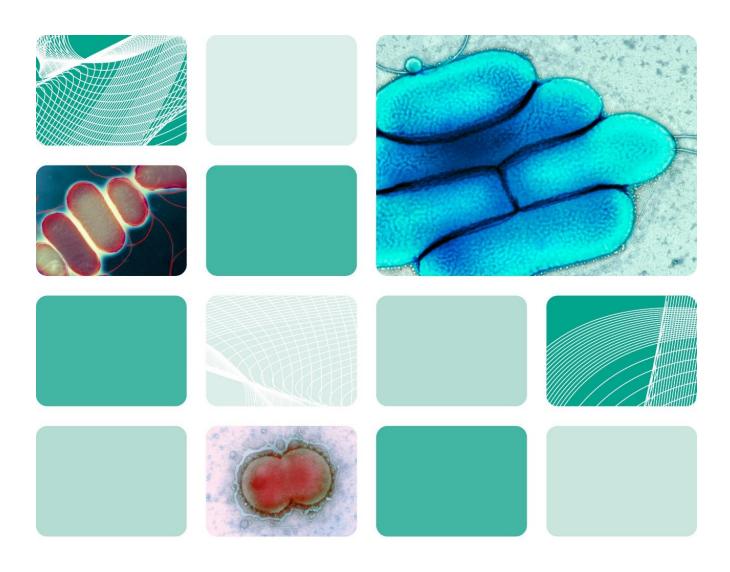


UK Standards for Microbiology Investigations

Investigation of pus and exudates



Acknowledgments

UK Standards for Microbiology Investigations (UK SMIs) are developed under the auspices of UKHSA working in partnership with the partner organisations whose logos are displayed below and listed on the UK SMI website. UK SMIs are developed, reviewed and revised by various working groups which are overseen by a steering committee.

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UK SMIs are produced in association with:













































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Amendment table

Each UK SMI document has an individual record of amendments. The amendments are listed on this page. The amendment history is available from standards@ukhsa.gov.uk.

Any alterations to this document should be controlled in accordance with the local document control process.

Amendment number/date	11/03.10.25				
Issue number discarded	6.2				
Insert issue number	6.3				
Section(s) involved	Amendment				
	This is an administrative point change.				
	The content of this UK SMI document has not changed.				
	The last scientific and clinical review was conducted on 22/08/2016.				
	Hyperlinks throughout document updated to Royal College of Pathologists website.				
Whole document.	Public Health England replaced with UK Health Security Agency throughout the document, including the updated Royal Coat of Arms				
	Partner organisation logos updated.				
	Broken links to devolved administrations replaced.				
	References to NICE accreditation removed.				
	Scope and Purpose replaced with General and Scientific information to align with current UK SMI template.				

Amendment no/date.	10/22.08.16
Issue no. discarded.	6.1
Insert issue no.	6.2
Section(s) involved	Amendment

Appendix.	Updated to reflect section 4.5.1 culture media, conditions and organisms table.
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Amendment no/date.	9/08.08.16
Issue no. discarded.	6
Insert issue no.	6.1
Section(s) involved	Amendment
4.3.2.	Section regarding swabs has been clarified.

Amendment no/date.	8/20.06.16				
Issue no. discarded.	5.2				
Insert issue no.	6				
Section(s) involved	Amendment				
Title.	Changed to 'investigation of pus and exudates'.				
Whole document.	References reviewed and updated.				
Type of specimen.	Pus or exudate.				
Key recommendations.	Key recommendation section included.				
	Document streamlined and re-structured.				
Introduction.	Information regarding skin related abscesses and post-operative wound infections removed and transferred to B 11 - Investigation of swabs from skin and superficial soft tissue infections.				
	Inclusion of throat and neck abscesses.				
Technical information /limitations.	Inclusion of information on selective media and rapid methods.				
Safety considerations.	Recommendations included regarding the use of Class I or Class II microbiological safety cabinets where Gram negative coccobacilli are identified.				
Specimen collection.	Samples of pus are preferred to swabs.				
Culture and investigation.	Section 4.5.1 - Table updated to include specimen type. Neomycin fastidious anaerobic agar replaced by				
	selective anaerobic agar for all specimens.				

	Cooked meat broth or equivalent added as alternative to fastidious anaerobic broth.
	GN Medium (NAV) replaced with selective Gram negative anaerobe medium.
	Minor changes to incubation and culture reads throughout for consistency.
	Section 4.6 - Minimum level of identity updated for the following organisms: anaerobes, β-haemolytic streptococci, enterobacteriaceae and yeast.
	Consider sending staphylococci isolates from post mortem samples for toxin testing.
	Section 4.7 - Antimicrobial susceptibility testing section updated. Recommendations for selective reporting are not included.
	Section 4.9 - Consider sending <i>S. aureus</i> isolates for toxin testing where appropriate clinical details are provided.
	Updated in line with bacteriology template.
Reporting procedure.	Report antimicrobial susceptibilities as clinically indicated.
Appendix.	Updated to reflect section 4.5.1 culture media, conditions and organisms table.

1 General information

View general information related to UK SMIs.

2 Scientific information

View scientific information related to UK SMIs.

3 Scope of document

Type of specimen

Pus, exudate

This UK SMI describes the processing and microbiological investigation of pus and exudate specimens from abscesses and infections which are deep seated or associated with specific organs (including the skin).

Investigations of these specimens for parasitic infections are in <u>UK SMI B 31</u> - <u>Investigations of specimens other than blood for parasites</u>, whereas investigations for mycobacterial cultures are in <u>UK SMI B 40 – Investigations of specimens for Mycobacterium</u> species.

For information regarding genital tract abscesses and infections refer to <u>UK SMI S 6 – infectious syndromes affecting the genitourinary tract and reproductive organs.</u>

For information on skin and soft tissue infections and regarding testing of swabs of pus refer to <u>UK SMI B 11 - Investigation of swabs from skin and superficial soft tissue infections</u>.

This UK SMI should be used in conjunction with other UK SMIs.

Key recommendations

Samples should be transported to the laboratory and processed rapidly.

Gram stain should be undertaken on all samples.

Gram stain results should be taken into consideration when choosing which investigations to carry out.

Significant microscopy, culture and sensitivity results should be issued as soon as they become available.

A mechanism for urgent reporting should be in place to communicate key, significant results.

4 Introduction

Abscesses are accumulations of pus in tissue and any organism isolated from them may be of significance. They occur in many parts of the body as superficial infections or as deep-seated infections associated with any internal organ. Many abscesses are caused by *Staphylococcus aureus* alone, but others are mixed infections. Anaerobes

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are predominant isolates in intra-abdominal abscesses and abscesses in the oral and anal areas. Members of the "Streptococcus anginosus" group and Enterobacteriaceae are also frequently present in lesions at these sites.

Bartholin gland abscesses and tubo-ovarian abscesses are considered in <u>UK SMI S 6</u> – <u>infectious syndromes affecting the genitourinary tract and reproductive organs</u>. Processing of specimens for *Mycobacterium* species, for example from subcutaneous cold abscesses, is described in <u>UK SMI B 40 – Investigation of specimens for Mycobacterium species</u>.

Brain abscess¹⁻⁴

Brain abscesses are serious and life-threatening.

Sources of abscess formation include⁵:

- direct contiguous spread from chronic otitic or paranasal sinus infection
- metastatic haematogenous spread either from general sepsis or secondary to chronic suppurative lung disease
- penetrating wounds
- surgery
- cryptogenic (ie source unknown)

Brain abscesses of dental origin are rare⁶. The mortality rate of these abscesses is high even when appropriately treated⁷.

