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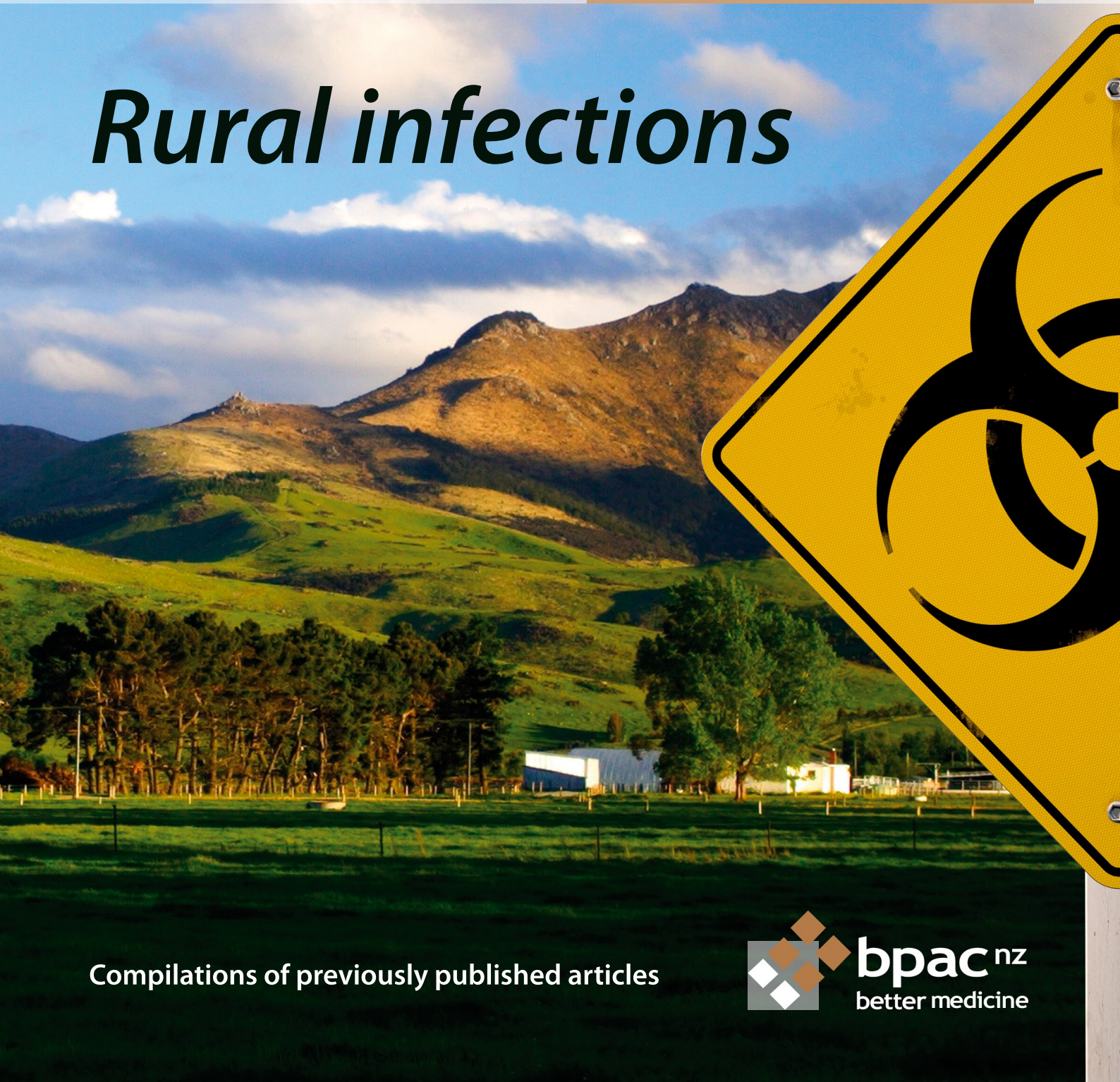
Rural infections



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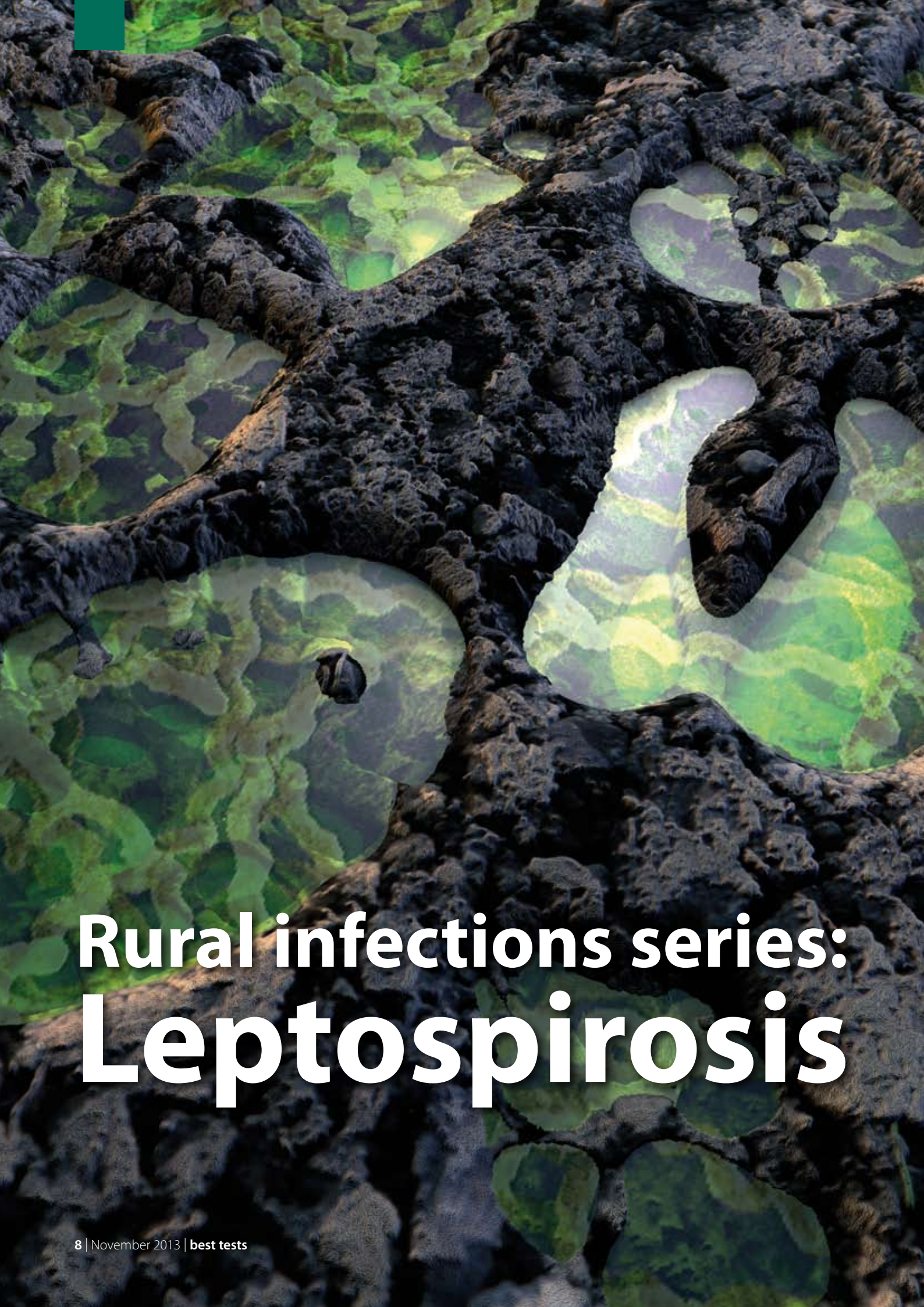
Rural infections series

This article is the first in a series addressing the diagnosis and management of infections that predominantly occur in people who work or live in a rural environment. Most of these infections are caused by bacteria, viruses, fungi or parasites which infect animals but can also pass to humans (known as zoonoses). Examples of rural infections in New Zealand include: leptospirosis, campylobacter (seasonal), giardiasis, orf, cryptosporidium, atypical tuberculosis, rickettsial fever and Q fever.

