

Chapter 10

Infectious Diseases: The Role of the Forensic Physician

Felicity Nicholson

Introduction

Infections have plagued doctors for centuries, both in the diagnosis of the specific diseases, and the identification and subsequent management of the causative agents. There is a constant need for information as new organisms emerge, existing ones develop resistance to current drugs or vaccines and there are changes in epidemiology and prevalence. In the twenty-first century, obtaining this information has never been more important. Population migration, and the relatively low cost of flying, means that unfamiliar infectious diseases may be brought into industrialised countries. An example of this was an outbreak of severe acute respiratory syndrome (SARS), which was first recognised in 2003. Despite modern technology and a huge input of money, it took months for the agent to be identified, a diagnostic test to be produced, and a strategy for disease reporting and isolation to be established.

A further example of how population migration can result in the spreading of disease was with the appearance of swine influenza A (H1N1). In March 2009, the first case was reported in Mexico; by June 2009, over 22,000 cases had been reported in 70 countries, thus fulfilling the criteria for a pandemic. Challenges ensued with decisions about treatment and then the distribution and administration of specific vaccine.

There is no doubt that other new and fascinating diseases will continue to emerge. For the forensic physician dealing with infections presents two main problems. First, managing detainees or police personnel who have contracted a disease and may be infectious or unwell, and second, handling complainants of assault,

F. Nicholson (✉)
Consultant in Infectious Diseases and Forensic Physician, London, UK
e-mail: felicitynicholson2@googlemail.com

including police officers, who have potentially been exposed to an infectious disease. The latter can be distressing for those involved, compounded in part from an inconsistency of management guidelines, if indeed they exist.

With the advent of human rights legislation, increasing pressure is being placed on doctors with regard to consent and confidentiality of the detainee. Therefore, it is prudent to pre-empt such situations before the consultation begins by obtaining either written or verbal consent from the detainee to allow certain pieces of information to be disclosed. If they do not agree, then the doctor must decide whether withholding relevant details will endanger the lives or health of those working within custody or others with whom they may have had close contact (whether or not deliberate). Issues of consent and confidentiality are discussed in detail in Chap. 2.

Adopting a universal approach with all detainees will decrease the risk to staff of acquiring such diseases and will help to stop unnecessary overreaction and unjustified disclosure of sensitive information. For victims of violent or sexual assault, a more open-minded approach is needed (see also Chap. 3). If the assailant is known, then it may be possible to make an informed assessment of the risk of certain diseases by ascertaining their lifestyle. If, however, the assailant is unknown, then it is wise to assume the worst. This chapter aims to highlight the most common infections encountered by the forensic physician. It aims to dispel “urban myths” and provide a sensible approach for achieving effective management.

Universal Precautions

The risk of exposure to infections, in particular to blood-borne viruses (BBVs), can be minimised by adopting measures that are considered good practice in the UK, the USA and Australia [1–3].

Forensic physicians or other healthcare professionals should wash their hands before and after contact with each detainee or victim. Police officers should be encouraged to wash their hands after exposure to body fluids or excreta. All staff should wear gloves when exposure to body fluids, mucous membranes or non-intact skin is likely. Gloves should also be worn when cleaning up body fluids or handling clinical waste including contaminated laundry. Single-use gloves should only be used and must conform to the requirements of European Standard 455 or equivalent [1–3]. A synthetic alternative conforming to the same standards should also be available for those allergic to latex.

All staff should cover any fresh wounds (less than 24 h old), open skin lesions or breaks in exposed skin with a waterproof dressing. Gloves cannot prevent percutaneous injury, but may reduce the chance of acquiring a blood-borne viral infection by limiting the volume of blood inoculated. Gloves should only be worn when

taking blood, providing this does not reduce manual dexterity and therefore increase the risk of accidental percutaneous injury.

Ideally, a designated person should be allocated to ensure the clinical room is kept clean and sharps containers and clinical waste bags should be removed on a regular basis. Clinical waste must be disposed of in “hazard bags” and should never be over-filled. After use they should be double-bagged and sealed with “hazard tape”. The bags should be placed in a designated waste disposal (preferably outside the building) and removed by a professional company.

When cells are contaminated with body fluids, a professional cleaning company should be called to attend as soon as possible. Until such time, the cell should be deemed “out of action”.

Sharps Awareness

There is a legal requirement in the UK under the Environmental Protection Act (1990) and the Control of Substances Hazardous to Health Regulations 1994 (COSHH) to dispose of sharps in an approved container. In the USA, the Division of Healthcare Quality Promotion (DHQP) on the Centers for Disease Control and Prevention (CDC) website provides similar guidance. In custody where sharps containers are transported off site, they must be of an approved type. In the UK, such a requirement is contained within the Carriage of Dangerous Goods (Classification, Packaging and Labelling) and Use of Transportable Pressure Receptacles Regulations 1996. These measures help to minimise the risk of accidental injury. Further precautions include wearing gloves when handling sharps and never bending, breaking or re-sheathing needles before disposal. Sharps bins should never be over-filled, left on the floor or placed above the eye level of the smallest member of staff.

Contaminated Bedding

Any bedding that is visibly stained with body fluids should be handled with gloves. There are only three acceptable ways of dealing with contaminated bedding:

1. Laundering with a detergent at a minimum temperature of 71°C (160°F) or at a lower temperature (22–50°C) with water containing detergent and 50–150 ppm of chlorine bleach.
2. Dry cleaning at elevated temperatures/dry cleaning cold followed by steam pressing.
3. Incineration.

It is not considered acceptable practice to share bedding between detainees.

Other Measures

It is not necessary for staff to wear masks or protective eyewear in the custodial setting as the risk of infection is low. However, single use eyewash should be available in the clinical room or contained in other first aid kits located within the police station in case of accidental exposure. Contact lenses should be removed prior to eye washing.

Formulation of Guidelines

An example of good practice is contained within the UK Health Department's 1998 document [1] which states "that it is the responsibility of Health Authorities, Health Boards and NHS Trusts to create their own local guidelines to prevent the spread of BBVs in the health care setting". Such guidelines may not exist in other work places. If this is the case, then they should be formulated as soon as possible. Forensic physicians working for the Royal Military Police in the UK can refer to the "Good Practice Guidelines" [4]. It is also prudent to pre-arrange a system of referral with the nearest hospital that has an Accident and Emergency Department, a Genito-Urinary Department and access to a Specialist. The latter may be a consultant in Virology, Microbiology, Infectious Diseases or Genito-Urinary Medicine. Similar guidance in the USA can be found in "The Guideline for Infection Control in Health Care Personnel" [5].

Most exposures to staff usually result from a failure to follow accepted practice; however, accidents can happen no matter how much care is taken. All forensic physicians and other healthcare professionals working in custody should understand what constitutes a risk. This involves taking a detailed history of the incident, including the type of exposure, the body fluids involved and when it occurred.

This information could help to allay unnecessary anxiety from the outset and will ensure that the recipient of the exposure is referred, if appropriate, to the designated hospital at the earliest opportunity. Knowledge of precise treatment protocols is not required, but it is helpful to be able to explain to the recipient what to expect. For example, he or she will be asked to provide a voluntary baseline blood sample for storage and a number of follow-up samples for testing depending on the nature of the exposure. This is especially relevant for hepatitis B (HBV), hepatitis C (HCV) and human immunodeficiency virus (HIV). Most usually, this will be complete by 12 weeks, but on rare occasions testing may be extended to 6 months after the incident.

Sexual assault victims should ideally be referred to specialist centres, if available. A police station should only be used as a last resort because the environment is often hostile and there is no ready access to the necessary treatment and ongoing management (see Chap. 3).

Routes of Transmission

Organisms may utilise more than one route. For ease of understanding, the infections discussed in this chapter are classified according to their primary route, i.e. transmission through blood and body fluids; through contact with lesions or organisms; through the respiratory route; and through the faecal-oral route.

Transmission Through Blood and Body Fluids

The BBVs which present most cross-infection hazard to staff or victims are those associated with persistent viral replication and viraemia. These include hepatitis B (HBV), hepatitis C (HCV), hepatitis D (HDV) and human immunodeficiency virus (HIV).

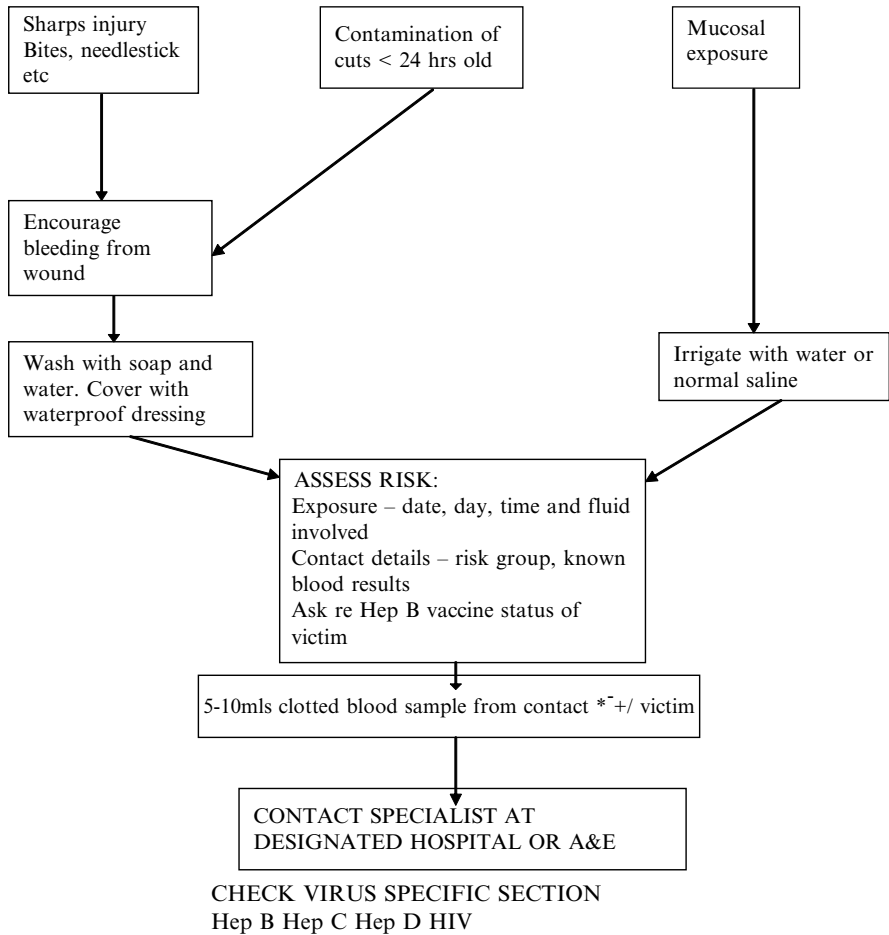
In general, risks of transmission of BBVs arise from the possible exposure to blood or other body fluids. The degree of risk varies with the virus concerned and will be discussed under the relevant sections. Figure 10.1 illustrates the immediate management following a percutaneous injury, mucocutaneous exposure, or exposure through contamination of fresh cuts or breaks in the skin.

Hepatitis B

Epidemiology and Prevalence

HBV is endemic throughout the world with populations showing a varying degree of prevalence. Approximately 2,000 million people have been infected with HBV, with more than 350 million having chronic infection. Worldwide HBV kills about 1 million people each year. With the development of a safe and effective vaccine in 1982, the World Health Organisation (WHO) recommended that HBV vaccine should be incorporated into national immunisation programmes by 1995 in those countries with a chronic infection rate of 8% or higher and into all countries by 1997. Although 135 countries had achieved this goal by the end of 2001, the poorest countries – often with the highest prevalence – have been unable to afford it. In particular, these include China, the Indian Sub-continent and Sub-Saharan Africa.

People in the early stages of infection or with chronic carrier status (defined by persistence of hepatitis B surface antigen [HBsAg] beyond 6 months) can transmit infection. In the United Kingdom, the overall prevalence of chronic HBV is around 0.2–0.3% [6, 7]. A detailed breakdown is shown in Table 10.1.



*In the UK written consent from the contact must be sent with the sample countersigned by the healthcare practitioner and an independent police officer should also sign.