Treatment of brain abscesses involves the drainage of pus and appropriate antimicrobial therapy⁵. Brain stem abscesses have a poor prognosis due to their critical anatomical location⁸.

Bacteria isolated from brain abscesses are usually mixtures of aerobes and obligate anaerobes, and the prevalent organism may vary depending upon geographical location, age and underlying medical conditions. The most commonly isolated organisms include⁹⁻¹¹:

- anaerobic streptococci
- anaerobic Gram negative bacilli
- "Streptococcus anginosus" group
- Enterobacteriaceae
- Streptococcus pneumoniae
- β-haemolytic streptococci
- S. aureus

Organisms commonly isolated vary according to the part of the brain involved. Many other less common organisms, for example *Haemophilus* species, may be isolated^{9,10,12}. *Nocardia* species often exhibit metastatic spread to the brain from the lung. Any organism isolated from a brain abscess must be regarded as clinically significant.

Organisms causing brain abscesses following trauma may often be environmental in origin, such as *Clostridium* species or skin derived, such as staphylococci and *Propionibacterium* species¹³.

Brain abscesses due to fungi are rare. Aspergillus brain abscess can occur in patients who are neutropenic. Zygomycosis (mucormycosis) is an uncommon opportunistic infection caused by *mucoraceous moulds, for example Lichtheimia (formerly Mycocladus, Absidia)* and related fungi. *Scedosporium apiospermum* (*Pseudallescheria boydii*) enters the body and spreads haematogenously¹⁴.

The use of culture based methods for organism identification is time consuming; molecular tests are becoming popular resulting in improved management of brain abscesses³.

Breast abscess

Breast abscesses occur in both lactating and non-lactating women. In the former, infections are commonly caused by *S. aureus*, but may be polymicrobial, involving anaerobes and streptococci^{7,15}. Signs include discharge from the nipple, swelling, oedema, firmness and erythema.

In non-lactating women a subareolar abscess forms often with an inverted or retracted nipple. Mixed growths of anaerobes are usually isolated¹⁶. Some patients require surgery involving complete duct excision¹⁶. Abscesses may also be caused by *Pseudomonas aeruginosa* and *Proteus* species¹⁷.

Dental abscess¹⁸⁻²⁰

Dental abscesses involve microorganisms colonising the teeth that may become responsible for oral and dental infections, leading to dentoalveolar abscesses and associated diseases. They may also occur as a direct result of trauma or surgery.

Periodontal disease involves the gingiva and underlying connective tissue, and infection may result in gingivitis or periodontitis²¹.

Organisms most commonly isolated in acute dentoalveolar abscesses are facultative or strict anaerobes. The most frequently isolated organisms are anaerobic Gram negative rods; however other organisms have also been isolated. Examples include^{18,19}:

- α-haemolytic streptococci
- anaerobic Gram negative bacilli
- anaerobic streptococci
- "S. anginosus" group
- Aggregatibacter actinomycetemcomitans
- Actinomyces species
- spirochaetes

Aspiration of dental abscesses may be taken, where possible, to assist in the identification of the causative organism(s). Swabs may be contaminated with superficial commensal flora. In cases of intraosseal abscess, swabs can be useful, but only if taken from a disinfected site.

Liver abscess²²

Liver abscesses can be amoebic or bacterial (so-called pyogenic) in origin or, more rarely, a combination of the two.

Pyogenic liver abscesses usually present as multiple abscesses and are potentially life-threatening. They require prompt diagnosis and therapy by draining and/or aspirating purulent material, although it is possible to treat liver abscesses with antibiotics alone. They occur in older patients than those with amoebic liver abscesses, and are often secondary to a source of sepsis in the portal venous distribution.

Examples of the sources of pyogenic liver abscess include:

- · biliary tract disease
- extrahepatic foci of metastatic infection
- surgery
- trauma

Many different bacteria may be isolated from pyogenic liver abscesses. The most common include²³⁻²⁵:

- Enterobacteriaceae
- Bacteroides species
- · Clostridium species
- anaerobic streptococci
- "S. anginosus" group
- enterococci
- P. aeruginosa
- B. pseudomallei (in endemic areas)

Other causes include Candida species.

Amoebic liver abscesses arise as a result of the spread of *Entamoeba histolytica* via the portal vein from the large bowel which is the primary site of infection (investigation of amoebae is described in <u>UK SMI B 31 – Investigation of specimens other than blood for parasites</u>).

Hydatid cysts may also occur as fluid-filled lesions in the liver. However, the clinical presentation is usually different from that of liver abscesses (refer to <u>UK SMI B 31 – Investigation of specimens other than blood for parasites</u>). Cysts may become super-infected with gut flora and progress to abscess formation.

Lung abscess

Lung abscesses involve the destruction of lung parenchyma and present on chest radiographs as large cavities often exhibiting air-fluid levels. This may be secondary to aspiration pneumonia, in which case the right middle zone is most frequently affected. Other organisms may give rise to multifocal abscess formation and the presence of widespread consolidation containing multiple small abscesses (<2 cm diameter) is sometimes referred to as necrotising pneumonia. Pneumonia caused by *S. aureus* and

Klebsiella pneumoniae may show this picture (refer to <u>UK SMI B 57 – Investigation of bronchoalveolar lavage, sputum and associated specimens</u>).

Lung abscesses most often follow aspiration of gastric or nasopharyngeal contents as a consequence of loss of consciousness, resulting for example from alcohol excess, cerebrovascular accident, drug overdose, general anaesthesia, seizure, diabetic coma, or sepsis. Other predisposing factors include oesophageal or neurological disease, tonsillectomy and tooth extraction.

Lung abscesses may arise from endogenous sources of infection. The bacteria involved in these cases are generally from the upper respiratory tract and anaerobes are often implicated, secondarily infecting consolidated lung after aspiration from the upper respiratory tract. Nosocomial infections involving *S. aureus*, *S. pneumoniae*, *Klebsiella* species and other organisms may also occur.

B. pseudomallei may cause lung abscesses or necrotising pneumonia in those who have visited endemic areas (mainly South East Asia and Northern Australia) especially in diabetics²⁶.

Nocardia infection is most often seen in the lung where it may produce an acute, often necrotising, pneumonia²⁷. This is commonly associated with cavitation. It may also produce a slowly enlarging pulmonary nodule with pneumonia, associated with empyema. Nocardiosis, almost always occurring in a setting of immunosuppression, may present as pulmonary abscesses.

Actinomyces species cause a thoracic infection that may involve the lungs, pleura, mediastinum or chest wall. Cases often go unrecognised until empyema or a chest wall fistula develops. Aspiration of oral contents is a risk factor for the development of thoracic actinomycosis, thus predisposing conditions include alcoholism, cerebral infarction, drug overdose, general anaesthesia, seizure, diabetic coma or sepsis. Abscesses as a result of blood borne spread of infection from a distant focus may occur in conditions such as infective endocarditis.

Lemierre's syndrome (or necrobacillosis) originates as an acute oropharyngeal infection usually in a young adult. Infective thrombophlebitis of the internal jugular vein leads to septic embolisation and metastatic infection. The lung is most frequently involved but multifocal abscesses may develop. *Fusobacterium necrophorum* is the most common pathogen isolated from blood cultures in patients with this syndrome²⁸.

Aspergillus species have been isolated from lung abscesses in patients who are immunocompromised.