Most rural infections are rare in the wider New Zealand population and may not be regularly encountered in a typical general practice. Some occur primarily in certain groups or occupations, e.g. leptospirosis in meat processors and farmers. Others have seasonal variations, e.g. campylobacter occurs in urban populations throughout the year, but in spring becomes more prevalent in rural areas as animal handling increases. Some rural infections, such as hydatid parasites, have been successfully eradicated from New Zealand. Others, such as brucellosis, are so rare that they are unlikely to ever be encountered. However, it is important to be aware of all rural infections, as in some instances, they are associated with significant morbidity and potential mortality if not identified early. In addition, reintroduction of infections which have previously been controlled or eradicated could have significant public health and economic consequences.

The rural infections series will cover some of the more common or most clinically significant rural diseases encountered in New Zealand. The first article in this series focuses on the diagnosis, laboratory investigation and management of patients with suspected leptospirosis.



Rural infections series: Leptospirosis

What is leptospirosis?

Leptospirosis is a potentially fatal infectious disease caused by spirochete bacteria of the genus *Leptospira*.[1] It is the most common occupationally-acquired infectious disease in New Zealand, but can be difficult to recognise and diagnose.[2] The incidence of leptospirosis in New Zealand fell considerably from 1980 to 2000,[2] largely due to the introduction of a livestock vaccine for leptospirosis. Incidence has fluctuated since then; the current incidence is 2.5 cases per 100 000 people per year.[3]

Leptospirosis is associated with a broad spectrum of severity, ranging from subclinical infection to severe illness. Typically the infection falls into one of two main clinical syndromes. Most people with leptospirosis will have a self-limiting, influenza-like illness.[4] However, a small proportion of people develop severe illness, often referred to as Weil's disease. This is characterised by jaundice, pulmonary haemorrhage and multiple organ failure.[4] In developed nations, death is rare, but may occur secondary to cardiac arrhythmias, renal failure or pulmonary haemorrhage.[1, 4] At least one confirmed fatal infection has occurred in New Zealand in recent years.[5]

Leptospires pass from mammals, such as rats, dogs, pigs and cattle, to humans across mucous membranes, conjunctivae or broken skin. Infection may occur through direct contact with urine or tissue from infected animals, or indirectly via infected water, damp soil and vegetation.[6, 7] It is rare for human-to-human transmission of leptospirosis to occur.

Because of this mode of transmission, most leptospirosis infections occur in people living or working in an agricultural or rural setting or undertaking recreational activities in these areas. This includes farmers, share milkers, abattoir workers, veterinarians, butchers, drain layers, sewage workers, plumbers, miners, fishermen, hunters, swimmers and trampers. Travellers returning from overseas, particularly from tropical areas, are also at higher risk of exposure to leptospirosis, especially those exposed to certain conditions (e.g. flooding) or activities (e.g. caving or fresh-water sports).

How is leptospirosis diagnosed?

The diagnosis of leptospirosis is usually clinical, with specific laboratory testing used to retrospectively confirm the diagnosis for Notification purposes (see: "Leptospirosis is a Notifiable disease", over page).

Clinical presentation and patient history

The incubation period of leptospirosis varies from 2 – 30 days (mean ten days).[6] The eventual symptomatic illness can range from mild to severe.[8] Approximately 90% of people will have an acute, self-limiting, febrile illness.[8] The remaining 10% will develop a more severe, potentially life-threatening condition.[8] Signs and symptoms of leptospirosis are classically biphasic, although in many severe cases the distinction between the two phases is not apparent.

The initial phase of leptospirosis is an acute-onset febrile illness lasting three to nine days.[8] The most common symptoms are chills or rigors, myalgia, headache and conjunctival suffusion.[4] Conjunctival suffusion is relatively specific to leptospirosis, and typically appears on the third

Leptospirosis is a Notifiable disease

Leptospirosis is a Notifiable disease. Suspected cases should be reported to the local Medical Officer of Health. Investigations should be requested for confirmation of the disease, but it is not necessary to wait for laboratory confirmation before reporting a case.

Confirmation for Notification purposes requires one of the following results:[2]

- Detection of leptospiral nucleic acid from blood, urine or spinal fluid
- A four-fold or greater rise in leptospiral microscopic agglutination titres (MAT) between acute and convalescent sera
- A single agglutination titre of > 400 by MAT
- Isolation of leptospires from blood, urine or spinal fluid

More information on testing can be found in "Investigations", Page 11.

Further information and the required forms for reporting occupational exposures can be found at: **www.business.govt.nz/healthandsafetygroup/notifications-forms/nods**

or fourth day of the illness.[8] It presents as bilateral redness (hyperaemia) and oedema (chemosis) of the conjunctiva, without an inflammatory exudate. An erythematous macular rash, nausea, vomiting and fatigue may also be present, but are less typical features of leptospirosis.[8]

The initial phase is usually (but not always) followed by an asymptomatic period lasting two to three days, before the second (immune) phase begins.[1, 3, 8]

The immune phase occurs as serum IgM antibodies increase and the spirochetes disappear from the blood and cerebrospinal fluid. The response to the antibodies ranges from a more severe form of the initial phase (as above), including aseptic meningitis, to Weil's disease, characterised by jaundice, renal failure, pulmonary symptoms (dyspnoea, chest pain and haemoptysis), myocarditis, cardiac arrhythmias and haemorrhagic diathesis (spontaneous bleeding).[4] In severe infection, multiple organ failure can cause a wide range of symptoms.

Examination

Findings on examination may differ widely among patients. Signs will vary depending on the stage and severity of the illness and the organ systems involved.

A general examination should be performed and will indicate features typical of an infection, such as fever (up to 40°C), tachycardia and muscle tenderness.[8] Localised tenderness in the calf muscles and, in particular, in the paraspinal muscles, is an important, relatively specific finding.[4] Hypotension may be found in patients with severe infection.

A brief eye examination is important for both diagnosis and identification of complications. Photophobia, jaundice and bilateral conjunctival suffusion are often present.[4] Optic neuritis (inflammation of the optic nerve) and uveitis (inflammation of the uvea, including the iris, ciliary body and choroid) can develop as secondary complications.[8]

Palpation of the abdomen may indicate abdominal tenderness and hepatosplenomegaly.[4]

Auscultation of the patient's chest may indicate crackles and wheeze associated with pulmonary oedema. Signs of consolidation, such as bronchial breath sounds, dullness to percussion and reduced chest movement, may be present in severe cases due to pulmonary haemorrhage.