Fig. 10.1 Immediate management following occupational exposure to blood-borne viruses (BBVs)

Table 10.1 Prevalence of chronic hepatitis B (HBV)

Blood-donating population – <1%
Intravenous drug users – 10–15%
Homosexual/bisexuals – 10–15%
Institutionalised patients – no data available
People from high-risk endemic areas, e.g. China and the Far East, up to 30% of the population are carriers and 75% have evidence of past infection. In Africa, 5–10% are carriers

Table 10.2 Significance of markers

Name	Infectivity	Immunity	Risk following needle stick (%)
HBsAg	Yes	No	Only marker = 10–20
HBeAg	Yes	No	With HBsAg = 30–40
HBeA	Yes	Yes	With HBsAg = <10
HBCA	No	Yes	0
HBSA	No	Yes	0

HBsAg Hepatitis B surface antigen; *HbeAg* hepatitis B e antigen; *HBeA* hepatitis B e antibody; *HBCA* hepatitis B core antibody; *HBSA* hepatitis B surface antibody

Symptoms and Complications

The incubation period is approximately 6 weeks to 6 months. As the name suggests, the virus primarily affects the liver. Typical symptoms include malaise, anorexia, nausea, mild fever, and abdominal discomfort and may last from 2 days to 3 weeks before the insidious onset of jaundice. Joint pain and skin rashes may also occur due to immune complex formation. Infections in the newborn are usually asymptomatic.

The majority of patients with acute HBV make a full recovery and develop immunity. Following acute infection, about 1 in 300 patients develop liver failure, which may result in death.

Chronic infection develops in around 90% of neonates, about 50% of children and between 5 and 10% of adults. Neonates and children are usually asymptomatic. Adults may have only mild symptoms or may also be asymptomatic. Approximately 15–25% of chronically infected individuals (depending on age of acquisition) will develop cirrhosis over a number of years. This may also result in liver failure or other serious complications including hepatocellular carcinoma, though the latter is rare. The overall mortality rate of HBV is estimated at less than 5%.

Period of Infectivity

A person is deemed infectious if HBsAg is detected in the blood. In the acute phase of the illness, this can be as long as 6 months. By definition, if HBsAg persists for after this time then the person is deemed a carrier. Carriers are usually infectious for life. The degree of infectivity depends on the stage of disease and the markers present in Table 10.2.

Routes of Transmission

The major routes include: parenteral (e.g. needlestick injuries, bites, unscreened blood transfusions, tattooing, acupuncture and dental procedures, where equipment is

inadequately sterilised); mucous membrane exposure (including mouth, eyes and genital mucous membranes) and contamination of broken skin (especially when <24 h old).

At-Risk Groups

HBV is an occupational hazard for anyone who may come into contact with blood or bloodstained body fluids through the routes described above. Saliva alone may transmit HBV. The saliva of some people infected with HBV has been shown to contain HBV-DNA concentrations 1/1,000–1/10,000 of that found in their serum [8]. This is especially relevant for penetrating bite wounds. Infection following exposure to other body fluids, e.g. bile, urine, faeces and CSF, has never been demonstrated unless the fluids are contaminated with blood.

Intravenous drug users who share needles or other equipment are also at risk. HBV can also be transmitted through unprotected sexual contact, whether homosexual or heterosexual. The risk is increased if blood is involved. Victims of sexual assault should be included in this category.

Evidence has shown that the virus may also be spread among members of a family through close household contact. This is thought to be through kissing, sharing toothbrushes, razors, bath towels, etc. [9–11]. This route of transmission probably applies to institutionalised patients, but there is no available data.

Studies of prisoners in western countries have shown a higher prevalence of antibodies to HBV and other BBVs than the general population [12–14]; the most commonly reported risk factor being intravenous drug use. However, the real frequency of transmission of BBVs in British prisons is unknown due to the difficulty in compiling reliable data.

HBV can be transmitted vertically from mother to baby during the perinatal period. Around 80% of babies born to mothers who have either acute or chronic HBV become infected and most will develop chronic HBV. This has been limited by the administration of HBV vaccine to the neonate. In industrialised countries all antenatal mothers are screened for HBV. Vaccine is given to the neonate ideally within the first 12 h of birth and at least two further doses are given at designated intervals. The WHO recommends this as a matter of course for all women in countries where prevalence is high. However, the practicalities of administering vaccine that has to be stored at the correct temperature and limited access to medical care mean that there is a significant failure of vaccine uptake and response.

Disease Prevention

In industrialised countries, HBV vaccination is recommended for those deemed at risk of acquiring the disease

1. Through occupational exposure
2. Homosexual/bisexual men
3. Intravenous drug users

4. Sexual partners of people with acute or chronic HBV
5. Family members of people with acute or chronic HBV
6. Newborn babies whose mothers are infected with HBV. If the mother is HBeAg positive, then HBV-specific immunoglobulin (HBSIG) should be given at the same time as the first dose of vaccine
7. Institutionalised patients and prisoners

Ideally, HBV vaccine should be administered prior to exposure. The routine schedule (RS) consists of three doses of vaccine given at 0, 1 and 6 months. Antibody levels should be checked at 4–16 weeks after the last dose. If titres are greater than 10 miU/ml, then an adequate response has been achieved. In the UK, this is considered to provide protection for 5–10 years when one further dose would be given. In the USA, if an initial adequate response has been achieved, then no further doses of vaccine are considered necessary.

Vaccine administration after exposure varies according to the timing of the incident, the degree of risk involved, and whether the individual has already been partly or fully vaccinated. An accelerated schedule (AS) when the third dose is given 2 months after the first dose with a booster a year later is used to prevent perinatal transmission. Where risks are greatest then it may be necessary to use a rapid schedule (RDS). The doses are given at 0, 7, 21–28 days after presentation, again with a booster dose at 6–12 months. This schedule is currently only licenced with Engerix B.

HBIG may also be used either alone or in conjunction with vaccine. The exact dose given is age-dependent, but must be administered by deep intra-muscular injection in a different site from the vaccine. In an adult, this is usually into the gluteus muscle.

HBIG is given in conjunction with the first dose of vaccine to individuals deemed at high risk of acquiring disease and the incident occurred within 72 h of presentation. It is also used for neonates born to mothers who are HBeAg-positive.

Between 5 and 10% of adults fail to respond to the routine schedule of vaccine. A further full course of vaccine should be tried before deeming them “non-responders”. Such individuals involved in a high-risk exposure should be given two doses of HBIG administered a month apart. Ideally, the first dose should be given within 48 h after exposure and no longer than 2 weeks after exposure.

Other measures include minimising the risk of exposure by adopting the safe working practices outlined in section “Universal Precautions”. Any potential exposures should be dealt with as soon as possible. In industrialised countries, blood, blood products and organs are routinely screened for HBV.

Intravenous drug users should be encouraged to be vaccinated and to avoid sharing needles or any other drug paraphernalia (see “Management in Custody” in section “Other Bacteria Associated with Abscess Formation in Injecting Drug Users”).

Management in Custody

For staff or victims in contact with disease, it is wise to have a procedure in place for immediate management and risk evaluation. An example is shown in Fig. 10.1.

While forensic physicians are not expected to administer treatment, it is often helpful to inform persons concerned what to expect. Tables 10.3 and 10.4 outline treatment protocols as used in the United Kingdom.

Detainees with disease can usually be managed in custody. If the detainee is bleeding, then the cell should be deemed out of action after they have left until it can be professionally cleaned. Contaminated bedding should be dealt with as described in section “Contaminated Bedding”. If the detainee has chronic HBV and is on an antiviral agent (e.g. Lamivudine), then the treatment course should be continued, if possible.

Hepatitis C

Epidemiology and Prevalence

HCV is endemic in most parts of the world. Approximately 3% (200 million) of the world’s population are infected with HCV [15]. For many countries, no reliable prevalence data exist.

Seroprevalence studies carried out among blood donors have shown that the highest prevalence exists in Egypt (17–26%). This has been ascribed to contaminated needles used in the treatment of schistosomiasis carried out between the 1950s and the 1980s [16].

Intermediate prevalence (1–5%) exists in Eastern Europe, the Mediterranean, the Middle East, the Indian Sub-continent and parts of Africa and Asia. In Western Europe, most of Central America, Australia and limited regions in Africa including South Africa, the prevalence is low (0.2–0.5%). Previously, America was included in the low prevalence group, but a report published in 2003 [17] indicated that almost 4 million Americans, i.e. 1.8% of the population, have antibody to HCV, representing either ongoing or previous infection. It also states that HCV accounts for approximately 15% of acute viral hepatitis in America.

The lowest prevalence (0.01–0.1%) has been found in the UK and Scandinavia. However, within any country, there are certain groups that have a higher chance of carrying HCV. In the UK, these are given in Table 10.5.

Symptoms and Complications

After an incubation period of 6–8 weeks, the acute phase of the disease lasts approximately 2–3 years. Unlike hepatitis A (HAV) or HBV, the patient is usually asymptomatic; therefore, the disease is often missed unless the individual has reported a specific exposure and is being monitored. Other cases are found by chance, when raised liver enzymes are found on a routine blood test.

A “silent phase” follows the acute phase when the virus lies dormant and the liver enzymes are usually normal. This period lasts approximately 10–15 years.

Table 10.3 Management following high-risk exposure

Vaccination status	Hepatitis B-specific immunoglobulin (HBSIG)	Hepatitis B vaccine	Follow up	Notes
Not vaccinated	Yes if <3 days after exposure No if >3 days	Yes	AS via GP	
Vaccinated: non-responder	Yes if within 3 days	No	Rapid schedule (RDS) via GP Repeat HBSIG at 1 month	Advise GP of timing Consider trying newer vaccines at later stage
Course completed. Levels >10 miU/ml	No	Yes if primary course >3 years ago	No	
Course completed within 3 years. Levels not checked	No	Yes	GP to check results of baseline blood test	If baseline antibodies <10 miU/ml advise RDS
If >3 years see below				
Incomplete course (one or two doses)	Yes if within 3 days No if >3 days	Yes Yes	GP to check results of baseline blood test	>10 miU/ml – RS <10 miU/ml – RDS

Contact in high-risk group or HBsAg positive and person has had a high-risk exposure

Table 10.4 Management following low-risk exposure

Vaccination status	HBSIG	Vaccine	Follow up	Notes
Not vaccinated	No	Yes	RS via GP	
Vaccinated non-responder	No	No		Consider using newer vaccines
Course completed	No	Yes if not checked or >3 years since 1 ^o course		
Incomplete course	No	Yes	GP to check results of baseline test	<10 miU/ml complete RS

Contact is in low-risk group or known to be HBsAg negative and person has had a low-risk exposure

Table 10.5 Prevalence of hepatitis C (HCV)

General blood-donating population	0.06%
Organ donors	0.72%
Haemophiliacs	100% ^a
Intravenous drug users	28–57%
Homosexual/bisexuals	<5%

^aStatistics apply to all who received blood products before the mid-1980s

Reactivation may then occur. Subsequent viral replication damages the hepatocytes, and liver enzymes rise to moderate or high levels.

Eighty percent of individuals who are HCV antibody-positive are infectious, regardless of the levels of their liver enzymes. Approximately 80% of people develop chronic infection, one fifth of whom progress to cirrhosis. There is a much stronger association with hepatocellular carcinoma than with HBV. An estimated 1.25–2.5% of patients with HCV-related cirrhosis develop liver cancer [18]. Less than 2% of chronic cases resolve spontaneously.

Routes of Transmission

Approximately 75% of cases are parenteral (e.g. needlestick, etc.) [19]. Transmission through the sexual route is not common and only appears to be significant if there is repeated exposure with one or more people infected with HCV. Mother-to-baby transmission is considered to be uncommon, but has been reported [20]. Theoretically, household spread is also possible through sharing contaminated toothbrushes or razors.

Since the disease is often silent, there is a need to raise awareness among the general population on how to avoid infection and to encourage high-risk groups to be tested. Healthcare professionals should also be educated to avoid occupationally acquired infection. An example of good practice is contained within the document ‘Hepatitis C strategy for England’, issued by the Department of Health in 2002 [18].

Risks from Exposure from HCV RNA-Positive Person

Blood or bloodstained body fluids need to be involved for a risk to occur. Saliva alone is not deemed to be a risk. The risk from a single needlestick incident is 1.8% (range 0–7%). Contact through a contaminated cut is estimated at 1%. For penetrating bite injuries, there is no data, but it is only considered a risk if blood is involved. Blood or bloodstained body fluids have to be involved in transmission through mucous membrane exposure. This may account for the lower than expected prevalence among the gay population.

Management in Custody

Staff/Victims in Contact with Disease

Follow the immediate management flow chart, making sure all available information is obtained. Inform the designated hospital and/or specialist as soon as possible. If the contact is known and is thought to be immunocompromised, and he or she has consented to provide a blood sample, it is important to tell the specialist, as their antibody tests may be spuriously negative. In this instance, a different test should be used (polymerase chain reaction [PCR] which detects viral RNA).