Pancreatic abscess

Pancreatic abscesses are potential complications of acute pancreatitis. Infections are often polymicrobial and common isolates include *Escherichia coli*, other Enterobacteriaceae, enterococci and anaerobes; longer-standing collections, especially after prolonged antibiotic therapy, are often infected with coagulase negative staphylococci and *Candida* species²⁹.

Perirectal abscess

Perirectal abscesses are encountered in patients with predisposing factors. These include³⁰:

immunodeficiency

Investigation of pus and exudates

- malignancy
- rectal surgery
- ulcerative colitis

They are often caused by³¹:

- anaerobes
- Enterobacteriaceae
- streptococci
- S. aureus

Pilonidal abscess

Pilonidal abscesses result from infection of a pilonidal sinus. Anaerobes and Enterobacteriaceae are usually isolated, but they may be caused by S. aureus and β-haemolytic streptococci³².

Prostatic abscess³³

Abscesses within the prostate may be caused by, or associated with³⁴:

- diabetes mellitus
- · acute and chronic prostatitis
- · instrumentation of the urethra and bladder
- lower urinary tract obstruction
- · haematogenous spread of infection

Organisms that may cause prostatic abscesses include³⁵:

- E. coli and other Enterobacteriaceae
- enterococci
- anaerobes
- Neisseria gonorrhoea
- S. aureus³³

Prostatic abscesses can act as reservoirs for *Cryptococcus neoformans* resulting in relapses of infection with this organism³⁶.

Psoas abscess

Psoas abscesses may be seen as secondary infections to:

- appendicitis
- diverticulitis
- osteomyelitis of the spine
- infection of a disc space
- bacteraemia

perinephric abscess

Pus tracks under the sheath of the psoas muscle. Infection often occurs in drug abusers after injection into the ipsilateral femoral vein.

Psoas abscesses are predominantly caused by^{37,38}:

- Enterobacteriaceae
- Bacteroides species
- S. aureus
- streptococci
- Mycobacterium tuberculosis

Renal abscess

Renal abscesses are typically caused by Gram negative bacilli and result from ascending urinary tract infection, pyelonephritis, renal calculi or sepsis³⁹.

Renal abscesses are localised in the renal cortex and may occur as a result of, for example, *Staphylococcus aureus* bacteraemia. Pyuria may also be present, but urine culture is usually negative. Renal abscesses are increasingly being seen as complications of acute pyelonephritis caused by Gram negative bacilli. The rare condition of emphysematous pyelonephritis, which results in multifocal intrarenal abscesses and gas formation within the renal parenchyma, is usually seen in diabetic patients or as a complication of renal stones. The commonest cause is *Escherichia coli* and the condition carries a 70% mortality rate.

Perinephric abscess are an uncommon complication of UTI, which usually affects patients with one or more anatomical or physiological abnormalities⁴⁰. The abscess may be confined to the perinephric space or extend into adjacent structures. Pyuria, with or without positive culture, is normally, but not invariably seen on examination of urine. Causative organisms are usually Gram negative bacilli, but can also be staphylococci or *Candida* species. Mixed infections have also been reported.

Salivary gland abscess

There are three pairs of major salivary glands; the parotid, submandibular and sublingual. Parotic abscesses are more commonly seen in the elderly. Common organisms include:

- S. aureus
- anaerobes

Spinal epidural abscess⁴¹

Spinal epidural abscesses may occur in patients with:

- predisposing disease (such as diabetes)
- prior infection elsewhere in the body which may serve as a source for haematogenous spread
- abnormality of, or trauma to, the spinal column (often involving invasive medical procedures such as epidural catheterisation)

The most common isolate is *S. aureus*⁴². *Staphylococcus epidermidis* may be isolated in patients following invasive spinal manipulation. Streptococci (α -haemolytic, β -haemolytic and *S. pneumoniae*), Enterobacteriaceae and pseudomonads may also be isolated^{42,43}.

Subphrenic abscess

Subphrenic abscesses occur immediately below the diaphragm, often as a result of⁴⁴:

- gastric, duodenal or colonic perforation
- acute cholecystitis
- procedures on the liver and upper part of the gastrointestinal tract
- ruptured appendix
- trauma

Subphrenic abscesses are caused by mixed infections from the normal gastrointestinal flora⁴⁴.

Throat/neck abscess

Throat and neck abscess are relatively common⁴⁵⁻⁴⁷.

Causative organisms include^{45,48}:

- β-haemolytic streptococci
- anaerobes

Surgical incision and drainage may be undertaken through intraoral or extra oral procedures.

Unusual cases of abscess formation

Unusual cases of abscess formation can occur in patients with many underlying conditions and may be caused by a vast range of organisms⁴⁹⁻⁵⁶. Any organism isolated from abscess pus is potentially significant.

Actinomycosis is a chronic suppurative infection characterised by chronic abscess formation with surrounding fibrosis. It is rare and usually follows perforation of a viscous, trauma or surgery. It is caused by *Actinomyces israelii*, usually in mixed culture with other bacteria⁵⁷.

Abscess formation is most often associated with the gastrointestinal tract, the jaw and the pelvis. Other areas of the body may be involved and the formation of abdominal abscesses may occur. Thoracic involvement occurs in 15% of cases of actinomycosis. Pulmonary actinomycosis can be difficult to diagnose prior to cutaneous involvement, which results in direct extension through the chest wall. The disease progresses to form a chronic indurated mass with draining fistulae. Material should be drained from abscesses and biopsies taken. Skin biopsies may reveal the presence of organisms (refer to UK SMI B 17 – Investigation of tissues and biopsies from deep-seated sites and organs)

"Sulphur granules" are sought in the pus specimen⁵⁸. These are discharged from actinomycosis abscesses. Sulphur granules are colonies of organisms forming a filamentous inner mass which is surrounded by host reaction. They are formed only *in vivo*. They are hard, buff to yellow in colour, and have a clubbed surface.

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Intra-abdominal sepsis

Intra-abdominal sepsis is infection occurring in the normally sterile peritoneal cavity⁵⁹. The term covers primary and secondary peritonitis, as well as intra-abdominal abscesses.

Primary peritonitis is infection of the peritoneal fluid in which no perforation of a viscous has occurred. Infection usually arises via haematogenous spread from an extra-abdominal source and is often caused by a single pathogen⁵⁹. It is common in patients with ascites following hepatic failure. In females it may also result from organisms ascending the genital tract (refer to <u>UK SMI S 6 – infectious syndromes affecting the genitourinary tract and reproductive organs</u>).

Secondary peritonitis is acute, suppurative inflammation of the peritoneal cavity usually resulting from bowel perforation or postoperative gastrointestinal leakage. Secondary peritonitis is most often treated with a combination of drainage and antibiotics.

The most frequent isolates encountered in intra-abdominal sepsis with secondary peritonitis are derived from the normal gastrointestinal flora. Anaerobic bacteria are isolated from the majority of cases with *Bacteroides* species being isolated. However, infections are usually polymicrobial and organisms that have been isolated include:

- Enterococcus species
- Bacteroides species
- pseudomonads
- Peptostreptococcus species
- yeasts (mostly Candida species)
- β-haemolytic streptococci
- Clostridium species
- Enterobacteriaceae

Tuberculous peritonitis is a rare disease in the UK. It is more common on the Indian sub-continent, so it is important to consider this in immigrants from that area. In most cases a primary pulmonary focus is present with secondary spread of *Mycobacterium tuberculosis* (refer to <u>UK SMI B 40 – Investigation of specimens for *Mycobacterium* species).</u>

5 Technical information/limitations

Limitations of UK SMIs

The recommendations made in UK SMIs are based on evidence (eg sensitivity and specificity) where available, expert opinion and pragmatism, with consideration also being given to available resources. Laboratories should take account of local requirements and undertake additional investigations where appropriate. Prior to use, laboratories should ensure that all commercial and in-house tests have been validated and are fit for purpose.