A brief neurological examination should be conducted in patients with suspected leptospirosis with severe signs and symptoms. Aseptic meningitis is suggested by vomiting, headache and meningeal irritation (neck stiffness and photophobia). Immediate referral to hospital is required for anyone presenting with signs and symptoms of suspected meningitis.

Differential diagnosis

The symptoms and signs associated with leptospirosis are non-specific, therefore there are a wide range of other conditions that should be considered in the differential diagnosis, including:[8]

- Influenza
- Other causes of meningitis and meningococcal disease
- Viral hepatitis
- Septicaemia
- HIV seroconversion illness
- Toxoplasmosis
- Other rural infections, e.g. rickettsial infections such as murine typhus (Page 13)
- Tropical diseases, such as yellow fever and malaria; consider in people returning from overseas travel

Investigations

Specific testing for leptospirosis should be used to confirm a suspected diagnosis. However, as the results will not be immediately available (results may take up to three days, or more, depending on the testing laboratory), treatment can be commenced based on the clinical diagnosis.

Serology should be requested first-line for a patient with suspected leptospirosis.[9] Polymerase chain reaction (PCR) testing for DNA should be added if the patient's symptoms are severe or if infection is thought to be acquired through occupational exposure.

N.B. Patients who have laboratory-confirmed leptospirosis due to an occupational exposure are eligible for ACC cover.

Serology is used to retrospectively confirm leptospirosis,[4] and should be requested whenever there is a reasonable suspicion of the infection.[2] Antibodies begin to appear five to seven days after the onset of symptoms,[10] and can remain raised for several months.[7] Two serology samples are required: referred to as the acute and convalescent samples. The first sample should be taken at the initial presentation, with the approximate date of the start of the illness recorded on the form. The second sample should be taken a minimum of 10 – 14 days after the first and ideally at 21 days after the onset of symptoms. A four-fold increase between titre levels in the first and second samples is considered diagnostic of leptospirosis.[10] In some patients, seroconversion is delayed (>30 days), therefore if both samples are negative but there is still a suspicion of leptospirosis, a third serum sample should be requested. Patients with a previous exposure to leptospirosis will often have a positive first sample, but this does not necessarily indicate current infection. An increase in titre levels in the second sample would suggest active infection.

The serology test is specific to the Leptospira genus, but does not differentiate between serovars, i.e. different "strains" of leptospirosis. Positive serology samples are forwarded to Environmental Science & Research (ESR) for microscopic agglutination titres (MAT) which test for antibodies to specific serovars. This information is of limited use for the management of leptospirosis, but is important for epidemiological monitoring.

PCR testing for Leptospira DNA should be requested in addition to serology if the infection is severe, or for confirmation of an occupationally acquired infection, as the results will be available more rapidly than those from paired serology. The type of sample for PCR depends on the duration of the illness: during the first week of signs and symptoms a blood sample should be collected (approximately 5 mL in an EDTA tube; usually purple top). After the first week, a urine sample (at least 20 mL) is used, as leptospires will no longer be reliably detectable in the blood. Due to the intermittent excretion of leptospires in urine, a negative result does not exclude leptospirosis, and a repeat sample may be necessary if there is still a strong clinical suspicion of the infection.

If the patient develops meningitis, a cerebrospinal fluid sample obtained in a secondary care setting can also be used for PCR testing in the first ten days of infection.[10]

Leptospirosis culture is available from some New Zealand laboratories, however, the results usually take a significant time, making it impractical for clinical use.

Other laboratory investigations

Additional laboratory investigations are not necessary for the

diagnosis of leptospirosis. However, some tests may be useful to add evidence to a suspected diagnosis of leptospirosis while waiting for results of leptospirosis-specific testing. Most findings will, however, be non-specific. The following tests may be considered:

- Full blood count – lymphocytopaenia is common in people with leptospirosis.[9] Leukocytes may be low, normal or high, but are commonly associated with a left shift.[8] Thrombocytopaenia is also present in up to 50% of people with leptospirosis.[6]
- LFTs – increases in transaminases, alkaline phosphatase and bilirubin may be seen on liver function tests.[6]
- Serum creatinine – levels may be elevated due to tubular damage and dehydration.[8]
- Urinalysis – proteinuria, pyuria and microscopic haematuria may be present, with granular casts on microscopy.[6]

Managing leptospirosis

It is not necessary to wait for the results from laboratory testing for leptospirosis before starting treatment if there is a strong clinical suspicion of the infection.[1] Discussion with an Infectious Diseases Physician is encouraged in addition to notification to the Medical Officer of Health.

Doxycycline 100 mg, twice daily, for five to seven days is the first-line treatment for leptospirosis in the community setting. Amoxicillin 500 mg, three times daily, for five to seven days is an alternative.[1, 11] Treatment is most effective if antibiotics are initiated within five days of symptom onset, after which the efficacy of antibiotic treatment is less certain.[1, 12] In practice, however, treatment is usually initiated in patients with severe illness regardless of the date of onset.[1]

As with other spirochete infections, e.g. syphilis, antibiotic treatment can be associated with the development of a septicaemia-like reaction in the first few hours after starting treatment, due to the sudden release of endotoxins as the bacteria die.[4] This is referred to as a Jarisch–Herxheimer reaction. This reaction is assumed to be rare, although the exact prevalence in patients with leptospirosis treated with antibiotics is unknown.[13] Patients should be instructed to seek immediate medical attention if they become acutely unwell after starting the course of antibiotics.

When should a patient with leptospirosis be referred?

All patients with severe infection or signs of meningitis should be referred to hospital immediately.[1] Treatment with intravenous antibiotics, e.g. benzylpenicillin 1200 mg IV, every four to six hours, for five to seven days is usually required.[11] Intensive supportive care with particular attention to fluid and electrolyte balance is also often necessary.[1] Further treatment is dependent on complications, e.g. patients who develop acute renal failure may require haemodialysis.[1]

All women who are pregnant and are suspected of having leptospirosis of any severity should be referred to hospital. Leptospiral infection in either early or late pregnancy results in miscarriage or premature delivery in more than 50% of cases.[4]

Consideration should be given to referral to an Infectious Diseases Physician for people with risk factors for developing severe illness. Risk factors include age less than five years or over 65 years and the presence of co-morbidities, such as liver disease or an immunocompromised status.[4]

Preventing future infections

Primary prevention of leptospirosis focuses on educating people to avoid high-risk exposure, such as immersion in fresh water that could be infected, contact with stagnant water and contact with animal urine. However, for many people who are occupationally exposed, avoidance will not be possible. Minimising exposure to animal urine through the use of protective clothing (e.g. gloves, goggles or face shields, gumboots) and good hygiene is recommended. Preventive measures are now widespread in certain industries, such as dairy and meat processing.[5]

Advise patients who have a high level of unavoidable occupational risk to be aware of leptospirosis and its prevention and to present to primary care if they develop flu-like symptoms.