The staff member/victim will be asked to provide a baseline sample of blood with further samples at 4–6 weeks and again at 12 weeks. If tests are negative at 12 weeks, but the risk was deemed high, then follow-up may continue for up to 24 weeks. If any of the follow-up samples are positive, then the original baseline sample will be tested to ascertain whether the infection was acquired through the particular exposure.

Chronic HCV may be treatable with a combination of pegylated or non-pegylated interferons and ribavirin. Treatment success depends upon many factors including disease stage and viral genotype [21, 22]. Treatment in the early stages of infection is nearly always successful regardless of the genotype, so early detection is important.

Detainees with disease: Unless they are severely ill, detainees can be managed in custody. Special precautions are only required if they are bleeding. Custody staff should wear gloves if contact with blood is likely. Contaminated bedding should be handled appropriately and the cell cleaned professionally after use.

Hepatitis D (Delta Agent)

This defective transmissible virus discovered in 1977 requires HBV for its own replication. It has a worldwide distribution in association with HBV with approximately 15 million people infected. The prevalence of HDV is higher in southern Italy, the Middle East, and parts of Africa and South America occurring in greater than 20% of asymptomatic HBV carriers and greater than 60% of those with chronic HBV-related liver disease. Despite the high prevalence of HBV in China and South East Asia, HDV in these countries is rare.

HDV is associated with acute (co-infection) and chronic hepatitis (superinfection) and can exacerbate pre-existing liver damage caused by HBV. The routes of transmission and at-risk groups are the same as for HBV. Staff/victims in contact with a putative exposure and detainees with disease should be managed as for HBV. Interferon-alpha (e.g. Roferon) can be used to treat patients with chronic HBV and HDV [23], though it would not be practical to continue this treatment in the custodial setting.

Human Immunodeficiency Virus

Epidemiology and Prevalence

HIV was first identified in 1983 – 2 years after the first reports were made to the CDC Atlanta, GA, of an increased incidence of two unusual diseases (Kaposi's sarcoma and *Pneumocystis carinii* pneumonia) occurring among the gay population in San Francisco. The scale of the virus gradually emerged over the years, and by the end of 2002, there was an estimated 42 million people throughout the world living with HIV or AIDS. Over 80% of the world total live in Africa and India. A report by UNAIDS and the WHO in 2002 stated that one in five adults in Lesotho, Malawi, Mozambique, Swaziland, Zambia and Zimbabwe has HIV or AIDS. There is also expected to be a sharp rise in cases of HIV in China, Papua New Guinea and other countries in Asia and the Pacific over the next few years.

In the UK by the end of 2002, the cumulative data reported that there were 54,261 individuals with HIV, AIDS (including deaths from AIDS) reported, though this is likely to be an underestimate [24]. By the end of 2006, the HIV prevalence increased to 78,000 with one-third being undiagnosed. The group still considered at greatest risk of acquiring HIV in the United Kingdom is men who have sex with men (MSM) with 5.4% of those aged 15–44 being infected [25].

Among intravenous drug users, the overall estimated prevalence is 1 in 73, but in London the figure is higher at 1 in 20 [25]. In the 1980s, up to 90% of users in Edinburgh and Dundee were reported to be HIV positive, but the majority have now died.

In 2006, 78,000 new diagnoses were made and nearly half of these were in black Africans. Each year, more HIV infections are being acquired heterosexually within the UK [25].

The incidence of mother-to-baby transmission has been estimated at 15% in Europe, and around 45% in Africa. The transmission rates among African women are thought to be much higher due to a combination of more women with end-stage disease with a higher viral load and concomitant placental infection, which renders it more permeable to the virus [26, 27]. The use of anti-retroviral therapy during pregnancy together with the advice to avoid breastfeeding has proved efficacious in reducing both vertical and horizontal transmission among HIV positive women in the western world. For those in third world countries, the reality is stark. Access to

treatment is limited and there is no realistic substitute for breast milk, which provides a valuable source of antibodies to other life-threatening infections. Patients receiving blood transfusions, organs or blood products where screening is not routinely carried out must also be included.

Incubation Period and Phases of Infection

The incubation is estimated at 2 weeks to 6 months after exposure. This is dependent to some extent on the ability of current laboratory tests to detect HIV antibodies or viral antigen. The majority of cases are now diagnosed within 12 weeks. The development of PCR for viral RNA has improved sensitivity.

During the acute phase of the infection, about 50% experience a seroconversion “flu-like” illness. The individual is infectious at this time, as viral antigen (p24) is present in the blood. As antibodies start to form, the viral antigen disappears and the individual enters the latent phase. They are non-infectious and remain well for a variable period of time (7–15 years). Development of the AIDS marks the terminal phase of disease. Viral antigen re-emerges and the individual is once again infectious. The onset of AIDS has been considerably delayed with the use of anti-retroviral treatment.

Routes of Transmission

Parenteral transmission includes needle stick injuries, bites, unscreened blood transfusions, tattooing, acupuncture and dental procedures where equipment is inadequately sterilised. Risk of transmission is increased with deep penetrating injuries with hollow bore needles that are visibly bloodstained, especially when the device has previously been in the source patient’s (contact) artery or vein. Other routes include mucous membrane exposure, (eyes, mouth and genital mucous membranes) and contamination of broken skin.

The higher the viral load in the contact, the greater the risk of transmission. This is more likely at the terminal stage of infection. HIV is transmitted mainly through blood or other body fluids that are visibly bloodstained with the exception of semen, vaginal fluid and breast milk. Saliva alone is most unlikely to transmit infection. Therefore, people who have sustained penetrating bite injuries can be reassured that they are not at risk providing the contact was not bleeding from the mouth at the time.

Risk of Seroconversion

The risk from a single percutaneous exposure from a hollow bore needle is low and a single mucocutaneous exposure is even less likely to result in infection.

The risk from sexual exposure varies, though it appears that there is a greater risk with receptive anal intercourse compared with receptive vaginal intercourse [28].

Body Fluids Containing HIV

High-risk fluids include blood, semen, vaginal fluid and breast milk. There is little or no risk from saliva, urine, vomit or faeces unless they are visibly bloodstained. Other fluids that constitute a theoretical risk include CSF, peritoneal, pleural, synovial or pericardial fluid.

Management in Custody of Staff/Victims in Contact with Disease

Management in custody of staff/victims in contact with disease includes following the immediate management flow chart (Fig. 10.1) and contacting the designated hospital/specialist with details of the exposure. Where possible, obtain a blood sample from the contact. Like HBV and HCV, blood samples taken for HIV testing can only be taken with informed consent in the United Kingdom. There is no need for the forensic physician to go into details about the meaning of the test, but the contact should be encouraged to attend the genito-urinary department (or similar) of the designated hospital to discuss the test results. Should the contact refuse to provide a blood sample, then any information about their lifestyle, ethnic origin, state of health, etc. may be useful for the specialist to decide whether post-exposure prophylaxis (PEP) should be given to the recipient. Where saliva only is involved in a penetrating bite injury, there is every justification to reassure the recipient that there is little risk of acquiring HIV. However, it is still wise to take advice from a specialist.

In the United Kingdom, the current recommended regime for PEP is one Truvada tablet (254 mg tenofovir and 200 mg emtricitabane (FTC)) once a day plus two Kaletra film-coated tablets (200 mg lopinavir and 50 mg of ritonavir) twice a day [29].

It is only given following a significant exposure to a high-risk fluid or any that is visibly bloodstained and the contact is known or is highly likely to be HIV positive. Ideally, treatment should be started within an hour after exposure, and certainly within 72 h. Extenuating circumstances beyond the remit of this chapter may allow for PEP to be given after a longer period following exposure [29]. It is usually given for 4 weeks unless the contact is subsequently identified as HIV negative or the recipient develops tolerance or toxicity occurs. Weekly follow-up of the recipient should take place during treatment to improve adherence, monitor drug toxicity and deal with other concerns.

Other useful information that may influence the decision whether to treat with the standard regime or use alternative drugs include interaction with other medications that the recipient may be taking or if the contact has been on anti-retroviral therapy or if the recipient is pregnant. No data exist as to the efficacy of PEP beyond occupational exposure [30].

PEP is not considered for exposure to low or no risk fluids through any route, nor where the source is unknown, e.g. a discarded needle. Despite the appropriate use and timing of PEP, there have been reports of failure [31, 32].

Management in Custody of Detainees with HIV

Unless they are severely ill, they can be kept in custody. Every effort should be made to continue any treatment they may be receiving. Apply universal precautions when dealing with the detainee and ensure that contaminated cells and/or bedding are managed appropriately.

Transmission Through Contact with Lesions or Organisms

Varicella (Chicken Pox)

Epidemiology and Prevalence

Cases of this highly infectious disease occur throughout the year, but are more frequent in winter and early spring. This seasonal endemicity appears to be blurring with global warming. In the UK, the highest prevalence occurs in the 4–10 years age group. Ninety percent of the population over the age of 40 are immune [33]. A similar prevalence has been reported in other parts of Western Europe and the USA. In South East Asia, Varicella is mainly a disease of adulthood [34]. Therefore, people born in these countries who have moved to the UK are more likely to be susceptible to chicken pox.

There is a strong correlation between a history of chicken pox and serologic immunity (97–99%). Most adults born and living in industrialised countries with an uncertain or negative history of chicken pox are also seropositive (70–90%). In March 1995, a live-attenuated vaccine was licenced for use in the USA and a policy for vaccinating children and susceptible healthcare personnel was introduced. In the summer of 2002 in the UK, GlaxoSmithKline launched a live-attenuated vaccine called Varilrix. In December 2003, the Department of Health, following advice from the Joint Committee on Vaccination and Immunisation (JCVI), recommended that the vaccine be given for non-immune healthcare workers who are likely to have direct contact with individuals with chicken pox. Any healthcare worker with no previous history of chicken pox should be screened for immunity, and if no antibodies are found, they should receive two doses of vaccine 4–8 weeks apart. The vaccine is not currently recommended for children and should not be given during pregnancy.

Incubation Period and Symptoms

Following an incubation period of 10–21 days (this may be shorter in the immunocompromised), there is usually a prodromal “flu-like” illness before the onset of the rash. This coryzal phase is more likely in adults. The lesions typically appear in crops; rapidly progressing from red papules through vesicles to open sores that crust over and separate by 10 days. The distribution of the rash is centripetal, i.e. more over the trunk and face than on the limbs. This is the converse of small pox.

In adults, the disease is often more severe with lesions involving the scalp and mucous membranes of the oropharynx.

Complications

In children, the disease is often mild, unless they are immunocompromised, so are unlikely to experience complications. In adults (defined as 15 years or older), the picture is rather different [35]. Secondary bacterial infection is common, but rarely serious. There is an increased likelihood of permanent scarring. Haemorrhagic chickenpox typically occurs on the second or third day of the rash. Usually, this is limited to bleeding into the skin, but life-threatening melaena, epistaxis or haematuria can occur.

Varicella pneumonia ranges from patchy lung consolidation to overt pneumonitis and occurs in 1 in 400 cases [36]. It can occur in previously healthy individuals (particularly adults), but the risk is increased in those who smoke. Immunocompromised people are at the greatest risk of developing this complication. It runs a fulminating course and is the commonest cause of Varicella-associated death.

Fibrosis and permanent respiratory impairment may occur in those who survive. Any suspicion of lung involvement is an indication for immediate treatment and any detainee or staff member should be sent to hospital. Involvement of the CNS includes a variety of conditions including meningitis, Guillain–Barre and encephalitis. The latter is more common in the immunocompromised and can be fatal.

Period of Infectivity

This is taken as 3 days before the first lesions appear to the end of new vesicle formation and the last vesicle has crusted over. This typically is 5–7 days after onset, but may last up to 14 days.

Routes of Transmission

The primary route is through direct contact with open lesions of chicken pox. However, it is also spread through aerosol or droplets from the respiratory tract. Chicken pox may also be acquired through contact with open lesions of shingles (Varicella zoster), but this is less likely as shingles is less infectious than chicken pox.

At-Risk Groups

Non-immune individuals are at risk of acquiring disease. Approximately 10% of the adult population born in the UK and <5% of adults in the USA fall into this category. Therefore, it is more likely that if chicken pox is encountered in the custodial setting, it will involve people born outside the UK, (in particular South East Asia) or individuals who are immunocompromised and have lost immunity. Non-immune pregnant women are at risk of developing complications.