Selective media in screening procedures

Selective media which does not support the growth of all circulating strains of organisms may be recommended based on the evidence available. A balance therefore must be sought between available evidence, and available resources required if more than one media plate is used.

Specimen containers^{60,61}

UK SMIs use the term "CE marked leak proof container" to describe containers bearing the CE marking used for the collection and transport of clinical specimens. The requirements for specimen containers are given in the EU in vitro Diagnostic Medical Devices Directive (98/79/EC Annex 1 B 2.1) which states: "The design must allow easy handling and, where necessary, reduce as far as possible contamination of, and leakage from, the device during use and, in the case of specimen receptacles, the risk of contamination of the specimen. The manufacturing processes must be appropriate for these purposes".

Rapid methods

To reduce turnaround times, rapid identification and sensitivity tests may be performed in conjunction with routine methods where appropriate. A variety of rapid identification and sensitivity methods have been evaluated; these include molecular techniques and the Matrix Assisted Laser Desorption Ionisation Time-of-Flight (MALDI-TOF)^{62,63}. It is important to ensure that fresh cultures of pure single isolates are tested to avoid reporting misleading results.

Laboratories should follow manufacturers' instructions and all rapid tests must be validated and be shown to be fit for purpose prior to use.

6 Safety considerations^{60,61,64-78}

6.1 Specimen collection, transport and storage^{60,61,64-67}

Use aseptic technique.

Collect specimens in appropriate CE marked leak proof containers and transport in sealed plastic bags.

Avoid accidental injury when pus is aspirated.

Compliance with postal, transport and storage regulations is essential.

6.2 Specimen processing^{60,61,64-78}

ContGram positiveainment Level 2.

If infection with a Hazard Group 3 organism eg *Mycobacterium* species, *Paracoccidioides brasiliensis* or *Brucella* species is suspected, all specimens must be processed in a microbiological safety cabinet under full Containment Level 3 conditions. Thus initial examination and all follow up work on specimens from patients with suspected *Mycobacterium* species, or suggesting a diagnosis of blastomycosis, coccidioidomycosis, histoplasmosis, paracoccidioidomycosis or penicilliosis must be performed inside a microbiological safety cabinet under full Containment Level 3 conditions.

It is recommended that all Gram negative coccobacilli from sterile sites should be processed in a Class I or Class II microbiological safety cabinet until Hazard Group 3 pathogens (ie Brucella) have been definitively excluded⁷⁹.

Laboratory procedures that give rise to infectious aerosols must be conducted in a microbiological safety cabinet⁷⁰.

Any grinding of sulphur granules should be performed in a microbiological safety cabinet.

Prior to staining, fix smeared material by placing the slide on an electric hotplate (65-75°C), under the hood, until dry. Then place in a rack or other suitable holder.

Note: Heat-fixing may not kill all *Mycobacterium* species⁸⁰. Slides should be handled carefully.

Specimen containers must also be placed in a suitable holder.

Refer to current guidance on the safe handling of all organisms documented in this UK SMI.

The above guidance should be supplemented with local COSHH and risk assessments.

7 Specimen collection

7.1 Type of specimens

Pus, exudate

7.2 Optimal time and method of collection⁸¹

For safety considerations refer to Section 6.1.

Collect specimens before antimicrobial therapy where possible⁸¹.

Samples of pus are preferred to swabs. However, pus swabs are often received. When using a swab disinfect the superficial areas first. The deepest part of the wound should be sampled, avoiding the superficial microflora.

The specimen will usually be collected by a medical practitioner. Cleaning the site with sterile saline or 70% alcohol is recommended by some sources⁸².

Collect specimens other than swabs into appropriate CE marked leak proof containers and place in sealed plastic bags.

Unless otherwise stated, swabs for bacterial and fungal culture should then be placed in appropriate transport medium⁸³⁻⁸⁷.

7.3 Adequate quantity and appropriate number of specimens⁸¹

Ideally, a minimum volume of 1mL of pus should be submitted.

Swabs are not the optimal sample type. However, if received, swabs should be well soaked in pus. Refer to <u>UK SMI B 11 - Investigation of swabs from skin and superficial soft tissue infections</u>.

Numbers and frequency of specimen collection are dependent on clinical condition of patient.

8 Specimen transport and storage^{60,61}

8.1 Optimal transport and storage conditions

For safety considerations refer to Section 6.1.

Specimens should be transported and processed as soon as possible⁸¹.

The volume of specimen influences the transport time that is acceptable. Large volumes of purulent material maintain the viability of anaerobes for longer^{88,89}.

The recovery of anaerobes in particular is compromised if the transport time is delayed.

If processing is delayed, refrigeration is preferable to storage at ambient temperature.

9 Specimen processing/procedure^{60,61}

9.1 Test selection

Divide specimen on receipt for appropriate procedures such as examination for parasites (<u>UK SMI B 31 – Investigation of specimens other than blood for parasites</u>) and culture for *Mycobacterium* species (<u>UK SMI B 40 – Investigation of specimens for Mycobacterium species</u>), depending on clinical details.

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9.2 Appearance

Describe presence or absence of sulphur granules (if sought).

9.3 Sample preparation

9.3.1 Pre-treatment

Exudates

Centrifuge in a sterile, capped, conical-bottomed container at 1200 x g for 5-10 min.

Transfer the supernatant with a sterile pipette, leaving approximately 0.5mL, to another CE marked leak proof container in a sealed plastic bag for additional testing if required.

Resuspend the deposit in the remaining fluid.

Supplementary

Wash any sulphur granules that are present in saline.

Suspend an aliquot of pus containing sulphur granules in sterile water or saline in a CE marked leak proof container in a sealed plastic bag. Agitate gently to wash pus from the granules.

Grind the washed granules in a small amount of sterile water or saline, with a sterile tissue grinder (Griffiths tube or unbreakable alternative) or a pestle and mortar.

Use this homogenised sample to make a smear for Gram staining and to inoculate agar plates.

Note 1: All grinding of sulphur granules should be performed in a microbiological safety cabinet.

Note 2: If a fungal infection is suspected then grinding of the whole specimen should be avoided. This is to prevent damaging hyphae that would result in a reduced yield, particularly with zygomycetes.

9.3.2 Specimen processing

Pus

Inoculate agar plates and enrichment broth with the pus or centrifuged deposit with a sterile pipette (refer to <u>UK SMI Q 5 – Inoculation of culture media for bacteriology</u>).

If sulphur granules are present, these should be ground and included in the culture.

For the isolation of individual colonies, spread inoculum with a sterile loop.

All additional pus/fluid from the specimen should be stored at 4°C for at least 7 days after the issue of the final report.

Swabs

If a swab of pus is received, follow the recommendations in <u>UK SMI B 11 – Investigation</u> of swabs from skin and superficial soft tissue infections.

9.4 Microscopy

9.4.1 Standard

Swab

Prepare a thin smear on a clean microscope slide for Gram staining after performing culture (refer to UK SMI Q 5 – Inoculation of culture media for bacteriology).