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Murine typhus: an important differential diagnosis

Murine typhus is a flea-borne infection caused by the bacteria *Rickettsia typhi*.[14] Infected fleas are usually carried by rats. In New Zealand it is present in warmer, wetter areas of the North Island, particularly Waikato and Auckland.[9] It is increasingly associated with people who have a rural occupation and/or residence.[9]

Patients with murine typhus present in a similar way to those with leptospirosis, and clinically the two infections are difficult to differentiate. An erythematous macular rash on the trunk is more typical of murine typhus and conjunctival suffusion is more indicative of leptospirosis.[9] A Waikato study found that in people presenting with febrile illness, a low lymphocyte level plus a rural occupation was associated with leptospirosis, whereas a low platelet count (thrombocytopaenia) and a rural residence was associated with murine typhus.[9] However, this would not be sufficient to differentiate between the conditions as leptospirosis can be associated with a low platelet count also.

Serology can be used to differentiate between leptospirosis and murine typhus.[9] It is recommended to also test for murine typhus in patients with suspected leptospirosis who were exposed in areas with higher prevalence of rickettsial infection, e.g. the Waikato. The same sample that has been used for leptospiral serology can be used for rickettsial serology. Indicate on the request form that the laboratory should add rickettsial serology if the leptospiral antibodies are negative.

Patients with murine typhus are managed in the same way as those with leptospirosis; doxycycline is the first-line treatment.[9] Murine typhus is a Notifiable Disease.

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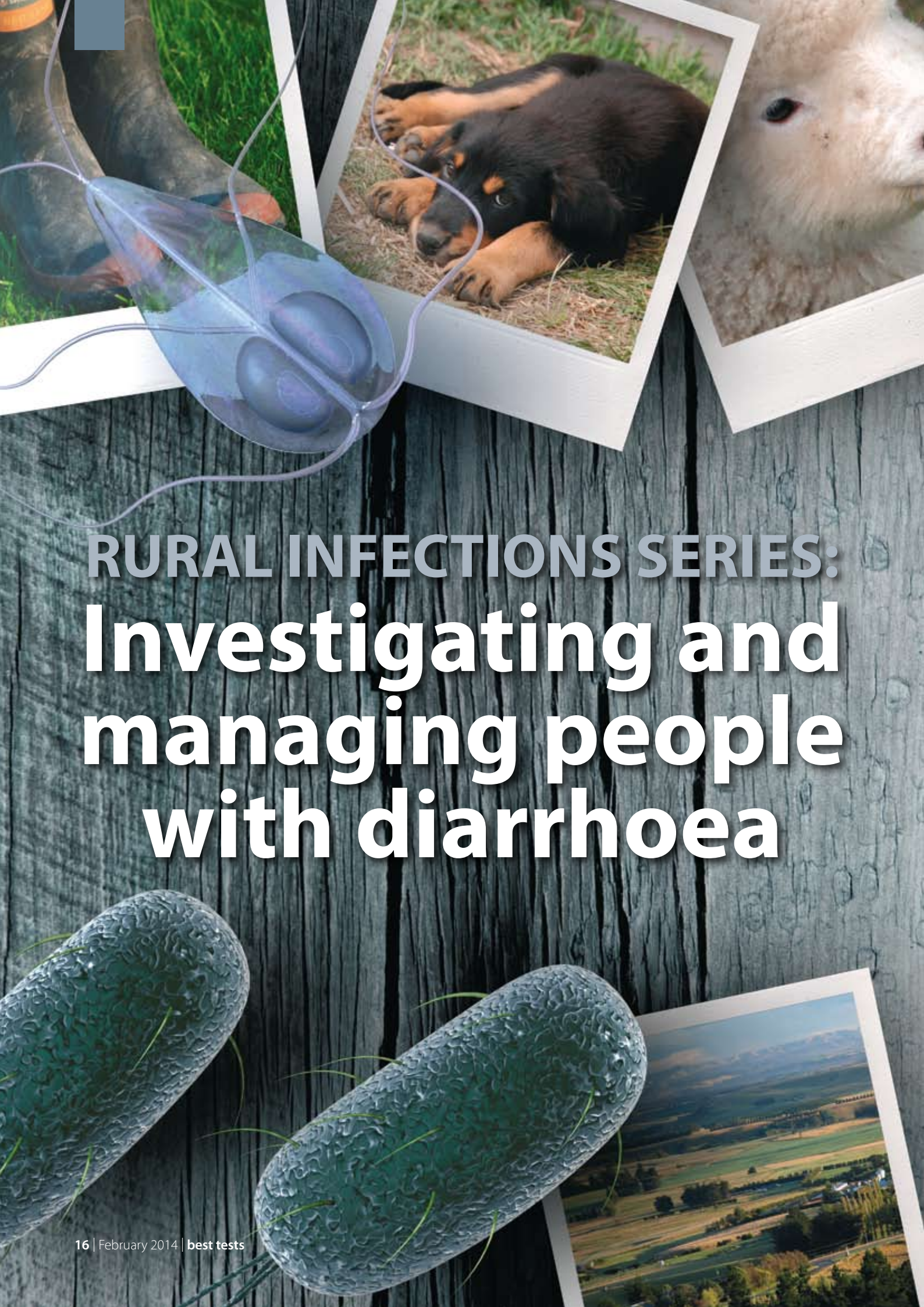

RURAL INFECTIONS SERIES:

Investigating and managing people with diarrhoea

Campylobacter, *Salmonella*, *Cryptosporidium* and *Giardia* cause diarrhoeal illnesses in thousands of people annually in New Zealand. The incidence of these infections is significantly higher in New Zealand compared to most other developed nations.[1] Animal, environmental and waterborne sources are a common cause of isolated illnesses and outbreaks, and exposure to these sources is a significant risk-factor for infection. This edition of the rural infections series focuses on these four notifiable pathogens, each of which causes a similar set of symptoms, and discusses the investigation and management of diarrhoeal illnesses in a person with rural occupation, residence or recent contact with animals or untreated water.

Infectious diarrhoea: the common quartet of causes

Campylobacter, *Salmonella*, *Cryptosporidium* and *Giardia* represent four of the five most frequently notified illnesses in New Zealand (pertussis is the fifth).[1,2] From July to September, 2013 (the most recent surveillance period*), these four pathogens caused a total of 50 outbreaks and approximately 2880 confirmed illnesses.[2] Rural occupation, living in a rural area or contact with farm animals is a significant risk factor for contracting these infections; approximately 10% of the notified cases in the reporting period were traced to an environmental, animal or waterborne source.[2] Any illness caused by these four organisms is notifiable to the Medical Officer of Health.

Campylobacter, *Salmonella*, *Cryptosporidium* and *Giardia* cause clinically similar illnesses, typically profuse diarrhoea, abdominal pain and nausea, with or without vomiting (Table 1, over page). Bloody diarrhoea, fever, malaise and a range of other symptoms may also be present. The patient's gastrointestinal symptoms usually last less than two weeks before resolving spontaneously, however, *Cryptosporidium* and *Giardia* can cause persistent or chronic diarrhoea in some people.

Viral infections and *E. coli* (VTEC, Page 18) are also common causes of diarrhoea in all patients.