Pneumonia can occur in up to 10% of pregnant women with chicken pox and the severity appears to be increased in later gestation [37]. They can also transmit infection to the unborn baby [38]. If infection is acquired in the first 20 weeks, there is <3% chance of it leading to congenital Varicella syndrome. Infection in the last trimester can lead to neonatal Varicella unless more than 7 days elapse between onset of maternal rash and delivery when antibodies have time to cross the placenta leading to either mild or inapparent infection in the newborn. In this situation, Varicella immunoglobulin (VZIG) should be administered to the baby as soon as possible after birth [39].

Management in Custody

Staff with chicken pox should stay off work until the end of the infective period (approximately 7–14 days). Those in contact with disease who are known to be non-immune or who have no history of disease should contact the designated occupational health physician.

Detainees with disease should not be kept in custody if at all possible (especially pregnant women). If this is unavoidable, then non-immune or immunocompromised staff should avoid entering the cell or having close contact with the detainee.

Non-immune immunocompromised or pregnant individuals exposed to chicken-pox should seek expert medical advice or the administration of VZIG. Aciclovir (or similar antiviral agent) should be given as soon as possible to immunocompromised people with chicken pox. It should also be considered for anyone over 15 years old as they are more likely to develop complications.

Anyone suspected of severe complications should be sent straight to hospital.

Herpes Zoster (Shingles)

Epidemiology

After chicken pox, the virus lies dormant in the dorsal root or cranial nerve ganglia, but may re-emerge and typically involves one dermatome [40]. The site of involvement depends on the sensory ganglion initially involved. Shingles is more common over the age of 50, except in the immunocompromised when attacks can occur at an earlier age. The latter are also more susceptible to secondary attacks and involvement of more than one dermatome. Bilateral zoster is even rarer, but is not associated with a higher mortality.

In the UK, there is an estimated incidence of 1.2–3.4 per 1,000-person years [41].

Symptoms

There may be a prodromal period of paraesthesia and burning or shooting pains in the involved segment. This is usually followed by the appearance of a band of

vesicles. Rarely, the vesicles fail to appear and only pain is experienced. This is known as *zoster sine herpette*. In individuals who are immunocompromised, disease may be prolonged and dissemination may occur but is rarely fatal.

Shingles in pregnancy is usually mild. The foetus is only affected if viraemia occurs before maternal antibody has had time to cross the placenta.

Complications

The most common complication of shingles is post-herpetic neuralgia occurring in about 10% of cases. It is defined as pain lasting more than 120 days from rash onset [42]. It is more frequent in people over 50 and can lead to depression. It is rare in children including the immunocompromised. Infection of the brain includes encephalitis, involvement of motor neurones leading to ptosis, paralysis of the hand, facial palsy or contralateral hemiparesis. Involvement of the oculomotor division of the trigeminal ganglion can cause serious eye problems including corneal scarring.

Period of Infectivity

Shingles is far less infectious than chicken pox and is only considered to be infectious up to 3 days after lesions appear.

Routes of Transmission

Shingles is only infectious following prolonged contact with lesions. Unlike chickenpox, airborne transmission is not a risk.

At-Risk Groups

Individuals who are immunocompromised may reactivate the dormant virus and develop shingles. People who have not had primary Varicella are at risk of developing chicken pox following prolonged direct contact with shingles.

Despite popular belief, it is untrue that immunocompetent people who have had chicken pox develop shingles when in contact with either chicken pox or shingles. Such occurrences are merely coincidental unless immunity is lowered.

Management in Custody

Staff with shingles should stay off work until the lesions are healed unless they can be covered. Staff who have had chicken pox are immune (including pregnant

women) and are therefore not at risk. If they are non-immune (usually accepted as those without a history of chickenpox), they should avoid prolonged contact with detainees with shingles. Pregnant non-immune women should avoid contact altogether.

Detainees with disease may be kept in custody and any exposed lesions should be covered. It is well documented that prompt treatment attenuates the severity of the disease, reduces the duration of viral shedding, hastens lesion healing, and reduces the severity and duration of pain. It also reduces the likelihood of developing post-herpetic neuralgia [43]. Prompt treatment with Famciclovir (500 mg tds for 7 days, for example) should be initiated if the onset is 3 days or less. It should also be considered after this time if the detainee is over 50. Pregnant detainees with shingles can be reassured that there is minimal risk for both the mother and the unborn child. Expert advice should be sought before initiating treatment for the mother.

Scabies

Epidemiology

This tiny parasitic mite (*Sarcoptes scabiei*) has infested humans for over 2,500 years. Experts estimate that in excess of 300 million cases occur worldwide each year. The female mite burrows into the skin especially around the hands, feet and male genitalia, in about 2.5 min. Eggs are laid and hatch into larvae that travel to the skin surface as newly developed mites.

Symptoms

The mite causes intense itching which is often worse at night and is aggravated by heat and moisture. The irritation spreads outside the original point of infection due to an allergic reaction to mite faeces. This irritation may persist for about 2 weeks after treatment, but can be alleviated by antihistamines.

Crusted scabies is a far more severe form of the disease. Large areas of the body may be involved. The crusts hide thousands of live mites and eggs making them difficult to treat. This so-called Norwegian scabies is more common in the elderly or the immunocompromised, especially those with HIV.

Incubation Period

Following a primary exposure, it takes about 2–6 weeks before the onset of itching. However, further exposures reduce the incubation time to around 1–4 days.

Period of Infectivity

Without treatment, this is assumed to be indefinite. With treatment, the person should be considered infectious until the mites and eggs are destroyed – usually 7–10 days. Crusted scabies is highly infectious.

Management in Custody

Since transmission is through direct skin-to-skin contact with an infected case, gloves should be worn when dealing with individuals suspected of infestation. Usually prolonged contact is needed, unless the person has crusted Scabies where transmission occurs more easily. The risk of transmission is much greater in households where repeated or prolonged contact is likely.

Since mites can survive in bedding of clothing for up to 24 h, gloves should also be worn when handling these items. Bedding should be treated using one of the methods above. Professional cleaning of the cell is only warranted in cases of crusted scabies.

Treatment

The preferred treatment for scabies is either permethrin cream (5%) or aqueous Malathion (0.5%) [44]. Either treatment has to be applied to the whole body and should be left on for at least 8 h in the case of permethrin and 24 h for Malathion before washing off. Lindane is no longer considered the treatment of choice, as there may be complications in pregnancy [45].

Treatment in custody may not be practical, but should be considered when the detainee is thought to have Norwegian scabies.

Head Lice

General Information

Like scabies, head lice occur worldwide and are found in the hair close to the scalp. The eggs or nits cling to the hair and are difficult to remove, but are not harmful. If you see nits then you can be sure that lice are also present. The latter are best seen when the hair is wet. The lice bite the scalp and suck blood causing intense irritation and itching.

Route of Transmission

They can only be passed from direct hair to hair contact.

Management in Custody

It is only necessary to wear gloves when examining the head for whatever reason. The cell does not need to be cleaned after use, since the lice live on or near skin. Bedding may be contaminated with shed skin so should be handled with gloves and laundered or incinerated.

The presence of live lice is an indication for treatment either by physical removal with a comb or the application of an insecticide. The latter may be more practical in custody. 0.5% aqueous Malathion should be applied to dry hair and washed off after 12 h. The hair should then be shampooed as normal.

Crabs or Body Lice

General Information

They are more commonly found in the pubic, axillary, chest and leg hair. However, eyelashes and eyebrows may also be involved. They are associated with people who do not bath or change clothes regularly. The person usually complains of intense itching or irritation.

Routes of Transmission

The main route is from person to person by direct contact, but eggs can stick to fibres so clothing and bedding should be handled with care (see section below).

Management in Custody

Staff should always wear gloves if they are likely to come into contact with any hirsute body part. Clothing or bedding should be handled with gloves and either laundered or incinerated.

Treatment of a detainee in custody is good in theory but probably impractical as the whole body has to be treated.

Fleas

General Information

Fleas lay eggs on floors, carpets and bedding. In the UK, most fleabites come from cats or dogs. The eggs and larvae fleas can survive for months and are reactivated in response to animal or human activity. Since animal fleas jump off humans after biting, most detainees with fleabites will not have fleas, unless they are human fleas.

Management in Custody

Treatment is only necessary if fleas are seen. After use, the cell should be vacuumed and cleaned with a proprietary insecticide. Any bedding should be removed wearing gloves, bagged and either laundered or incinerated.

Bedbugs

General Information

Bedbugs live and lay eggs on walls, floors, furniture and bedding. If you look carefully, faecal tracks may be seen on hard surfaces. If they are present for long enough, then they emit a distinct odour. Bedbugs are rarely found on the person, but may be brought in on clothing or other personal effects.

Symptoms

Bedbugs bite at night and can cause sleep disturbance.

Management in Custody

The detainee does not need to be treated, but the cell should be deemed out of use until it can be vacuumed and professionally cleaned with an insecticide solution. Any bedding or clothing should be handled with gloves and disposed of as appropriate.

Methicillin-Resistant Staphylococcus aureus

Epidemiology

Staphylococcus aureus is commonly carried on the skin or in the nose of healthy people. Approximately 25–30% of the population is colonised with the bacteria, but remain well [46]. From time to time, the bacteria cause minor skin infections that usually do not require antibiotic treatment. However, more serious problems can occur, e.g. infection of surgical wounds, drug injection sites, osteomyelitis, pneumonia or septicaemia. Over the last 50 years, the bacteria have become increasingly resistant to penicillin-based antibiotics [47] and in the last 20 years to an increasing number of alternative antibiotics. These multi-resistant bacteria are known as methicillin-resistant *S. aureus* (MRSA).

MRSA is prevalent worldwide. Like non-resistant staphylococci, it may remain undetected as a reservoir in colonised individuals, but can also produce clinical disease. It is more common among the elderly, debilitated or immunocompromised people or those with open wounds. Clusters of skin infections with MRSA have been reported among injecting drug users since 1981 in America [48, 49] and more recently similar strains have been found in the UK in IDUs in the community [50]. This may have particular relevance for the forensic physician when dealing with injecting drug users' sores. Immunocompetent people rarely get MRSA and should not be considered at risk. Between April 2003 and December 2008, there were 74 recorded cases of a community-acquired MRSA in injecting drug users in England and Wales [51].

Route of Transmission

The bacteria are usually spread via the hands of staff after contact with colonised or infected detainees or devices, items (e.g. bedding, towels, soiled dressings) or environmental surfaces that have been contaminated with MRSA-containing body fluids.

Management in Custody

With either known or suspected cases (consider all abscesses/ulcers of injecting drug users as infectious), standard precautions should be applied. Staff should wear gloves when touching mucous membranes, non-intact skin, blood or other body fluids or any items that could be contaminated. They should also be encouraged to wash hands with an antimicrobial agent whether or not gloves have been worn. After use, gloves should be disposed of in a yellow "hazard" bag and not allowed to touch surfaces. Masks and gowns should only be worn when carrying out procedures that generate aerosols of blood or other body fluids. Since this is an unlikely scenario in the custodial setting, they should not be necessary. Gloves should be worn when handling bedding or clothing and all items should be disposed of in the appropriate manner. Any open wounds should be covered as soon as possible. The cell should be cleaned professionally after use if there is any risk that it has been contaminated.

Other Bacteria Associated with Abscess Formation in Injecting Drug Users

Epidemiology

Over the last decade, there has been an increasing awareness of the bacterial flora colonising injection sites that may potentially lead to life-threatening infection [52]. In 1997, a sudden increase in needle abscesses caused by a clonal strain of Group A

Streptococcus was reported among hospitalised IDUs in Berne, Switzerland [53]. A recent study in the UK showed that the predominant isolate is *S. aureus*, with *Streptococcus* species forming just under one fifth (50% beta-haemolytic streptococci) [54]. There have also been reports of both non-sporing and sporing anaerobes (e.g. *Bacteroides* and *Clostridia* species including *Clostridium botulinum*) [55, 56]. In terms of numbers, in 2003–2004, injecting drug use was one of the most important risk factors for Group A streptococcal infection in the United Kingdom accounting for 20%. More recently, the number of cases has fallen year on year [51].

In particular, in 2000, laboratories in Glasgow were reporting isolates of *C. novyi* among IDUs with “serious unexplained illness”. By 12 June 2000, a total of 42 cases (18 definite and 24 probable) had been reported. A definite case was defined as an IDU with both severe local and systemic inflammatory reactions. A probable case was defined as an IDU who presented to hospital with an abscess or other significant inflammation at an injecting site and had either a severe inflammatory process at or around an injection site or a severe systemic reaction with multiorgan failure and a high white cell count [57].