Pus

Using a sterile pipette place one drop of neat specimen or centrifuged deposit (see section 9.5.1), as applicable, on to a clean microscope slide. Spread this using a sterile loop to make a thin smear for Gram staining (refer to UK SMI TP 39 - Staining procedures). The Gram film result should be used as a guide for supplementary cultures (eg fungal, Actinomyces) when appropriate.

9.4.2 Supplementary

Gram stain of sulphur granules

With care, either squash the sulphur granules that have been washed in saline between two slides using gentle pressure, or use the homogenised granules (see section 9.5.1) and make a thin smear for Gram staining.

Note: Any grinding of sulphur granules should be performed in a microbiological safety cabinet.

For microscopy, *Mycobacterium* species (<u>UK SMI B 40 – Investigation of specimens for Mycobacterium species</u>) and parasites (<u>UK SMI B 31 – Investigation of specimens other than blood for parasites</u>). For fungi and other staining procedures refer to <u>UK SMI TP 39 – Staining procedures</u>.

9.5 Culture and investigation

Inoculate each agar plate using a sterile pipette (<u>UK SMI Q 5 - Inoculation of culture media for bacteriology</u>).

For the isolation of individual colonies, spread inoculum with a sterile loop.

9.5.1 Culture media, conditions and organisms

Clinical details/	Specimen	Standard media	Incubation			Cultures read	Target organism(s)
conditions			Temp °C	Atmos	Time	read	organisin(s)
All clinical conditions	All pus and exudates	Blood agar	35-37	5–10% CO ₂	40- 48hr	daily	Any organism
	Ma ag Se an wit me 5µ	CLED/ MacConkey agar	35-37	Air	18- 24hr	≥18hr	
		Selective anaerobe agar with a metronidazole 5µg disc	35-37	Anaerobic	5 d	≥40hr and at 5 d	Anaerobes
		Fastidious anaerobic cooked meat broth or equivalent.	35-37	Air	5 d	N/A	Any organism

		Subculture if evidence of growth (≥40hr), or at day 5 to above media (excluding MacConkey)	35-37	As above	40- 48hr	≥40hr	
For these situat	ions, add the f	ollowing:					
Clinical details/	Specimen	Supplementary media	Incubati	on		Cultures read	Target organism(s)
conditions		media	Temp °C	Atmos	Time	reau	
Submandibul ar abscess	Pus	Chocolate agar	35–37	5–10% CO ₂	40 – 48hr	≥40hr	Fastidious organisms
Brain abscess							
Liver abscess							
Lung empyema/ abscess							
Psoas abscess							
Spinal abscess							
Actinomycosis (or where microscopy suggestive of actinomycete s)	Pus	Blood agar supplemented with metronidazole and nalidixic acid	35-37	Anaerobic	10 d	≥40hr, at 7 d and 10 d	Actinomyces species
Nocardiosis	Pus	Blood agar	35-37	5-10% CO ₂	16- 48hr	daily	Nocardia species*
Immunocomp romised or fungi suspected (Gram film or clinical details)	Pus	Sabouraud agar	28-30	Air	14 d	daily	Yeast Mould
Prostatic abscess	Pus	GC selective/ Chocolate agar	35-37	5-10% CO ₂	40- 48hr	≥40hr	N. gonorrhoeae
Primary peritonitis in females							
Clinical details/	Specimen	Optional media	a Incubation			Cultures read	Target organism(s)
conditions			Temp °C	Atmos	Time	IGAU	
When clinical details or microscopy suggestive of	Pus	Staph/strep selective agar	35-37	Air	40- 48hr	daily	S. aureus Streptococci

mixed infection	Selective Gram negative anaerobe medium	35-37	Anaerobic	Up to 5 d	≥40hr and 5 d	Gram negative anaerobes
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Other organisms for consideration - Fungi (<u>UK SMI B 39 – Investigation of dermatological specimens for superficial mycoses</u>) and *Mycobacterium* species (<u>UK SMI B 40 – Investigation of specimens for Mycobacterium species</u>)

9.6 Identification

Refer to individual UK SMIs for organism identification.

9.6.1 Minimum level of identification in the laboratory

Actinomycetes	species level UK SMI ID 10 – Identification of aerobic Actinomycetes
	UK SMI ID 15 – Identification of Actinomyces species
<u>Anaerobes</u>	genus level
	(in brain samples to species level)
<u>β-haemolytic streptococci</u>	species level
Coagulase negative staphylococci	"coagulase negative" level
<u>Enterobacteriaceae</u>	genus level
Yeast	species level
Mould	species level
<u>Neisseria</u>	species level
<u>Pseudomonads</u>	species level
S. aureus	species level
	(consider Panton-Valentine leukocidin (PVL) and toxin testing if appropriate clinical details)
	(consider toxin testing on samples from postmortem samples)
"S. anginosus" group	"S. anginosus" group level
Mycobacterium	UK SMI B 40 - Investigation of specimens for <i>Mycobacterium</i> species
Parasites	UK SMI B 31 - Investigation of specimens other than blood for parasites

Organisms may be further identified if this is clinically or epidemiologically indicated.

9.7 Antimicrobial susceptibility testing

Refer to <u>British Society for Antimicrobial Chemotherapy (BSAC)</u>, <u>EUCAST</u> and/or <u>CSLI</u> guidelines or manufacturer's validation for preparatory methods.

^{*}Refer to <u>UK SMI B 17 – Investigation of tissues and biopsies from deep-seated sites and organs</u>) for isolation of *Nocardia* species using specimen type, biopsy (this includes bronchoalveolar lavage (BAL)).

This UK SMI does not contain recommendations for the selective and restrictive reporting of susceptibilities to antimicrobials due to the diversity of organisms associated with pus and exudate samples. Local decisions on antimicrobial susceptibility testing should be subject to consultation that should include local antimicrobial stewardship groups.

9.7.1 Antimicrobial susceptibility testing and reporting table

N/A

9.8 Referral for outbreak investigations

N/A

9.9 Referral to reference laboratories

For information on the tests offered, turn around times, transport procedure and the other requirements of the reference laboratory <u>click here for user manuals and request forms</u>.

Organisms with unusual or unexpected resistance and whenever there is a laboratory or clinical problem, or anomaly that requires elucidation should be sent to the appropriate reference laboratory.

Consider sending *S. aureus* isolates for toxin testing where appropriate clinical details are provided. For example, isolates from post mortems where the specimen is suspected to be the cause of death should be sent for toxin testing.

Contact appropriate devolved national reference laboratory for information on the tests available, turnaround times, transport procedure and any other requirements for sample submission:

England

Wales

Scotland

Northern Ireland

10 Reporting procedure

10.1 Microscopy

Gram stain

Report on WBCs and organisms detected.

10.1.1 Microscopy reporting time

All results should be issued to the requesting clinician as soon as they become available, unless specific alternative arrangements have been made with the requestors.

Urgent results should be telephoned or transmitted electronically in accordance with local policies.

Investigation of pus and exudates

For the reporting of microscopy for fungi, *Mycobacterium* species and parasites (<u>UK SMI B 40 – Investigation of specimens for Mycobacterium species</u>) and parasites (<u>UK SMI B 31 – Investigation of specimens other than blood for parasites</u>).

10.2 Culture

The following results should be reported:

- · clinically significant organisms isolated
- other growth
- · absence of growth

Report on the presence of sulphur granules.

Also, report results of supplementary investigations: fungi, *Mycobacterium* species and parasites. (UK SMI B 31 – Investigation of specimens other than blood for parasites).