* Data for *Giardia* comes from the previous quarter (April to June, 2013) as more recent data is not available

Diagnosis and assessment of infectious diarrhoea

Patients with *Campylobacter*, *Salmonella*, *Cryptosporidium* and *Giardia* infection will typically present to primary care with diarrhoea. History and examination is generally sufficient to establish a working diagnosis and appropriate management.

Diarrhoea can be defined by duration as follows:

- Acute – lasting less than 14 days (most infections from these four pathogens will be acute)
- Persistent – lasting between 14 – 30 days (*Giardia*, and occasionally *Cryptosporidium* can cause persistent diarrhoea)
- Chronic – lasting longer than 30 days

Finding the cause of diarrhoea

The patient's description of their symptoms and recent activity may suggest a cause for their diarrhoea. Enquire about:

- The characteristics of the diarrhoea, e.g. duration, consistency, the presence of blood
- Other general symptoms, particularly abdominal pain, fever, malaise or fatigue
- The patient's occupation (to identify people working in a rural occupation, food handlers and day care workers), where they live and any recent activity that may have increased the risk of environmental exposure, e.g. camping or tramping
- Any recent contact with farm animals or wildlife

- Recent contact with or ingestion of untreated water, e.g. effluent ponds, dams or tank water
- Where, what and when the patient ate prior to their symptoms starting, particularly asking about any high-risk food, e.g. chicken or seafood ingestion
- Other general risk factors, including recent international travel, similar symptoms in household members and recent hospitalisation or antibiotic use
- Risk-factors for non-infectious or non-gastrointestinal causes of diarrhoea, including family history of coeliac or Crohn's disease, symptoms associated with certain foods, e.g. following milk ingestion, if similar symptoms have occurred previously and the presence of any non-gastrointestinal symptoms

Defining the cause of diarrhoea

If the patient reports large-volume, watery or bloody diarrhoea with diffuse abdominal pain, enteric bacterial infection is likely.[10] In a person with a rural occupation, residence or recent contact with animals, *Salmonella*, *Campylobacter*, *Cryptosporidium* and *Giardia* are among the most likely causes, but other, more general causes of diarrhoea should also be considered, which would apply to patients in any setting.

If the patient' symptoms have occurred within six hours of ingestion of potentially contaminated food, food poisoning with pre-formed bacterial toxins (i.e. toxins that are present in the food and cause symptoms in the gut, rather than the bacteria themselves) should be suspected. However, the source of infection is not always apparent, or symptoms may occur coincidentally following ingestion of high-risk foods without being related.

Clinical differentiation between viral diarrhoea and an early presentation of enteric infection is difficult. Patients who have had diarrhoea for less than two to three days may have viral gastroenteritis, e.g. infection with norovirus, rotavirus or enteric adenovirus, or this may be an early presentation of enteric infection. The longer the patient's symptoms have been present, the more likely enteric infection becomes. In addition, viral gastroenteritis is less likely if the patient has bloody diarrhoea, fever and severe abdominal pain.[10]

Verotoxin-producing *E. coli* (VTEC)

Escherichia coli are common bacteria in the human gastrointestinal flora, and are not usually pathogenic. However, overgrowth and certain strains of *E. coli* can cause severe diarrhoea. The incidence of illness related to *E. coli* in New Zealand is significantly lower than that caused by *Campylobacter*, *Salmonella*, *Cryptosporidium* or *Giardia*. Infections often have an environmental or animal source; from July to September, 2013, there were five environmental, animal or waterborne outbreaks.[2] Food-borne illness also occurs, particularly following ingestion of contaminated meat.

E. coli itself is a causative pathogen in cases of notified gastroenteritis. Gastroenteritis is a notifiable illness if there is believed to be a common source or if the person with the illness is in a high-risk category, such as a food handler. In such a case, *E. coli* will be added retrospectively as the causative organism, if identified through laboratory testing.

E. coli must also be notified if a shiga-toxin producing (also known as verotoxin-producing *E. coli* or "VTEC") strain is found to be present. Only some *E. coli* can produce these toxins and cause disease if ingested.[8] The genes required to produce these toxins are thought to have been acquired from another, more pathogenic bacteria, *Shigella dysenteriae*, via bacteriophage transfer. The shiga toxin will only cause illness in certain species. Cattle, pigs and deer can carry shiga-toxin producing bacteria without developing an illness, but when spread to humans, the toxin produces symptoms ranging from mild diarrhoea to haemorrhagic colitis.[8, 9] Most infections are caused by ingestion of food or drinking water contaminated with faeces from ruminant animals.[9]

Table 1: The natural history and presentation of the four most common notifiable infections in New Zealand[3–7]

	Salmonella enterocolitis	Campylobacter enterocolitis	Cryptosporidiosis	Giardiasis
Organism	Bacteria	Bacteria	Parasite	Parasite
Mode of transmission	Primarily food-borne, but also stagnant water, animals (particularly birds), and person-to-person	Water, animals, food and person-to-person	Animals (particularly calves and lambs), water, food and person-to-person	Water, animals and person-to-person
Seasonality	Year-round	Spring and summer	Spring	Year-round
Incidence – Cases in previous 12 months*	1118	6212	1350	1654
Incidence –five-year trend**	Variable/Stable	Decreasing	Increasing	Decreasing
Diarrhoea duration	Usually less than 14 days	Usually less than 14 days	Usually less than 14 days, potential for persistence	Variable, from less than 14 days to more than 30 days
Diarrhoea symptoms	Profuse, watery and occasionally bloody	Variable severity, watery and often bloody	Profuse and watery	Greasy, malodorous, floating stool, i.e. symptoms of malabsorption
Incubation period, average (Range)	12 – 36 hours (6 – 72 hours)	2 – 5 days (1 – 10 days)	7 days (1 – 12 days)	7 – 10 days (3 – 25 days)
Nausea and vomiting	Nausea, occasional vomiting	Nausea and vomiting	Nausea and vomiting	Nausea, rarely vomiting
Fever	Common	Common	Common	Less common
Period of infectivity to others	Typically several days to several weeks after onset of symptoms, can be up to one year in children	Several weeks after onset of symptoms	Several weeks after symptoms resolve	Up to several months after onset of symptoms

* Incidence data are from July, 2012, to June, 2013.

** Change in incidence from December, 2009, to December, 2013.

Non-infectious causes of diarrhoea should also be considered. Patients with small-volume, bloody diarrhoea, lower abdominal cramping and tenesmus (feeling as though they constantly need to defaecate or that the bowel is not completely empty following a bowel movement), may have inflammation of the bowel due to a conditions such as coeliac disease or inflammatory bowel disease. Family history and the duration of symptoms may suggest non-infective causes; however, first presentations of these illnesses can be difficult to differentiate from an acute episode of infectious diarrhoea.

Non-gastrointestinal infections can sometimes cause acute diarrhoea and should be considered when symptoms suggest another system could be involved, including urinary tract infection, pneumonia, otitis media or systemic infections. In one United States study, retrospective analysis of patients initially diagnosed with gastroenteritis found that 8% had a non-gastrointestinal systemic infection.[11] Vomiting will usually be more prominent than diarrhoea in people with these infections.[12]

Assessing for dehydration

The examination should focus on identifying dehydration. This includes basic observations, with attention to skin turgor, mucus membranes and capillary refill rate, and an abdominal examination. Although rare, other possible complications of enteric infection causing diarrhoea include reactive arthritis and Guillain-Barré syndrome.[10]

Laboratory investigation

Laboratory investigation is not routinely required for patients with acute diarrhoea.