In the UK, the presence of *C. botulinum* in infected injection sites is a relatively new phenomenon. Up to the end of 1999, there were no cases reported to the PHLS. Since then the number has increased with a total of 13 cases in the UK and Eire being reported since the beginning of 2002. It is thought that these cases are associated with contaminated batches of heroin. Simultaneous injection of cocaine increases the risk by encouraging anaerobic conditions. Anaerobic flora in wounds may have serious consequences for the detainee but the risk of transmission to staff is virtually non-existent. By the end of 2008, a cumulative total of 132 suspected cases have been reported from the United Kingdom, with 86% of cases occurring in England. Four cases were reported in 2008 alone [51].

More recently in December 2009, two injecting drug users in Scotland died from anthrax contaminated heroin. Following this, two further deaths occurred in England in February 2010. An alert was issued first by the Department of Health and then, by the National Treatment Agency warning any drug user of the possible risks. Spores from the bacillus anthracis can contaminate heroin without any obvious signs. Anthrax can be acquired by injecting, smoking or inhaling heroin. Obvious symptoms include excessive swelling and redness at injection sites, fever, headache or shortness of breath when the heroin is smoked. Users are advised to go straight to the Emergency Department if they are at all worried. Such infections with spore forming bacteria will always occur from time to time. Healthcare practitioners should be mindful at all times.

Management in Custody

Staff should be reminded to wear gloves when coming into contact with detainees with infected skin sites exuding pus or serum and that any old dressings found in the cell should be disposed of into the yellow bag marked “clinical waste” in the medical

room. Likewise, any bedding should be bagged and laundered or incinerated after use. The cell should be deemed out of use and professionally cleaned after the detainee has gone.

The healthcare professional managing the detainee should clean and dress open wounds as soon as possible to prevent the spread of infection. It may also be appropriate to start a course of antibiotics if there is abscess formation, signs of cellulitis and/or the detainee is systemically unwell. However, infections can often be low-grade because the skin, venous and lymphatic systems have been damaged by repeated penetration of the skin. In these cases, signs include lymphoedema, swollen lymph glands and darkly pigmented skin over the area. Fever may or may not be present, but septicaemia is uncommon unless the individual is immunocompromised (e.g. HIV positive). Co-Amoxiclav is the preferred treatment of choice as this covers majority of staphylococci, streptococci and anaerobes (the dose used is dependent on the degree of infection).

Necrotising fasciitis and septic thrombophlebitis are rare but life-threatening complications of iv drug use. Any detainee suspected of either of these needs hospital treatment. Advice about harm reduction should also be given. This includes encouraging drug users to smoke rather than inject or at least to advise them to avoid injecting into muscle or skin. Although most injecting drug users are aware of the risk of sharing needles, they may not realise that sharing any drug paraphernalia could be hazardous. Advice should be given to use the minimum amount of citric acid to dissolve the heroin as the acid can damage the tissue under the skin allowing bacteria to flourish. Drugs should be injected at different sites using fresh works for each injection. This is particularly important when “snowballing” as crack cocaine creates an anaerobic environment. Medical help should be requested if any injection site become painful and swollen or shows signs of pus collecting under the skin. Since intravenous drug users are at increased risk of acquiring HBV and HAV, they should be informed that vaccination against both diseases is advisable.

Another serious but relatively rare problem is the risk from broken needles in veins. Embolisation can take anything from hours to days or even longer if it is not removed. Complications may include endocarditis, pericarditis or pulmonary abscesses [58, 59]. IVDUs should be advised to seek medical help as soon as possible, and should such a case present in custody, then send the detainee straight to hospital.

Management of Human and Dog Bites

The Forensic Physician May Encounter Bites in Four Circumstances

1. During the examination of victims of assault (both children and adults) where presentation is more likely to be late
2. Among police officers bitten during the arrest of a detainee

3. In detainees during the arrest if dogs have been used
4. Where detainees have been involved in a fight either around the time of arrest or earlier

A detailed forensic examination of bites is given in Chap. 4. With any bite that has penetrated the skin, the goals of therapy are to minimise soft tissue deformity and to prevent or treat infection.

Epidemiology

In the UK and USA, dog bites represent approximately three-quarters of all bites presenting to Accident and Emergency departments [60]. A single dog bite can produce up to 220 psi of crush force in addition to the torsional forces as the dog shakes its head. This can result in massive tissue damage. Human bites may cause classical bites or puncture wounds (e.g. impact of fists on teeth) resulting in crush injuries.

Rates and Risks of Infection

An estimated 10–30% of dog bites and 9–50% of human bites lead to infection. Compare this with an estimated 1–12% of non-bite wounds managed in Accident and Emergency Departments.

The risk of infection is increased with puncture wounds, hand injuries, full thickness wounds, wounds requiring debridement, and those involving joints, tendons, ligaments or fractures.

Co-morbid medical conditions such as diabetes, asplenia, chronic oedema of the area, liver dysfunction, the presence of a prosthetic valve or joint and an immunocompromised state may also increase the risk of infection.

Other Complications of Bites

Infection may spread beyond the initial site, leading to septic arthritis, osteomyelitis, endocarditis, peritonitis, septicaemia and meningitis. Inflammation of the tendons or synovial lining of joints may also occur. If enough force is used, bones may be fractured or the wounds may be permanently disfiguring.

Initial Management

Assessment as to whether hospital treatment is necessary should be made as soon as possible. Always refer if the wound is bleeding heavily or fails to stop when pressure is applied. Penetrating bites involving arteries, nerves, muscles, tendons, the

hands or feet or resulting in a moderate-to-serious facial wound, or crush injuries also require immediate referral.

If management within custody is appropriate, then ask about current tetanus vaccine status, HBV vaccination status and known allergies to antibiotics.

Wounds that have breached the skin should be irrigated with 0.9% (isotonic) sodium chloride or Ringer's lactate solution in preference to antiseptics, as the latter may delay wound healing.

A full forensic documentation of the bite should be made as detailed in Chap. 4.

Note if there are clinical signs of infection such as erythema, oedema, cellulitis, purulent discharge, or regional lymphadenopathy. Cover the wound with a sterile, non-adhesive dressing. Wound closure is not generally recommended as data suggest that this may increase the risk of infection. This is particularly relevant for non-facial wounds, deep puncture wounds, bites to the hand, clinically infected wounds, and those occurring more than 6–12 h before presentation. Head and neck wounds in cosmetically important areas may be closed if less than 12 h old and not obviously infected.

Pathogens Involved

1. Bacteria

- Dog bites – *Pasteurella canis*, *P. multocida*, *S. aureus*, other staphylococci, *Streptococcus* species, *Eikenella corrodens*, *Corynebacterium* species and anaerobes including *Bacteroides fragilis* and *C. tetani*.
- Human bites – *Streptococcus* species, *S. aureus*, *E. corrodens*, and anaerobes including *Bacteroides* (often penicillin resistant), peptostreptococci species, and *C. tetani*. TB and syphilis may also be transmitted.

2. Viruses

- Dog bites – Outside of the UK, Australia and New Zealand, rabies should be considered. In the USA, domestic dogs are mostly vaccinated against rabies [61], and police dogs have to be vaccinated, so the most common source is from racoons, skunks and bats.
- Human bites – HBV, HCV, HIV, and herpes simplex.

Antibiotic Prophylaxis

Antibiotics are not generally needed if the wound is more than 2 days old and there is no sign of infection or in superficial non-infected wounds evaluated early that can be left open to heal by secondary intention in compliant people with no significant co-morbidity [62]. Antibiotics should be considered with high-risk wounds that involve the hands, feet, face, tendons, ligaments, joints or suspected fractures or for any penetrating bite injury in a person with diabetes, asplenia, cirrhosis, or who is immunosuppressed.

Co-amoxiclav (amoxicillin and clavulanic acid) is the first-line treatment for mild–moderate dog or human bites resulting in infections managed in primary care. For adults, the recommended dose is 500/125 mg tds and for children 40 mg/kg tds (based on amoxicillin component). Treatment should be continued for 10–14 days. It is also the first-line drug for prophylaxis when the same-dose regime should be prescribed for 5–7 days. If the individual is known or suspected to be allergic to penicillin, then a tetracycline (e.g. doxycycline 100 mg bd) and metronidazole (500 mg tds) or an aminoglycoside (e.g. erythromycin) and metronidazole can be used. In the UK, doxycycline use is restricted to those over 12 and in the USA over 8 years old. Specialist advice should be sought for pregnant women.

Anyone with severe infection or who is clinically unwell should be referred to hospital. Tetanus vaccine should be given if the primary course or last booster was more than 10 years ago. Human tetanus immunoglobulin should be considered for tetanus-prone wounds (e.g. soil contamination, puncture wounds, signs of devitalised tissue, or for wounds sustained more than 6 h old). If the person has never been immunised or is unsure of their tetanus status, then a full three-dose course, spaced at least a month apart, should be given.

Management of Suspected Viral Infections from Human Bites

Penetrating bite wounds that involve only saliva may present a risk of HBV if the perpetrator belongs to a high-risk group. For management see “Disease Prevention” and “Management in Custody” in section “Hepatitis B”. HCV and HIV are only a risk if blood is involved. The relevant management is dealt with in “Management in Custody” in section “Hepatitis C” and “Management in Custody of Staff/Victims in Contact with Disease” in section “Human Immunodeficiency Virus”.

Infections Transmitted Through the Respiratory Route

General Information

Respiratory tract infections are common, are usually mild and self-limiting, though may require symptomatic treatment with paracetamol or a non-steroidal anti-inflammatory. These include the common cold (80% – rhinoviruses and 20% – coronaviruses), adenoviruses, influenza and parainfluenza, and during the summer and early autumn – enteroviruses. Special attention should be given to asthmatics or the immunocompromised detainee, as infection in these people may be more serious particularly if the lower respiratory tract is involved.

The following section includes respiratory pathogens of special note as they may pose a risk to both the detainee and/or staff who come into close contact.

Meningococcal Meningitis (Neisseria meningitidis)

General Information and Epidemiology

There are five serogroups of *Neisseria meningitidis*: A, B, C, W135 and Y. The prevalence of the different types varies from country to country. There is currently no available vaccine against type B, but two other vaccines (C and ACWY) are available. Overall, 10% of the UK population carry *N. meningitidis* (25% in the 15–19 age group) [63].

In the United Kingdom, most cases of meningitis are sporadic with less than 5% occurring as clusters (outbreaks) among school children. Between 1996 and 2000, 59% of cases were group B, 36% group C, and W135 and A accounted for 5%. There is a seasonal variation with a high level of cases in winter and a low level in the summer. The greatest risk group are the under 5s with a peak incidence under 1 year old. A secondary peak occurs in 15–19 years-old age group. In Sub-Saharan Africa, the disease is more prevalent in the dry season, but in many countries there is background endemicity all year round. The most prevalent serogroup is A.

Routine vaccination against group C was introduced in the UK in November 1999 for everybody up to the age of 18 and to all first-year university students. This has since been extended to include everyone under the age of 25. As a result of the introduction of the vaccination programme, there has been a 90% reduction of group C cases in the under 18s and an 82% reduction in those under 1 year old [64, 65].

An outbreak of serogroup W135 meningitis occurred among pilgrims on the Hajj in 2000. Cases were reported from many countries including the UK. In the UK, there is now an official requirement to be vaccinated with the quadrivalent vaccine (ACWY Vax) before going on a pilgrimage (Hajj or Umra), but illegal immigrants may enter the country who have not been vaccinated [66].

Symptoms

Following an incubation period of 3–5 days [67, 68], disease onset may either be insidious with mild prodromal symptoms or florid. Early symptoms and signs include malaise, fever and vomiting. Severe headache, neck stiffness, photophobia, drowsiness and a rash may develop. The rash may be petechial or purpuric and characteristically does not blanch under pressure. Meningitis in infants is more likely to be insidious in onset and lack the classical signs. In approximately 15–20% of cases, septicaemia is the predominant feature. Even with prompt antibiotic treatment, the case fatality rate is 3–5% in meningitis and 15–20% in those with septicaemia [33].

Period of Infectivity

A person should be considered infectious until the bacteria are no longer present in nasal discharge. With treatment, this is usually approximately 24 h.