10.2.1 Culture reporting time

Interim or preliminary results should be issued on detection of potentially clinically significant isolates as soon as growth is detected, unless specific alternative arrangements have been made with the requestors.

Urgent results should be telephoned or transmitted electronically in accordance with local policies.

Final written or computer generated reports should follow preliminary and verbal reports as soon as possible.

10.3 Antimicrobial susceptibility testing

Report susceptibilities as clinically indicated; guidance on selective reporting is not included in this UK SMI. Prudent use of antimicrobials according to local and national protocols is recommended.

Generally, all resistant results should be reported as this is good practice and informs the user.

11 Referral to reference laboratories

For information on the tests offered, turnaround times, transport procedure and the other requirements of the reference laboratory see user manuals and request forms

Contact appropriate reference laboratory for information on the tests available, turnaround times, transport procedure and any other requirements for sample submission:

England

Wales

Scotland

Northern Ireland

Note: In case of sending away to laboratories for processing, ensure that specimen is placed in appropriate package and transported accordingly.

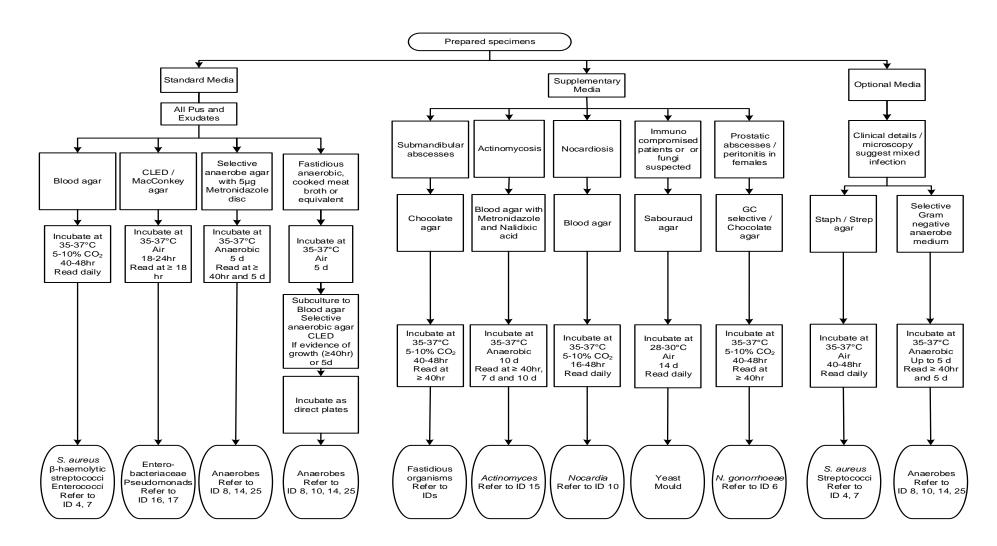
12 Public health responsibilities of diagnostic laboratories

Diagnostic laboratories have public health responsibility as part of their duties. Amongst these are additional local testing, or referral to further characterise the organism as required, primarily for public health purposes e.g. routine cryptosporidium detection; serotyping or microbial subtyping; and a duty to refer appropriate specimens and isolates of public health importance to a reference laboratory.

Diagnostic laboratory outputs inform public health intervention, and surveillance data is required to develop policy and guidance forming an essential component of healthcare. It is recognised that additional testing and referral of samples may entail some costs that has to be borne by the laboratory but in certain jurisdictions these costs are covered centrally.

Diagnostic laboratories should be mindful of the impact of laboratory investigations on public health and consider requests from the reference laboratories for specimen referral or enhanced information.

Algorithm: Investigation of pus and exudates⁹⁶



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References

An explanation of the reference assessment used is available in the <u>scientific</u> <u>information section on the UK SMI website</u>.

- 1. Slazinski T. Brain abscess. Crit Care NursClinNorth Am 2013;25:381-8.
- 2. Muzumdar D, Jhawar S, Goel A. Brain abscess: an overview. IntJSurg 2011;9:136-44.
- 3. Mishra AK, Dufour H, Roche PH, Lonjon M, Raoult D, Fournier PE. Molecular revolution in the diagnosis of microbial brain abscesses. EurJClinMicrobiolInfectDis 2014;33:2083-93.
- 4. Rau CS, Chang WN, Lin YC, Lu CH, Liliang PC, Su TM et al. Brain abscess caused by aerobic Gram-negative bacilli: clinical features and therapeutic outcomes. ClinNeurolNeurosurg 2002;105:60-5.
- 5. Akuzawa N, Osawa T, Totsuka M, Hatori T, Imai K, Kitahara Y et al. Secondary brain abscess following simple renal cyst infection: a case report. BMC Neurol 2014;14:130.
- 6. Yang J, Liu SY, Hossaini-Zadeh M, Pogrel MA. Brain abscess potentially secondary to odontogenic infection: case report. Oral Surg Oral Med Oral Pathol Oral Radiol 2014;117:e108-11.
- 7. Dabbas N, Chand M, Pallett A, Royle GT, Sainsbury R. Have the organisms that cause breast abscess changed with time?--Implications for appropriate antibiotic usage in primary and secondary care. Breast J 2010;16:412-5.
- 8. Hakan T, Ceran N, Erdem I, Berkman MZ, Goktas P. Bacterial brain abscesses: an evaluation of 96 cases. JInfect 2006;52:359-66.
- Prasad KN, Mishra AM, Gupta D, Husain N, Husain M, Gupta RK. Analysis of microbial etiology and mortality in patients with brain abscess. JInfect 2006;53:221-7.
- Carpenter J, Stapleton S, Holliman R. Retrospective analysis of 49 cases of brain abscess and review of the literature. EurJClinMicrobiolInfectDis 2007;26:1-11.
- 11. Ko SJ, Park KJ, Park DH, Kang SH, Park JY, Chung YG. Risk factors associated with poor outcomes in patients with brain abscesses. J Korean Neurosurg Soc 2014;56:34-41.
- 12. Han XY, Weinberg JS, Prabhu SS, Hassenbusch SJ, Fuller GN, Tarrand JJ et al. Fusobacterial brain abscess: a review of five cases and an analysis of possible pathogenesis. JNeurosurg 2003;99:693-700.