However, in a patient with a rural occupation, residence or recent exposure to animals, laboratory investigation is recommended to provide additional information to guide treatment and for notification purposes.

What tests should be requested for a patient with rural risk-factors?

In people with diarrhoea who live or work in a rural setting or with recent exposure to animals or untreated water sources, request: [13]

- Faecal culture and microscopy
- Faecal *Giardia* and *Cryptosporidium* antigen tests

Note the patient's relevant risk factors, e.g. rural occupation, on the laboratory request form, as the test may be declined by the laboratory if justification for testing is not recorded.

Only one stool sample should be sent for analysis. However, faeces are not homogenous; bacteria may not be evenly distributed within the sample and the volume of excreted bacterial material varies with the stage of infection. As a result, false-negatives can occur, particularly with faecal culture. A repeat test may be justified if a particular pathogen is strongly suspected and the initial test is negative and the patient has ongoing symptoms.[14] In this situation, discussing the patient with an Infectious Diseases Physician or Clinical Microbiologist should be considered.[14]

Faecal culture and microscopy

The faecal culture and microscopy test is used to assess a patient's stool for leucocytes, indicating inflammation of the bowel either due to an invasive pathogen or other inflammatory bowel disease and isolation of pathogenic bacteria. This is the first-line test for the investigation of infectious diarrhoea in someone with risk factors. It can identify *Campylobacter*, *Salmonella*, *Yersinia*, *E. coli* (VTEC) and *Shigella*.

Ask the patient to provide a faecal sample in a sterile collection container. The sample should be stored at room temperature and should not have a fixative applied to it. The sample should be transferred to the laboratory as soon as possible. If transfer will be delayed by more than 24 hours, refrigerate the sample and consult the collecting laboratory.

Giardia and Cryptosporidium testing

The *Giardia* and *Cryptosporidium* antigen test is an immunoassay test that identifies the presence of antigens from *Giardia* and *Cryptosporidium* in a patient's stool. The results will be reported as either positive or negative, i.e. antigens are present or absent.

As with faecal culture, an antigen test requires a fresh faecal sample. The same sample used for bacterial culture is used for the antigen test.

The antigen test has high sensitivity and specificity.[15]

Other tests are available, but less commonly used

Microscopy can be used to detect *Giardia* and *Cryptosporidium*, in samples from patients with acute diarrhoea, however, it is not routinely recommended. Microscopy is an alternative,

second-line option in a patient with a negative antigen sample but ongoing symptoms. Microscopy requires a high degree of technician skill, has a slower turnaround time and requires up to three serial samples in order to provide sufficient sensitivity, which decreases patient compliance and increases the cost of the test.

Other methods of detecting *Giardia* and *Cryptosporidium* such as microbial stains and enzyme immunoassays are available in some laboratories, but they are not routinely recommended. Stains require a different sample, with a fixative applied to the stool; this type of test would generally only be requested in consultation with an Infectious Diseases Specialist.

It has been suggested that real-time polymerase chain reaction (PCR) will eventually become the standard test for investigating infectious diarrhoea, due to a faster turn-around time, and may replace antigen testing.[16] At present PCR is not routinely available for investigating infectious diarrhoea.

Management of infectious diarrhoea

While awaiting laboratory results, management of the patient is similar to a standard case of gastroenteritis unless there is a strong clinical suspicion of a particular organism, e.g. confirmed household contacts. The focus of management is preventing or treating dehydration, and reassuring the patient that diarrhoeal illnesses are usually self-limiting.[17]

When should a patient be referred to hospital?

Most patients with infectious diarrhoea can be managed at home. However, referral to hospital should be considered for patients with persistent vomiting who are unable to retain oral fluids or who have severe dehydration. Referral should be considered for older patients who are unable to manage at home by themselves or younger children whose condition may deteriorate rapidly.[12] Some practices, particularly rural practices, may be equipped to manage patients with dehydration with IV fluids and monitoring, rather than referring to hospital.

For further information, see: "Community-based IV administration: primary care reducing hospital admissions", BPJ 38 (Sep, 2011).

The Laboratory Test Schedule

In October, 2013, the Laboratory Test Schedule and accompanying referral guidelines were released. DHBs can implement the guideline individually. All available laboratory tests have been categorised into two groups, termed Tier One and Tier Two. Tier One tests can be ordered by any clinician, Tier Two tests are limited to a list of named specialists. Guidelines for the appropriate ordering of selected tests were also developed.

Both faecal culture and *Giardia* and *Cryptosporidium* antigen test are funded Tier One tests in the Laboratory Schedule.

For further information, see: "The New Zealand laboratory test schedule and guidelines: What does it mean for general practice?", Best Tests (Nov, 2013).

Monitoring and preventing infectious disease in New Zealand

Notification and surveillance are key components of managing and preventing communicable illnesses in New Zealand. The data gathered from these activities guides the direction and scope of local and national public health efforts and campaigns.

The list of notifiable diseases was set out in the Health Act 1956, and is available from the Ministry of Health website. Illnesses are added to the list if they are deemed important to public health, e.g. avian influenza (H7N9) and Middle East Respiratory Syndrome (MERS) were added to the list in 2013.

All notifiable illnesses must be reported to the Medical Officer of Health once there is a reasonable clinical suspicion of the illness or confirmation through testing. Some illnesses, termed "Section A illnesses", must also be reported to the local health authority, e.g. the PHO or DHB.

Campylobacter, *Giardia*, *Salmonella* and *Cryptosporidium* are all Section A infectious illnesses. Laboratory testing is required to confirm the illness for notification. Both culture or antigen testing are sufficiently accurate for notification purposes.

In addition to the standard clinical tests performed for diagnosis, additional testing may be performed by the laboratory to provide better surveillance data for some notifiable illnesses. For example, *Campylobacter* bacteria identified via culture may undergo multilocus sequence typing in order to provide epidemiological data.[1] This information is not routinely provided to practitioners.

For further information, see: **www.health.govt.nz/our-work/diseases-and-conditions/notifiable-diseases**

Rehydration and preventing further fluid loss

Infants and children without signs of clinical dehydration, should continue breast feeding and other milk feeds as normal. Older children should be encouraged to drink regularly, in small amounts.[18] Oral rehydration solution can be offered as a supplemental fluid.[18] Oral rehydration solutions can be made at home (see recipe below) or prescribed, fully-subsidised. Drinking undiluted fruit juices or carbonated drinks should be discouraged,[18] as they contain high levels of sugar, and can increase dehydration through diuretic action and by altering the osmolality of the gut.

In infants and young children who are dehydrated, oral rehydration solution is recommended.[18] Chilling the oral rehydration solution (or freezing into ice blocks) can improve palatability. Fluids should be offered in regular, small amounts to help avoid vomiting. Replacement with 50 mL/kg over four hours is recommended.[18]

In adults with a diarrhoeal illness, oral rehydration solutions are not usually required. However, patients should be advised to increase oral fluid-intake to two litres per day, with fluids such as water or salty soups. As with children, adults should avoid sugary or caffeinated drinks, e.g. sports drinks. Advise patients to eat normally when they feel they are able; bland foods may be more palatable initially.