Routes of Transmission

The disease is spread through infected droplets or direct contact from carriers or those who are clinically ill. It requires prolonged and close contact, so is a greater risk for people who share accommodation, utensils, and kiss. It must also be remembered that unprotected mouth-to-mouth resuscitation can also transmit disease.

Management in Custody

It is not possible to tell if a detainee is a carrier. Nevertheless, the risk of acquiring infection even from an infected and sick individual is low unless they have carried out mouth-to-mouth resuscitation. Any staff member who thinks they have been placed at risk should report to the Occupational Health Department (or equivalent) or the nearest emergency department at the earliest opportunity for vaccination.

If they have performed mouth-to-mouth resuscitation, then prophylactic antibiotics should be given before receiving vaccination. Rifampicin, ciprofloxacin and ceftriaxone can be used; however, ciprofloxacin has a number of advantages [69]. Only a single dose of 500 mg (adults and children over 12) is needed and has fewer side effects and contraindications than rifampicin. Ceftriaxone has to be given by injection and is therefore best avoided in the custodial setting.

If the staff member is pregnant, then advice should be sought from a consultant obstetrician, as ciprofloxacin is not recommended [70].

For anyone dealing on a regular basis with illegal immigrants (especially from the Middle East or Sub-Saharan Africa), e.g. immigration services, custody staff at designated stations, medical personnel and interpreters, should consider being vaccinated with ACWY Vax. A single injection provides protection for 3 years. Detainees suspected of disease should be sent directly to hospital.

Tuberculosis

Prevalence and Epidemiology

Human tuberculosis (TB) is caused by infection with *Mycobacterium tuberculosis*, *M. bovis* or *M. africanum*. It is a notifiable disease under legislation specific to individual countries, for example in the UK this comes under the Public Health (Control of Disease) Act 1984. In 1993, the WHO declared TB to be a global

emergency with an estimated 7–8 million new cases and 3 millions deaths occurring each year, the majority of which were in Asia and Africa. However, these statistics are likely to be an underestimate since they are dependent on the accuracy of reporting, and in poorer countries, the surveillance systems are often inadequate through lack of funds.

Even in the UK, there has been an inconsistency of reporting particularly where an individual has concomitant infection with HIV. Some physicians found themselves caught in a dilemma of confidentiality until 1997, when the codes of practice were updated to encourage reporting with patient consent [71].

With the advent of rapid identification tests and treatment, and the use of BCG vaccination for prevention, TB declined during the first half of the twentieth century in the UK. However, since the early 1990s, numbers have slowly increased with some 6,800 cases reported in 2002 [72]. In 1998, 56% of reported cases were from people born outside the UK and 3% were associated with HIV infection [73, 74].

London has been identified as an area with a significant problem. This has been attributed to its highly mobile population, the variety of ethnic groups, a high prevalence of HIV, and the emergence of drug-resistant strains (1.3% in 1998 – PHLS unpublished data – Mycobnet).

A similar picture was initially found in the USA, when there was a reversal of a long-standing downward trend in 1985. However, between 1986 and 1992, the number of cases increased from 22,201 to 26,673 [75]. There were also serious outbreaks of multi-drug-resistant TB (MDR-TB) in hospitals in New York City and Miami [76]. Factors pertinent to the overall upswing included the emergence of HIV, the increasing numbers of immigrants from countries with a high prevalence of tuberculosis, and perhaps more significantly, the stopping of categorical federal funding for control activities in 1972. The latter led to a failure of the public health infrastructure for TB control. Since 1992, the trend has reversed as the CDC transferred most of its funds to tuberculosis surveillance and treatment programmes in states and large cities. From 1992 to 2001, the annual decline averaged by 7.3% [77], but the following year this was reduced to 2%, indicating that there was no room for complacency. The WHO has been proactive and redirecting funding to those countries most in need. In October 1998, a global partnership called Stop TB was launched to co-ordinate every aspect of TB control, and by 2002, the partnership had over 150 member states. A target was set to detect at least 70% of infectious cases by the year 2005.

The acquisition of tuberculosis infection is not necessarily followed by disease as the infection may heal spontaneously. It may take weeks or months before disease becomes apparent or infection may remain dormant for years before reactivation in later life especially if the person becomes debilitated or immunocompromised. Contrary to popular belief, the majority of cases of TB in immunocompetent people pass unnoticed. Of reported cases, 75% involve the lung, while non-respiratory (e.g. bone, heart, kidney, brain) or dissemination (miliary TB) are more common in immigrant ethnic groups and the immunocompromised [78]. They are also more likely to develop resistant strains. In the general population, there is an estimated 10% lifetime risk of tuberculosis infection progressing to disease [79].

Table 10.6 Symptoms of TB

Cough lasting >3 weeks
Fatigue
Anorexia and weight loss
Fever and night sweats
Mild haemoptysis (rusty coloured)
Cough with phlegm
Swollen lymph glands

There has been an increase in the number of cases of tuberculosis associated with HIV either due to new infection or reactivation. Tuberculosis infection is more likely to progress to active TB in HIV positive individuals with a greater than 50% lifetime risk [80]. TB can also lead to a worsening of HIV with an increase in viral load [81]. Therefore, the need for early diagnosis is paramount but it can be more difficult as pulmonary TB may present with non-specific features, e.g. bilateral, unilateral or lower lobe shadowing [82].

Symptoms of Pulmonary TB

After an incubation of 4–12 weeks, symptoms may develop (see Table 10.6).

Routes of Transmission

The main route is airborne through infected droplets, but prolonged or close contact is needed. Non-respiratory disease is not considered a risk unless the *Mycobacterium* is aerosolised under exceptional circumstances (e.g. during surgery) or there are open abscesses.

Period of Infectivity

A person is considered infectious as long as viable bacilli are found in induced sputum. Untreated or incompletely treated people may be intermittently sputum-positive for years.

Following 2 weeks of appropriate treatment, the individual is usually considered as non-infectious. This period is often extended for treatment of MDR-TB or for those with concomitant HIV. Patient compliance also plays an important factor.

At-Risk Groups

The risk of infection is directly proportional to the degree of exposure. More severe disease occurs in the malnourished, or the immunocompromised (e.g. HIV etc.) and substance misusers.

Immunocompromised people are at special risk of MDR-TB or *Mycobacterium avium-intracellulare* (MAI).

Management in Custody

Staff with disease should stay off work until the treatment course is complete and serial sputum samples no longer contain bacilli. Staff in contact with disease and have been vaccinated with BCG are at low risk of acquiring disease, but should minimise the time spent in the cell. Those who have not received BCG or who are immunocompromised should avoid contact with the detainee wherever possible. Detainees with MAI do not pose a risk to a staff member unless the latter is immunocompromised. Any staff member who is pregnant, regardless of BCG status or type of TB, should avoid contact all together.

Anyone performing mouth-to-mouth resuscitation with a person with untreated or suspected pulmonary TB should be regarded as a household contact and should report to Occupational Health or their GP if no other route exists. They should also be educated as to the symptoms of TB. Anyone who is likely to come into repeated contact with individuals at risk of TB should receive BCG (if they have not already done so) regardless of age, even though there is evidence to suggest that BCG administered in adult life is less effective. This does not apply to immunocompromised individuals or pregnant women. In the latter case, vaccination should preferably be deferred until after delivery.

Detainees with disease (whether suspected or diagnosed) who have not been treated or treatment is incomplete should be kept in custody for the minimum time possible. Immunocompromised individuals with TB are usually too ill to be detained, but if they are, they should be considered at greater risk of transmitting disease to staff. Any detainee with disease should be encouraged to cover their mouth and nose when coughing and sneezing.

Staff should wear gloves when in contact with the detainee and when handling clothing and bedding. Any bedding should be bagged after use and laundered or incinerated. The cell should be deemed out of action until it has been ventilated and professionally decontaminated, although there is no hard evidence to support that there is a risk of transmission from this route [73].

Severe Acute Respiratory Syndrome

General Information

On March 14 2003, the WHO issued a global warning to health authorities about a new atypical pneumonia called SARS. The earliest case was believed to have originated in the Guandong province of China on 16 November 2002. The causative agent was identified as a new Corona virus – SARS-CoV [83, 84]. By the end of June 2003, 8,422 cases had been reported from 31 different countries with a total of

916 deaths. Approximately 92% of cases occurred in China (including Hong Kong, Taiwan and Macao). The case fatality rate varied from <1% in people less than 24 years old, 6% in persons aged 25–44, 15% in the 44–64 years age group and >50% in persons 65 or older. On 5 July 2003, the WHO reported that the last human chain of transmission of SARS had been broken and lifted the ban from all countries. However, they warned that everyone should remain vigilant, as resurgence of SARS was still a possibility and this indeed proved the case with reports ongoing until May 2004. Since then, there have been no further reported cases. Knowledge about the epidemiology and ecology of SARS-CoV and the disease remains limited; however, the experience gained from the previous outbreak enabled the disease to be contained rapidly – reflected in the few cases reported since December. There is still no specific treatment or preventative vaccine that has been developed to date.

Incubation Period and Symptoms

The incubation period is short – around 3–6 days (maximum 10 days), and despite the media frenzy surrounding the initial outbreak, SARS is less infectious than influenza. The following clinical case definition of SARS has been developed for public health purposes [85].

A person with a history of:

- Fever (at least 38°C)
- And, one of more symptoms of lower respiratory tract illness (cough, difficulty in breathing, dyspnoea)
- And, radiographic evidence of lung infiltrates consistent with pneumonia or Respiratory Distress Syndrome, or post mortem findings of the above with no identifiable cause
- And, no alternative diagnosis can fully explain the illness

Laboratory tests have been developed which include detection of viral RNA by PCR from nasopharyngeal secretions or stool samples, detection of antibodies by ELISA or IFA in the blood, and viral culture from clinical specimens.

Route of Transmission

Available information suggests that close contact via aerosol or infected droplets from an infected individual provides the highest risk of acquiring disease. Most cases occurred in hospital workers caring for an index case or their close family members.

Management in Custody

Despite the re-emergence of SARS, it is highly unlikely that a case will be encountered in the custodial setting at the time of writing. However, forensic physicians

need to remain alert for the symptoms of SARS and keep up-to-date with recent outbreaks. Information can be obtained from the WHO on a daily basis from their web site. If SARS is suspected, then medical staff should wear gloves and a surgical mask when examining a suspected case; however, masks are not usually available in custody. Anyone suspected of SARS must be sent immediately to hospital and staff who have had prolonged close contact should be alerted as to the potential symptoms.

Infections Transmitted Through the Faecal-Oral Route

General Considerations

The most consistent feature of diseases transmitted through the faecal-oral route is diarrhoea (see Table 10.7). Infective agents include bacteria, viruses and protozoa. Since the causes are numerous, it is beyond the remit of this chapter to cover them all. It is safest to treat all diarrhoea as infectious unless the detainee has a proven non-infectious cause (e.g. Crohn's disease, ulcerative colitis).

All staff should wear gloves when in contact with the detainee or handling clothing, bedding etc. and contaminated articles should be laundered or incinerated. The cell should be professionally cleaned after use paying particular attention to the toilet area.

Hepatitis A

Epidemiology and Prevalence

This viral hepatitis occurs worldwide with variable prevalence. It is highest in countries where hygiene is poor and infection occurs all year round. In temperate climates, the peak incidence is in autumn and winter, but the trend is becoming less marked.

All age groups are susceptible if they are non-immune or have not been vaccinated. In developing countries, the disease occurs in early childhood, whereas the reverse is true in countries where the standard of living is higher.

In the UK, there has been a gradual decrease in the number of reported cases from 1990 to 2000 [86, 87]. This is due in part to improved standards of living and the introduction of an effective vaccine. The highest incidence occurs in the 15–34-year-old age group. Approximately 25% of people over the age of 40 have natural immunity leaving the remainder susceptible to infection [88].

Small clusters occur from time to time, associated with a breakdown in hygiene. There is also an increasing incidence of HAV in gay or bisexual men and their partners [89]. An unpublished study in London in 1996 showed a seroprevalence of 23% among gay men (Young Y et al., unpublished).