- 13. Colen CB, Rayes M, Rengachary S, Guthikonda M. Outcome of brain abscess by Clostridium perfringens. Neurosurgery 2007;61:E1339.
- 14. Nesky MA, McDougal EC, Peacock Jr JE. Pseudallescheria boydii brain abscess successfully treated with voriconazole and surgical drainage: case report and literature review of central nervous system pseudallescheriasis. Clin Infect Dis 2000;31:673-7.
- 15. Eryilmaz R, Sahin M, Hakan TM, Daldal E. Management of lactational breast abscesses. Breast 2005;14:375-9.
- 16. Leach RD, Eykyn SJ, Phillips I, Corrin B. Anaerobic subareolar breast abscess. Lancet 1979;1:35-7.
- 17. Edmiston CE, Jr., Walker AP, Krepel CJ, Gohr C. The nonpuerperal breast infection: aerobic and anaerobic microbial recovery from acute and chronic disease. J Infect Dis 1990;162:695-9.
- 18. Sousa EL, Gomes BP, Jacinto RC, Zaia AA, Ferraz CC. Microbiological profile and antimicrobial susceptibility pattern of infected root canals associated with periapical abscesses. Eur J Clin Microbiol Infect Dis 2013;32:573-80.
- 19. Siqueira JF, Jr., Rocas IN. Microbiology and treatment of acute apical abscesses. Clin Microbiol Rev 2013;26:255-73.
- 20. Tek M, Metin M, Sener I, Bereket C, Tokac M, Kazancioglu HO et al. The predominant bacteria isolated from radicular cysts. Head Face Med 2013;9:25.
- 21. Loesche WJ. Bacterial mediators in periodontal disease. Clin Infect Dis 1993;16 Suppl 4:S203-S10.
- 22. Hsu YL, Lin HC, Yen TY, Hsieh TH, Wei HM, Hwang KP. Pyogenic liver abscess among children in a medical center in Central Taiwan. Journal of microbiology, immunology, and infection = Wei mian yu gan ran za zhi 2015;48:302-5.
- 23. Pineiro-Carrero VM, Andres JM. Morbidity and mortality in children with pyogenic liver abscess. Am J Dis Child 1989;143:1424-7.
- 24. Moore-Gillon JC, Eykyn SJ, Phillips I. Microbiology of pyogenic liver abscess. Br Med J (Clin ResEd) 1981;283:819-21.
- 25. Dorvilus P, Edoo-Sowah R. Streptococcus milleri: a cause of pyogenic liver abscess. JNatlMedAssoc 2001;93:276-7.
- 26. Cheng AC, Currie BJ. Melioidosis: epidemiology, pathophysiology, and management. ClinMicrobiolRev 2005;18:383-416.

- 27. Bach MC. Nocardia. In: Gorbach SL, Bartlett JG, Blacklow NR, editors. Infectious Diseases. 2nd ed. Philadelphia: WB Saunders Company; 1998. p. 1969-73.
- 28. Finegold SM. Lung abscess. In: Mandell GL, Bennett JE, Dolin R, editors. Mandell Douglas and Bennett's Principles and Practice of Infectious Diseases. 5th ed. Philadelphia: Churchill Livingstone; 2000. p. 751-5.
- 29. Kumar S, Bandyopadhyay MK, Bhattacharyya K, Ghosh T, Bandyopadhyay M, Ghosh RR. A Rare Case of Pancreatic Abscess due to Candida Tropicalis. JGlobInfectDis 2011;3:396-8.
- 30. Arditi M, Yogev R. Perirectal abscess in infants and children: report of 52 cases and review of literature. PediatrInfectDis J 1990;9:411-5.
- 31. Brook I, Frazier EH. The aerobic and anaerobic bacteriology of perirectal abscesses. JClinMicrobiol 1997;35:2974-6.
- 32. Brook I, Anderson KD, Controni G, Rodriguez WJ. Aerobic and anaerobic bacteriology of pilonidal cyst abscess in children. AmJDisChild 1980;134:679-80.
- 33. Dubos M, Barraud O, Fedou AL, Fredon F, Laurent F, Brakbi Y et al. Prostatic abscesses and severe sepsis due to methicillin-susceptible Staphylococcus aureus producing Panton-Valentine leukocidin. BMC Infect Dis 2014;14:466.
- 34. Ludwig M, Schroeder-Printzen I, Schiefer HG, Weidner W. Diagnosis and therapeutic management of 18 patients with prostatic abscess. Urology 1999;53:340-5.
- 35. Liu KH, Lee HC, Chuang YC, Tu CA, Chang K, Lee NY et al. Prostatic abscess in southern Taiwan: another invasive infection caused predominantly by Klebsiella pneumoniae. JMicrobiolImmunolInfect 2003;36:31-6.
- 36. Larsen RA, Bozzette S, McCutchan JA, Chiu J, Leal MA, Richman DD. Persistent Cryptococcus neoformans infection of the prostate after successful treatment of meningitis. California Collaborative Treatment Group. Ann Intern Med 1989;111:125-8.
- 37. Bagul NB, Abeysekara AM, Jacob S. Primary psoas abscess due to Streptococcus milleri. AnnClinMicrobiolAntimicrob 2008;7:7.
- 38. van den BM, de Marie S, Kuipers T, Jansz AR, Bravenboer B. Psoas abscess: report of a series and review of the literature. NethJMed 2005;63:413-6.
- 39. Jaik NP, Sajuitha K, Mathew M, Sekar U, Kuruvilla S, Abraham G et al. Renal abscess. JAssocPhysicians India 2006;54:241-3.
- 40. Gardiner RA, Gwynne RA, Roberts SA. Perinephric abscess. BJUInt 2011;107 Suppl 3:20-3.

- 41. Shweikeh F, Saeed K, Bukavina L, Zyck S, Drazin D, Steinmetz MP. An institutional series and contemporary review of bacterial spinal epidural abscess: current status and future directions. Neurosurg Focus 2014;37:E9.
- 42. Darouiche RO, Hamill RJ, Greenberg SB, Weathers SW, Musher DM. Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Medicine (Baltimore) 1992;71:369-85.
- 43. Rubin G, Michowiz SD, Ashkenasi A, Tadmor R, Rappaport ZH. Spinal epidural abscess in the pediatric age group: case report and review of literature. PediatrInfectDisJ 1993;12:1007-11.
- 44. Brook I. Microbiology of subphrenic abscesses in children. PediatrInfectDisJ 1992;11:679-80.
- 45. Farmahan S, Tuopar D, Ameerally PJ. A study to investigate changes in the microbiology and antibiotic sensitivity of head and neck space infections. Surgeon 2014.
- 46. Sowerby LJ, Hussain Z, Husein M. The epidemiology, antibiotic resistance and post-discharge course of peritonsillar abscesses in London, Ontario. J Otolaryngol Head Neck Surg 2013;42:5.
- 47. Adamidou F, Anagnostis P, Karras S, Kita M. Neck abscess associated with a piriform fossa sinus tract in an adult. BMJ Case Rep 2013;2013.
- 48. Biron VL, Kurien G, Dziegielewski P, Barber B, Seikaly H. Surgical vs ultrasound-guided drainage of deep neck space abscesses: a randomized controlled trial: surgical vs ultrasound drainage. J Otolaryngol Head Neck Surg 2013;42:18.
- 49. Taguchi M, Nishikawa S, Matsuoka H, Narita R, Abe S, Fukuda K et al. Pancreatic abscess caused by Corynebacterium coyleae mimicking malignant neoplasm. Pancreas 2006;33:425-9.
- 50. Dobinsky S, Noesselt T, Rucker A, Maerker J, Mack D. Three cases of Arcanobacterium haemolyticum associated with abscess formation and cellulitis. EurJClinMicrobiolInfectDis 1999;18:804-6.
- 51. Aligeti VR, Brewer SC, Khouzam RN, Lewis JB, Jr. Primary gluteal abscess due to Salmonella typhimurium: a case report and review of the literature. AmJMedSci 2007;333:128-30.
- 52. Al Tawfiq JA, Ghandour J. Cryptococcus neoformans abscess and osteomyelitis in an immunocompetent patient with tuberculous lymphadenitis. Infection 2007;35:377-82.
- 53. Eiring P, Wagner D. Peptostreptococcus ivorii-associated skin abscess in a HIV-infected patient. Anaerobe 1999;5:1-3.