A recipe for oral rehydration fluid is:

- 1 litre of water
- 8 teaspoons of sugar
- 1 teaspoon of salt

Stir until dissolved and store in the refrigerator. The solution should be discarded after 24 hours.

For further information on the management of dehydration in people with gastroenteritis, see: "Assessment and management of infectious gastroenteritis", BPJ 25 (Dec, 2009).

Pharmacological management

Antibiotics are not recommended for people with acute diarrhoea of unknown pathology.[12]

Antibiotic treatment may be indicated for adults or children if a specific pathogen is identified by laboratory investigation

(Table 2, over page). Antibiotics are required for patients with giardiasis and symptomatic contacts of the patient. Antibiotic treatment may be appropriate in some patients with salmonella enterocolitis and campylobacter enterocolitis, depending on their risk-factors. *Cryptosporidium* is not treated with antibiotics, as they are not effective.

Antidiarrhoeal medicines are not routinely recommended and should not be used if the patient has blood or mucus in their stool.[12] In people with diarrhoea containing blood or mucus antidiarrhoeal medicines increase the risk of toxic megacolon and prolong duration of diarrhoea.[19] If an antidiarrhoeal is required for symptomatic relief in a patient without blood or mucus in their diarrhoea, loperamide can be considered.[12] Loperamide should be given at 4 mg initially, with 2 mg after each loose stool, up to a maximum of 16 mg in 24 hours.

Antiemetics are not routinely recommended.[12]

If pain relief is required, paracetamol can be given. NSAIDs should be avoided in people with dehydration or the potential for dehydration due to the risk of kidney injury.

The patient's current medicine use should also be reviewed, as certain medicines may worsen diarrhoea (e.g. laxatives), increase the risk of complications from the diarrhoea (e.g. diuretics, NSAIDs) or can be affected by diarrhoeal symptoms (e.g. reduced absorption of oral contraceptives).

Lactose intolerance or irritable bowel syndrome following infection

Secondary, or acquired, lactose intolerance can occur following any gastrointestinal illness that affects the gut mucosa. It is particularly common in adults following *Cryptosporidium* or *Giardia* infection and in children following any enteric infection.

Symptoms of lactose intolerance, shortly after consuming lactose, include:

- Diarrhoea
- Abdominal pain and distension
- Flatulence
- Dyspepsia

If the patient's diarrhoea continues following antibiotic treatment or begins again soon after symptoms cease consider lactose intolerance. A lactose challenge can be undertaken: instruct the patient to trial a lactose-free diet for two weeks, then reintroduce these foods, and report any symptoms that occur. All food containing lactose needs to be removed during the challenge, so food labels should be closely assessed. Many foods and some medicines contain unexpected lactose, such as instant soups, muesli bars and some processed meat.

Secondary lactose intolerance following enteric infection is usually transitory, but may persist for several weeks.[21] It can be managed with dietary restriction followed by gradual reintroduction of milk.

Irritable bowel syndrome may also develop following a significant enteric infection. Symptoms and signs will be similar to lactose intolerance; however, a lactose challenge will usually be negative. Irritable bowel syndrome may be short-term or may persist for several years. Management usually involves reassurance, stress management, lifestyle and diet changes and, in some patients, medicines such as loperamide and mebeverine.

For further information on lactose intolerance, see: "Investigating the gut: Lactose intolerance", Best Tests (Mar, 2010).

For further information on irritable bowel syndrome, see: "Irritable bowel syndrome in adults: not just a gut feeling", BPJ 58 (Jan, 2014).

Management of contacts

Symptomatic contacts should be managed based on their risk-factors and the severity of their illness.[3–6] For example, if they are a food handler or have a rural occupation, investigation may be required. If no risk-factors are present, investigation is not routinely recommended. A "probable" notification can be made based on contact with a confirmed case, without laboratory, testing if necessary.

Asymptomatic household contacts of people with salmonella enterocolitis, who are food handlers, should have a faecal culture and microscopy test requested to confirm they are not infected and can safely attend work (See Table 1 for incubation times).[4] Investigation, restriction from school or work and empiric treatment are not required for other asymptomatic contacts of people with notifiable infectious diarrhoea, although they should be made aware that if they develop symptoms, they need to present to primary care and will require assessment and potentially testing.[3–6]

Table 2: The management of four common causes of infectious diarrhoea [3,12,20]

Infection	When to treat with antibiotics	First-line	Comment
Salmonella enterocolitis	Treat patients with severe disease,* who are immunocompromised or who have cardiac valve disease or endovascular abnormalities, including prosthetic vascular grafts	Ciprofloxacin, 500 mg, twice daily, for three days Co-trimoxazole, 160 + 800 mg, twice daily, for three days is an alternative	Treatment may prolong excretion For children, discuss appropriate treatment with an Infectious Diseases specialist
Campylobacter enterocolitis	Treat if severe,* symptoms present for more than one week, women who are pregnant nearing term or people who are immunocompromised. Treatment may also be appropriate for food handlers, child care workers or people caring for immunocompromised people.	Erythromycin ethyl succinate 400 mg (child 10 mg/kg), four times daily, for five days** A second-line alternative for adults is ciprofloxacin, 500 mg, twice daily, for five days	Treatment has limited effect on symptoms, but may reduce stool carriage
Cryptosporidiosis	Antibiotics are not effective		Discussion with an Infectious Disease specialist is recommended for patients who are immunocompromised or have co-morbidities
Giardiasis	Antibiotic treatment is recommended if laboratory tests show infection is present (and for symptomatic contacts)	Children < 35 kg – ornidazole 125 mg/3 kg, once daily, for one to two days Adults and children > 35 kg – ornidazole 1.5 g, once daily, for one to two days Metronidazole can also be used first-line. Children – 30 mg/kg, once daily, for three days, to a maximum of 2 g per day. Adults – 2 g, once daily, for three days	If treatment with ornidazole appears to be ineffective, exclude re-infection from asymptomatic contacts; lower doses of metronidazole may be given for longer periods, e.g. 10 mg/kg/dose, three times daily for children or 400 mg, three times days, for seven days. An interval of two to three days between treatments is recommended. Ornidazole is only available in tablet form, which may be crushed. A child dose is equivalent to one-quarter of a tablet per 3 kg.