Table 10.7 Common causes of infective diarrhoea

Cause	Symptoms	Incubation	Infectivity	Notes
<i>Campylobacter</i>	C, F, N, V, BD	1–10 Days	Untreated – 7 weeks	Requires antibiotics Seek advice
<i>E. coli</i> O157:H7	BD (or WD), F unusual	3–8 Days	Up to 7 days	Acute phase exclude from custody Person to person spread Can be serious with TTP, HUS, dehydration Seek advice
Norwalk virus	N, V, D, AP, mild F	24–48 h	Up to 48 h after diarrhoea stops	Mild to moderate. Self-limiting
Rotavirus	F, V, WD	24–72 h	Up to 8 days Up to 30 days in immuno-compromised	Symptomatic treatment Persists in environment
<i>Salmonella</i>	H, AP, D, N, F, ±V	6–72 h	Days to weeks	Persistent carriage can occur Requires antibiotics Seek advice
<i>Shigella</i>	DY/WD, F, N, (C, V)	12–96 h	Up to 4 weeks	Usually mild in U.K. Can be severe in IC Requires antibiotics in custody Take advice Person to person spread

AP Abdominal pain; H headache; BD bloody diarrhoea; HUS haemolytic-uraemic syndrome; C cramps; IC immunocompromised; D diarrhoea; N nausea; DY dysentery (blood and mucus); TTP thrombotic thrombocytopenic purpura; F fever; V vomiting; WD watery diarrhoea

Symptoms

The clinical picture ranges from asymptomatic infection through a spectrum to fulminant hepatitis. Unlike HBV and HCV, HAV does not persist or progress to chronic liver damage. Infection in childhood is often mild or asymptomatic, but in adults tends to be more severe.

Following an incubation period of 15–50 days (mean 28 days), symptomatic infection starts with the abrupt onset of jaundice anything from 2 days to 3 weeks after the anicteric phase. It lasts for approximately the same length of time and is often accompanied by a sudden onset of fever.

HAV infection can lead to hospital admission in all age groups, but is more likely with increasing age as is the duration of stay.

The overall mortality is less than 1%, but 15% of people will have a prolonged or relapsing illness over 6–9 months (CDC Fact sheet). Fulminant hepatitis occurs in <1% of people, but is more likely over the age of 65 or in those with pre-existing liver disease. In hospitalised patients, case fatality ranges from 2% in 50–59 year olds to nearly 13% in those older than 70 years [87].

Period of Infectivity

The individual is most infectious in the 2 weeks before the onset of jaundice when they are asymptomatic. This can make control of infection difficult since the disease is not recognised.

Routes of Transmission

The main route is faecal-oral through the ingestion of contaminated water and food. It can also be transmitted by close personal contact including homosexuals practising anal intercourse and fellatio. There is a very slight risk from blood transfusions if the donor is in the acute phase of infection. It should not be considered a risk from needlestick injuries unless clinical suspicion of HAV is high.

Risk Groups

Risk groups include the homeless, homosexuals, IVDUs, travellers abroad who have not been vaccinated, patients with chronic liver disease and chronic infection with HBV and HCV, employees and residents in day-care centres and hostels, sewage workers, laboratory technicians and those handling non-human primates.

Several large outbreaks have occurred among injecting drug users – some with an epidemiological link to prisons [90, 91]. Transmission occurs during the viraemic phase of the illness through sharing injecting equipment and via faecal-oral routes because of poor living conditions [92]. There have also been reports of HAV being

transmitted through drugs that have been carried in the rectum. A study in Vancouver showed that 40% of injecting drug users had past infection of HAV and they also showed an increased prevalence among men who have sex with men [93].

Management in Custody

Staff with disease should report to Occupational Health and stay off work until the end of the infective period. Those in contact with disease (either through exposure at home or from an infected detainee) should receive prophylactic treatment as soon as possible (see below).

To minimise the risk of acquiring disease in custody, staff should wear gloves when dealing with the detainee then wash their hands thoroughly. Gloves should only be disposed of in the clinical waste bags.

Detainees with disease should be kept in custody for the minimum time possible. They should only be sent to hospital if fulminant hepatitis is suspected. The cell should be quarantined after use and professionally cleaned. Any bedding or clothing should be handled with gloves and laundered or incinerated according to local policy. Detainees reporting contact with disease should be given prophylactic treatment as soon as possible (see section “Prophylaxis and Treatment”).

Prophylaxis and Treatment

Contacts of HAV should receive HAV vaccine (e.g. Havrix Monodose or Avaxim) if they have not been previously immunised or had disease. Human normal immunoglobulin (HNIG) 500 mg deep im in gluteal muscle should be used in the following circumstances:

- The contact is over 50.
- Has cirrhosis or pre-existing HBV, HCV, or HDV.
- Contact has occurred >8 days but <28 days from exposure.

Staff at higher risk of coming in contact with HAV should consider being vaccinated prior to exposure. Two doses of vaccine given 6–12 months apart give at least 10 years of protection.

There is no specific treatment for HAV except supportive measures and symptomatic treatment.

Exotica

Although the chance of encountering a tropical disease in custody is small, it is worth bearing in mind. It is not necessary for a forensic physician to be able to diagnose the specific disease, but simply to recognise that the detainee/staff member is ill and whether or not they need to be sent to hospital (see Tables 10.8–10.10).

Table 10.8 Suspicion of exotica? History and examination aide memoir

Has the detainee travelled to Africa, South East Asia, the Indian Sub-continent, Central/South America or the Far East in the last 6–12 months

Ascertain whether they received any vaccinations prior to travel and what

Ask if they took malaria prophylaxis, what type and whether they completed the course

Ask if they swam in any stagnant lakes during the trip

If yes to any of the above, ask if they have experienced any of the following symptoms:

- A fever/hot or cold flushes/shivering
- Diarrhoea \pm abdominal cramps \pm blood or slime in the stool
- A rash
- Persistent headaches \pm light sensitivity
- Nausea or vomiting
- Aching muscles/joints
- A persistent cough (dry or productive) lasting at least 3 weeks
- Take temperature
- Check skin for signs of a rash and note nature and distribution
- Check throat
- Listen carefully to the lungs for signs of infection/consolidation

Table 10.9 Tropical diseases that present with fever

Disease	Countries	Incubation	Transmission	Management
Dengue	Most hot climates	3–14 Days	Mosquito No person to person in UK	Symptomatic
Hantavirus	Eastern Europe	2 Days to 8 weeks	No person to person in UK	Symptomatic
Lassa Fever	West Africa			Hospital
Malaria	Sub-Saharan Africa SE. Asia S. America	7 Days to 1 year	Mosquito No person to person in UK	Requires urgent treatment
Typhoid	Hot climates	Up to 72 h	Oral-faecal	Hospital Requires antibiotics
Yellow fever	Sub-Saharan Africa Parts of South America	3–6 Days	Mosquito No person to person in UK	Hospital

Table 10.10 Tropical diseases that present with diarrhoea

Disease	Incubation	Infectivity	Transmission	Management
Amoebic dysentery	Days to months	Years	Oral-faecal	Requires antibiotics
Cholera	Hours to 5 days	3–5 Days after recovery	Oral-faecal Vomit	Requires antibiotics
Giardia	3–25 Days	Months	Oral-faecal	Treat with tinidazole
Malaria	7 Days to 1 year	None	No person to person	Urgent treatment Hospital
Typhoid	Up to 72 h	Days to weeks	Oral-faecal	Requires antibiotics

This is best achieved by knowing the right questions to ask and carry out the appropriate examination. Tables 10.8–10.10 should be used as an aide memoir in order not to miss some more unusual diseases.

References

1. UK Health Guidelines (1998) Guidance for clinical health care workers: protection against infection with blood-borne viruses: recommendations of the Expert Advisory Group on AIDS and the Advisory Group on Hepatitis [HSC 1998/063]. NHS Executive, London, UK
2. Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force (2002) Guidelines for hand hygiene in health care settings. *MMWR Morb Mortal Wkly Rep* 51:1–44
3. National Occupational Health and Safety Committee (1994) National model regulations for the control of workplace hazardous substances. Commonwealth of Australia [NOHSC:1005]
4. Nicholson F (2009) Infectious diseases and an at risk exposure (Chapter 4). In: Stark MM, Rogers DJ, Norfolk GA (eds) Good practice guidelines for forensic medical examiners, 2nd edn. Royal Military Police, Oxford, UK
5. Bolyard EA, Tablan OC, Williams WW, Pearson ML, Shapiro CN, Deitchman SD; and the Hospital Infection Control Practices Advisory Committee (1998) Guideline for infection control in health care personnel. *Am J Infect Control* 26:289–354
6. Report from the Unlinked Anonymous Surveys Steering Group (2001) Prevalence of HIV and hepatitis infections in the United Kingdom 2000. Annual report of the Unlinked Anonymous Prevalence Monitoring Programme. Department of Health, London, UK
7. (2002) A strategy for infectious diseases – progress report. Blood-borne and sexually transmitted viruses: hepatitis. Department of Health, London, UK
8. Centers for Disease Control (CDC) (1988) Perspectives in disease prevention and health promotion update. Universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus and other bloodborne pathogens in health-care settings. *MMWR Morb Mortal Wkly Rep* 37:377–388
9. Martinson FE, Weigle KA, Royce RA, Weber DJ, Suchindran CM, Lemon SM (1998) Risk factors for horizontal transmission of hepatitis B in a rural district in Ghana. *Am J Epidemiol* 147:478–487
10. Verma G, Dalai P, Bapat M, Rathi P, Abraham P (2003) Familial clustering of hepatitis B infection: study of a family. *Indian J Gastroenterol* 22:22–23
11. Erol S, Ozkurt Z, Ertek M, Tasyaran MA (2003) Intrafamilial transmission of hepatitis B in the Eastern Anatolian region of Turkey. *Eur J Gastroenterol Hepatol* 15:345–349
12. Hutchinson S, Goldberg D, Gore S et al (1998) Hepatitis B outbreak at Glenochil prison during January to June 1993. *Epidemiol Infect* 121:185–191
13. Christensen PB et al (1998) European network for HIV/AIDS and hepatitis prevention in prisons. Second annual report. The Network, Bonn and Marseille
14. Weild AR, Gill ON, Bennett D, Livingstone SJM, Parry JV, Curran L (2000) Prevalence of HIV, hepatitis B and hepatitis C antibodies in prisoners in England and Wales; a national survey. *Commun Dis Public Health* 3:121–126
15. Alter MJ (1997) The epidemiology of acute and chronic hepatitis C. *Clin Liver Dis* 1:559–562
16. Frank C, Mohamed MK, Strickland GT (2000) The role of the parenteral antischistosomal therapy in the spread of hepatitis C virus in Egypt. *Lancet* 355:887–891
17. (2003) Chronic hepatitis C: disease management. NIH publication No. 03-4230 Feb 2003. www.digestive.niddk.nih.gov/diseases/pubs/chronichepc/index.htm. Accessed 15 June 2011
18. Chief Medical Examiner, Sir Liam Donaldson (2002) Hepatitis C strategy for England. Department of Health, London, UK
19. Gish RG, Lau JYN (1997) Hepatitis C virus: eight years old. *Viral Hepat Rev* 3:17–37