- 54. Hoefele J, Kroener C, Berweck S, Peraud A, Grabein B, Wintergerst U et al. Haemophilus paraphrophilus, a rare cause of intracerebral abscess in children. EurJPediatr 2008;167:629-32.
- 55. Tsai SH, Peng YJ, Wang NC. Pyomyositis with hepatic and perinephric abscesses caused by Candida albicans in a diabetic nephropathy patient. AmJMedSci 2006;331:292-4.
- 56. Arya M, Arya PK. Pancreatic abscess caused by s. typhi. Indian JMedMicrobiol 2001;19:18-9.
- 57. Garner JP, MacDonald M, Kumar PK. Abdominal actinomycosis. Int JSurg 2007;5:441-8.
- 58. Altaie SS, Kohout-Dutz E. Actinomycosis masquerading as incarcerated incisional hernia. Clin Microbiol Newslett 1993;15:100-4.
- 59. Sawyer MD, Dunn DL. Antimicrobial therapy of intra-abdominal sepsis. InfectDisClinNorth Am 1992;6:545-70.
- 60. European Parliament. UK Standards for Microbiology Investigations (SMIs) use the term "CE marked leak proof container" to describe containers bearing the CE marking used for the collection and transport of clinical specimens. The requirements for specimen containers are given in the EU in vitro Diagnostic Medical Devices Directive (98/79/EC Annex 1 B 2.1) which states: "The design must allow easy handling and, where necessary, reduce as far as possible contamination of, and leakage from, the device during use and, in the case of specimen receptacles, the risk of contamination of the specimen. The manufacturing processes must be appropriate for these purposes". 1998.
- 61. Official Journal of the European Communities. Directive 98/79/EC of the European Parliament and of the Council of 27 October 1998 on *in vitro* diagnostic medical devices 1998. 1-37.
- 62. Clark AE, Kaleta EJ, Arora A, Wolk DM. Matrix-assisted laser desorption ionization-time of flight mass spectrometry: a fundamental shift in the routine practice of clinical microbiology. ClinMicrobiolRev 2013;26:547-603.
- 63. Espy MJ, Uhl JR, Sloan LM, Buckwalter SP, Jones MF, Vetter EA et al. Real-time PCR in clinical microbiology: applications for routine laboratory testing. ClinMicrobiolRev 2006;19:165-256.
- 64. Health and Safety Executive. Safe use of pneumatic air tube transport systems for pathology specimens. 2009.
- 65. Department for transport. Transport of Infectious Substances, 2011 Revision 5. 2011.

- 66. World Health Organization. Guidance on regulations for the Transport of Infectious Substances 2013-2014. 2012.
- 67. Home Office. Anti-terrorism, Crime and Security Act. 2001.
- 68. Advisory Committee on Dangerous Pathogens. The Approved List of Biological Agents. Executive HaS 2013. 1-32.
- 69. Advisory Committee on Dangerous Pathogens. Infections at work: Controlling the risks. Her Majesty's Stationery Office 2003.
- 70. Advisory Committee on Dangerous Pathogens. Biological agents: Managing the risks in laboratories and healthcare premises. Health and Safety Executive 2005.
- 71. Advisory Committee on Dangerous Pathogens. Biological Agents: Managing the Risks in Laboratories and Healthcare Premises. Appendix 1.2 Transport of Infectious Substances Revision. Health and Safety Executive 2008.
- 72. Centers for Disease Control and Prevention. Guidelines for Safe Work Practices in Human and Animal Medical Diagnostic Laboratories. MMWR Surveill Summ 2012;61:1-102.
- 73. Health and Safety Executive. Control of Substances Hazardous to Health Regulations. The Control of Substances Hazardous to Health Regulations 2002. 5th ed.: HSE Books; 2002.
- 74. Health and Safety Executive. Five Steps to Risk Assessment: A Step by Step Guide to a Safer and Healthier Workplace. HSE Books. 2002.
- 75. Health and Safety Executive. A Guide to Risk Assessment Requirements: Common Provisions in Health and Safety Law. HSE Books. 2002.
- 76. Health Services Advisory Committee. Safe Working and the Prevention of Infection in Clinical Laboratories and Similar Facilities. HSE Books 2003.
- 77. British Standards Institution (BSI). BS EN12469 Biotechnology performance criteria for microbiological safety cabinets 2000.
- 78. British Standards Institution (BSI). BS 5726:2005 Microbiological safety cabinets. Information to be supplied by the purchaser and to the vendor and to the installer, and siting and use of cabinets. Recommendations and guidance. 2005. 1-14.
- 79. Reddy S, Manuel R, Sheridan E, Sadler G, Patel S, Riley P. Brucellosis in the UK: a risk to laboratory workers? Recommendations for prevention and management of laboratory exposure. JClinPathol 2010;63:90-2.
- 80. Allen BW. Survival of tubercle bacilli in heat-fixed sputum smears. J Clin Pathol 1981;34:719-22.

- 81. Baron EJ, Miller JM, Weinstein MP, Richter SS, Gilligan PH, Thomson RB, Jr. et al. A Guide to Utilization of the Microbiology Laboratory for Diagnosis of Infectious Diseases: 2013 Recommendations by the Infectious Diseases Society of America (IDSA) and the American Society for Microbiology (ASM). ClinInfectDis 2013;57:e22-e121.
- 82. Murray Patrick R EJB, James Jorgensen, Michael Pfaller, Marie Louise Landry Manual of Clinical Microbiology 9th edition 2007:293.
- 83. Rishmawi N, Ghneim R, Kattan R, Ghneim R, Zoughbi M, Abu-Diab A et al. Survival of fastidious and nonfastidious aerobic bacteria in three bacterial transport swab systems. JClinMicrobiol 2007;45:1278-83.
- 84. Barber S, Lawson PJ, Grove DI. Evaluation of bacteriological transport swabs. Pathology 1998;30:179-82.
- 85. Van Horn KG, Audette CD, Sebeck D, Tucker KA. Comparison of the Copan ESwab system with two Amies agar swab transport systems for maintenance of microorganism viability. JClinMicrobiol 2008;46:1655-8.
- 86. Nys S, Vijgen S, Magerman K, Cartuyvels R. Comparison of Copan eSwab with the Copan Venturi Transystem for the quantitative survival of *Escherichia coli, Streptococcus agalactiae* and *Candida albicans*. EurJClinMicrobiolInfectDis 2010;29:453-6.
- 87. Tano E, Melhus A. Evaluation of three swab transport systems for the maintenance of clinically important bacteria in simulated mono- and polymicrobial samples. APMIS 2011;119:198-203.
- 88. Miller JM, Holmes HT. Specimen collection, transport, and storage. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Yolken RH, editors. 7th ed.: American Society for Microbiology; 1999. p. 33-63.
- 89. Holden J. Collection and transport of clinical specimens for anaerobic culture. In: Isenberg HD, editor. Clinical Microbiology Procedures HandbookVol 1. Washington D.C.: American Society for Microbiology; 1992. p. 2..1-7.
- 90. Public Health England. Laboratory Reporting to Public Health England: A Guide for Diagnostic Laboratories 2013. 1-37.
- 91. Department of Health. Health Protection Legislation (England) Guidance. 1-112. 2010.
- 92. Scottish Government. Public Health (Scotland) Act. 2008.
- 93. Scottish Government. Public Health etc. (Scotland) Act 2008. Implementation of Part 2: Notifiable Diseases, Organisms and Health Risk States. 2009.

- 94. The Welsh Assembly Government. Health Protection Legislation (Wales) Guidance. 2010.
- 95. Home Office. Public Health Act (Northern Ireland) 1967 Chapter 36. 1967.
- 96. Song Y, Liu C, Finegold SM. Development of a flow chart for identification of gram-positive anaerobic cocci in the clinical laboratory. JClinMicrobiol 2007;45:512-6.