20. Ramsay ME, Balogun MA, Collins M, Balraj V (1998) Laboratory surveillance of hepatitis C virus in England and Wales: 1992–1996. *Commun Dis Public Health* 1:89–94
21. Interferon alfa (pegylated and non pegylated) and ribavirin for the treatment of chronic hepatitis C. Technology Appraisal 75 (2004) <http://www.nice.org.uk/nicemedia/pdf/TA075guidance.pdf>. Accessed Jan 2010
22. Peginterferon alfa and ribavirin for the treatment of mild chronic hepatitis C. NICE technology appraisal guidance 106 (2006) <http://www.nice.org.uk/nicemedia/pdf/TA106guidance.pdf>. Accessed Jan 2010
23. Hepatitis D, Sean R Lacey, Assistant Professor, Department of Medicine, Case Western Reserve University). <http://emedicine.medscape.com/article/178038-overview>. Last modified June 2011
24. (2003) Cumulative UK data to end of December 2002. AIDS/HIV quarterly surveillance tables provided by the PHLS AIDS centre (CDSC) and the Scottish centre for Infection and Environmental Health. No 57: 02/4
25. Testing Times. HIV and other sexually transmitted infections in the United Kingdom (2007). http://www.hpa.org.uk/web/HPAweb_C/1203496897276. Accessed June 2011
26. International Perinatal HIV Group (1999) Mode of vertical transmission of HIV-1: a meta-analysis of fifteen prospective cohort studies. *N Engl J Med* 340:977–987
27. Duong T, Ades A, Gibbs DM et al (1999) Vertical transmission rate for HIV in the British Isles estimated on Surveillance data. *BMJ* 319:1227–1229
28. Limb S, Kawar M, Forster GE (2002) HIV post-exposure prophylaxis after sexual assault: the experience of a sexual assault service in London. *Int J STD AIDS* 13:602–605
29. HIV post-exposure prophylaxis. Evidence from the UK Chief Medical Officers' Expert Advisory Group on AIDS (2008). http://www.dh.gov.uk/dr_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_089997.pdf. Accessed Jan 2010
30. (2000) HIV post-exposure prophylaxis: guidance from the UK Chief Medical Officer's Expert Advisory Group on AIDS. UK Health Department, London, UK
31. Jochimsen EM (1997) Failures of zidovudine post exposure prophylaxis. *Am J Med* 102: 52–55
32. Hawkins DA, Asboe D, Barlow K, Evans B (2001) Seroconversion to HIV-1 following a needlestick injury despite combination post-exposure prophylaxis. *J Infect* 43:12–15
33. Salisbury DM, Begg NT (1996) Immunisation against infectious disease. Her Majesty's Stationery Office, London, UK
34. Sinha DP (1996) Chickenpox – disease predominantly affecting adults in rural West Bengal. *Int J Epidemiol* 5:367–374
35. Centers for Disease Control and Prevention (1996) Prevention of *varicella*: recommendations of the Advisory Committee on Immunization Practices. *MMWR Morb Mortal Wkly Rep* 45:1–36
36. Fairley CK, Miller E (1996) *Varicella-zoster* virus epidemiology. A changing scene? *J Infect Dis* 174(suppl 3):314–319
37. Smego RA, Asperilla MO (1991) Use of Acyclovir for *varicella* pneumonia during pregnancy. *Obstet Gynecol* 78:1112–1116
38. Pastuszak AL, Levy M, Schick B et al (1994) Outcome after maternal varicella infection in the first 20 weeks of pregnancy. *N Engl J Med* 330:901–905
39. Miller E, Cradoc-Watson JE, Ridehalgh MK (1989) Outcome in newborn babies given anti-*varicella zoster* immunoglobulin after perinatal maternal infection with *varicella zoster* virus. *Lancet* 2:371–373
40. Gilden DH, Vafai A, Shtram Y et al (1983) *Varicella-zoster* virus DNA in human sensory ganglia. *Nature* 306:478–480
41. Dworkin RH, Schmader KE (2001) Epidemiology and natural history of herpes zoster and post herpetic neuralgia. In: Watson CPN, Gershon AA (eds) *Herpes zoster and postherpetic neuralgia*, 2nd edn. Elsevier, New York, NY, pp 39–64
42. Desmond RA, Weiss HL, Arani RB et al (2002) Clinical applications for change-point analysis of herpes zoster pain. *J Pain Symptom Manage* 23:510–516
43. Gnann JW Jr, Whitley RJ (2002) Herpes zoster. *N Engl J Med* 347:340–346

44. Haustein UF, Hlawa B (1989) Treatment of scabies with permethrin versus lindane and benzoyl benzoate. *Acta Derm Venereol* 69:348–351
45. Brown S, Becher J, Brady W (1995) Treatment of ectoparasitic infections; review of the English-language literature 1982–1992. *Clin Infect Dis* 20(suppl 1):S104–S109
46. Klutymans J, Van Belkum A, Verbrugh H (1989) Nasal carriage of *Staphylococcus aureus*: epidemiology and control measures. *Infect Dis Clin North Am* 3:901–913
47. Lowry FD (1998) *Staphylococcus aureus* infections. *N Engl J Med* 339:520–532
48. Centers for Disease Control and Prevention (1981) Community-acquired methicillin-resistant *Staphylococcus aureus* infections – Michigan. *MMWR Morb Mortal Wkly Rep* 30:185–187
49. Saravolatz LD, Markowitz N, Arking L, Pohlod D, Fisher E (1982) Methicillin-resistant *Staphylococcus aureus*, epidemiologic observations during a community acquired outbreak. *Ann Intern Med* 96:11–16
50. Health Protection Agency (2003) Emergence of PVL-producing strains of *Staphylococcus aureus*. *Commun Dis Rep CDR Wkly* [serial online] 13. <http://www.phls.org.uk/publications>. Accessed Jan 2004
51. Shooting Up. Infections among injecting drug users in the United Kingdom 2008. An update (2009). http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1254510657318. Accessed Jan 2010
52. Summanen PH, Talan DA, Strong C et al (1995) Bacteriology of skin and soft tissue infections: comparison of infections in intravenous drug users and individuals with no history of intravenous drug use. *Clin Infect Dis* 20(suppl 2):S279–S282
53. Bohlen LM, Muhlemann K, Dubuis O, Aebi C, Tauber MG (2000). Outbreak among drug users caused by a clonal strain of group a *Streptococcus*. *Dispatches – emerging infectious diseases*. <http://www.cdc.gov>. Accessed Mar 2003. *Emerg Infect Dis*. 6(2):175–179
54. Lettington W (2002) Bacteriological skin and subcutaneous infections space in injecting drug users – relevance for custody. *J Clin Forensic Med* 9:65–69
55. Passaro DJ, Werner SB, McGee J, MacKenzie WR, Vugia DJ (1998) Wound botulism associated with black tar heroin among injecting drug users. *JAMA* 279:859–863
56. Brazier JS, Duerden BI, Hall V et al (2002) Isolation and identification of *Clostridium* spp from infections associated with injection of drugs: experiences of a microbiological investigation team. *J Med Microbiol* 51:985–989
57. Greater Glasgow Health Board, SCIFH (2001) Unexplained illness among drug injectors in Glasgow. *Eurosurveillance* 4:500518
58. Kuyalaylat MN, Barakat N, Stephan RN, Gutierrez I (1993) Embolization of illicit needle fragments. *J Emerg Med* 11:403–408
59. Ngaage DL, Cowen ME (2001) Right ventricular needle embolus in an injecting drug user: the need for early removal. *Emerg Med J* 18:500–501
60. Spanierman C (2004) Departments of Emergency Medicine and Pediatrics, Lutheran General Hospital of Oak Brook, Advocate Health System. eMedicine-Human Bites. <http://www.emedicine.com/ped/topic246.htm>. Accessed Feb 2004
61. Presutti JP (2001) Prevention and treatment of dog bites. *Am Fam Physician* 63:1567–1572
62. Revis DR, Jr, Seagel MB (2003) Human bites. Department of Plastic Surgery, University of Florida College of Medicine. eMedicine-Human Bites. <http://www.emedicine.com/ent/topic728.htm>. Accessed Feb 2004
63. PHLS (2002) Guidelines for public health management of meningococcal diseases in the UK. *Commun Dis Public Health* 5:187–204
64. Miller E, Salisbury D, Ramsay M (2001) Planning, registration and implementation of an immunisation campaign against meningococcal serogroup C disease in the UK: a success story. *Vaccine* 20(suppl 1):S58–S67
65. Ramsay M, Andrews N, Kaczmarek E, Miller E (2001) Efficacy of meningococcal serogroup C conjugate vaccine in teenagers and toddlers in England. *Lancet* 357:195–196
66. PHLS (2001) Quadrivalent meningococcal immunisation required for pilgrims to Saudi Arabia. *Commun Dis Rep CDR Wkly* 11. <http://www.phls.org.uk/publications>. Accessed 11 May 2004
67. Boutet R, Stuart JM, Kaczmarek E, Gray SJ, Jones M, Andrews N (2001) Risk of laboratory-acquired meningococcal disease. *J Hosp Infect* 49:282–284

68. Orr H, Kaczmarek E, Sarangi J, Pankhania B, Stuart J (2001) Cluster of meningococcal disease in rugby match spectators. *Commun Dis Public Health* 4:316–317
69. CDSC (2001) Ciprofloxacin as a chemoprophylactic agent for meningococcal disease – low risk of anaphylactoid reactions. *Commun Dis Rep CDR Wkly* 11:45
70. Joint Formulary Committee (2002–2003) British National Formulary. British Medical Association and Royal Pharmaceutical Society of Great Britain, London, UK
71. Omerod LP, Watson JM, Pozniak A et al (1997) Notification of tuberculosis an updated code of practice for England and Wales. *J R Coll Physicians Lond* 31:299–303
72. Statutory notifications to the Communicable Disease Surveillance Centre(2002). Preliminary annual report on tuberculosis cases reported in England, Wales, and N. Ireland. <http://www.hpa.org.UK/infections>. Accessed Dec 2003
73. The Interdepartmental Working Group on Tuberculosis (1998) The prevention and control of tuberculosis in the United Kingdom: UK guidance on the prevention and control of transmission of 1. HIV-related tuberculosis 2. Drug-resistant, including multiple drug-resistant, tuberculosis. Department of Health, Scottish Office, The Welsh Office, Scotland
74. Joint Tuberculosis Committee of the British Thoracic Society (2000) Control and prevention of tuberculosis in the United Kingdom: code of practice 2000. *Thorax* 55:887–901
75. Cantwell MF, Snider DE, Cauthen GM, Onorato IM (1994) Epidemiology of tuberculosis in the United States, 1985 through 1992. *JAMA* 272:535–539
76. Centers for Disease Control (1991) Nosocomial transmission of multi-drug resistant tuberculosis among HIV-infected persons – Florida, New York, 1988–1991. *MMWR Morb Mortal Wkly Rep* 40:585–591
77. Navin TR, McNabb SJN, Crawford JT (2002) The continued threat of tuberculosis. *Emerg Infect Dis* [serial online]. <http://www.cdc.gov/ncidod/EID/vol8no11/02-0468>. Accessed Nov 2002
78. Sepkowitz DV (1995) Tuberculosis in HIV-infected individuals (Chapter 5). In: Lutwick LI (ed) *Tuberculosis – a clinical handbook*. Chapman & Hall Medical, London, UK
79. Murray JF (1989) The White Plague: down and out, or up and coming? *Am Rev Respir Dis* 140:1788–1795
80. Selwyn PA, Hartel D, Lewis VA et al (1989) A prospective study of the risk of tuberculosis among intravenous drug users with human immunodeficiency virus infection. *N Engl J Med* 320:545–550
81. Wallis RS, Vjecha M, Amir-Tahmassebi M et al (1993) Influence of tuberculosis on human immunodeficiency virus (HIV-1): enhanced cytokine expression and elevated B₂-microglobulin in HIV-1 associated tuberculosis. *J Infect Dis* 167:43–48
82. Long R, Maycher B, Scalcini M, Manfreda J (1991) The chest roentogram in pulmonary tuberculosis patients seropositive for human immunodeficiency virus type 1. *Chest* 99:123–127
83. Peiris JSM, Lais T, Poon LLM et al (2003) Coronavirus as a possible cause of severe acute respiratory syndrome. *Lancet* 361:1319–1325
84. Donnelly CA, Ghani AC, Leung GM et al (2003) Epidemiological determinants of spread of causal agents of severe acute respiratory syndrome in Hong Kong. *Lancet* 361:761–766
85. World Health Organization. Alert, verification and public health management of SARS in post-outbreak period (2003) <http://www.who.int/csr/sars/postoutbreak/en/>. Accessed 14 Dec 2003
86. Gay NJ, Morgan-Capner P, Wright J, Farrington CP, Miller E (1994) Age-specific antibody prevalence to hepatitis A in England: implications for disease control. *Epidemiol Infect* 113:113–120
87. Crowcroft NS, Walsh B, Davison KL, Gungabissoon U; PHLS Advisory Committee on Vaccination and Immunisation (2001) Guidelines for the control of hepatitis A infection. *Commun Dis Public Health* 4:213–227
88. Irwin DJ, Millership S (1999) Control of a community hepatitis A outbreak using hepatitis A vaccine. *Commun Dis Public Health* 2:184–187
89. Katz MH, Hsu L, Wong E, Liska S, Anderson L, Janssen RS (1997) Seroprevalence of and risk factors for hepatitis A infection among young homosexual and bisexual men. *J Infect Dis* 175:1225–1229

90. Harkess J, Gildon B, Istre GR (1989) Outbreaks of hepatitis A among illicit drug users, Oklahoma, 1984–1987. *Am J Public Health* 79:463–466
91. Hutin YJ, Bell BP, Marshall KL et al (1999) Identifying target groups for a potential vaccination program during a hepatitis A community outbreak. *Am J Public Health* 89:918–919
92. Hutin YJ, Sabin KM, Hutwagner LC et al (2000) Multiple modes of hepatitis A transmission among metamphetamine users. *Am J Epidemiol* 152:186–192
93. Ochnio JJ, Patrick D, Hom TG et al (2001) Past infection with hepatitis A among Vancouver Street youth, injection drug users and men who have sex with men; implications for vaccination programmes. *CMAJ* 165:293–297