* High fever, bloody diarrhoea or more than eight stools per day[12]

** Erythromycin 800 mg, twice daily, can be considered for patients where adherence is likely to be an issue

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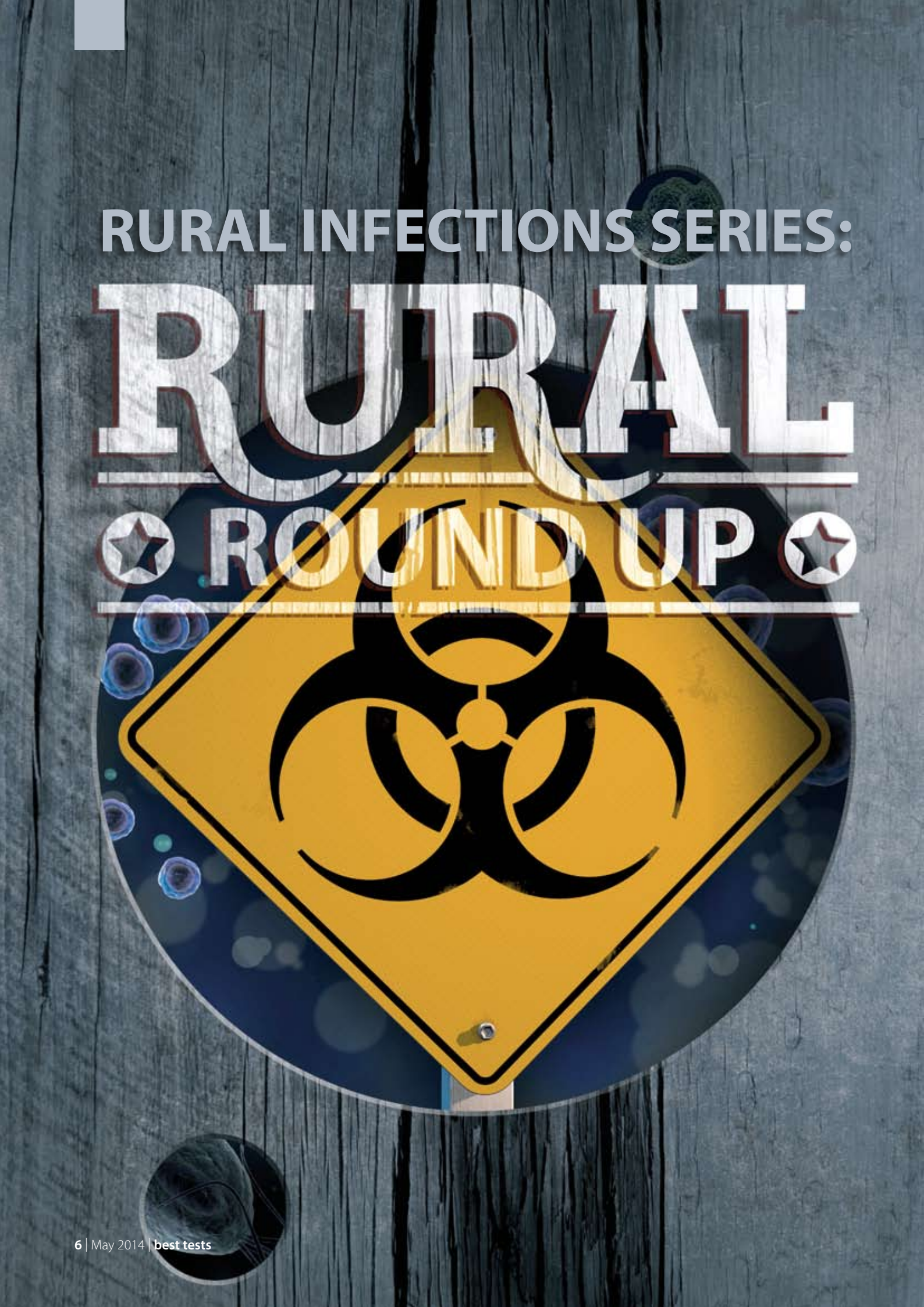
RURAL INFECTIONS SERIES:



In the final instalment of the rural series we present a round-up of infections that may be seen in patients living in, working in or visiting a rural environment. Most of these infections will be rarely encountered, but it is useful to be aware of their features and recommended management.

People who live, work or undertake recreational activities in a rural, agricultural or horticultural setting, are potentially exposed to a large number of infectious pathogens that can cause disease. Individually, most of these infections are rare, but the possibility of a rurally-acquired infection should be considered in symptomatic patients who have been exposed to this setting.

Many infections that were once prevalent in rural New Zealand have now been eliminated, e.g. hydatid parasites and brucellosis. However, some infections, e.g. leptospirosis, orf and *Listeria*, are still occasionally seen in rural communities.

Leptospirosis, campylobacter enterocolitis, salmonella enterocolitis, cryptosporidiosis and giardiasis are the most common rurally-acquired infections in New Zealand; these have been covered in previous articles in the rural infections series.

To round up the list of other rural infections, we have categorised them by their primary risk factors, which are:

- Consumption of unprocessed foods and untreated water
- Exposure to animals
- Exposure to plants or soil

N.B. Many of these infections have more than one contributing cause, and some are not unique to the rural environment.

Infections acquired via consumption of unprocessed foods or untreated water

Many people living in a rural community do not have access to a reticulated water supply, and collect and store their own water for household use. A rural lifestyle also often involves raising, growing and gathering food, e.g. raw milk, home-butchered or recreationally-caught meat and seafood. These practices are all associated with an increased risk of infectious diseases.

Drinking unpasteurised (raw) milk

Drinking milk "straight from the cow" is a way of life for many people living or working on a farm. The consumption of raw milk products is also gaining popularity in the wider community. However, although regarded as "wholesome" or "healthy", drinking raw milk actually increases a person's risk of illness.

Milk from cows, goats and sheep can be contaminated with bacteria, such as *Campylobacter jejuni*, *Escherichia coli*, *Listeria monocytogenes*, *Mycobacterium bovis*, *Salmonella enteritidis*, *Shigella spp*. and *Yersinia enterocolitica*. Pathogens can pass into milk directly via an infection in the animal, e.g. mastitis in the udder, or indirectly from the farm environment during the milking process, e.g. faecal contamination.[1] Commercially produced milk is pasteurised to destroy these bacteria. Pasteurisation is a heat treatment process which usually involves milk being rapidly heated to 72°C for 15 seconds.

There have been several small outbreaks of infectious diarrhoea associated with raw milk consumption in New Zealand in recent years.[1] The Ministry for Primary Industries monitors dairy products in New Zealand; an ongoing survey has found *Listeria monocytogenes*, Shiga-toxin producing *E. coli* and *Campylobacter jejuni* in raw milk.[1] In the United States, the Centers for Disease Control and Prevention (CDC) states that "the consumption of non-pasteurised dairy products cannot be considered safe under any circumstances".[2]

Facts about pasteurised milk:[1, 3]

- Pasteurisation is a highly reliable method for eliminating pathogens in milk
- Pasteurisation has a minimal effect on the fat and protein composition of milk
- Pasteurisation does not affect mineral content, stability or gastric absorption of milk
- Riboflavin, vitamin B6 and B12 are reasonably heat stable so remain in pasteurised milk at high levels
- Pasteurisation reduces the vitamin C content in milk by approximately 10%, however, milk is not a significant dietary source of vitamin C
- Some enzymes in milk are inactivated during the pasteurisation process but these are not thought to be important for human health

It is recommended that:[1]

- Raw milk products should not be consumed by young children, elderly people, pregnant women or people who are immunocompromised
- If raw milk is consumed, ensure it is from a source where good hygiene practices are adhered to during milking and storage (this reduces, but does not eliminate, the risk of contamination)
- Refrigerate raw milk at ≤ 4 ° C (this will not eliminate *Listeria* – see below)
- Discard raw milk if it has been at room temperature for more than two hours
- If diarrhoea develops after ingestion of raw milk, consider the possibility of an infectious pathogen as the cause

For further information on *Salmonella*, *Campylobacter* and *E. coli*, which can all be contaminants in unpasteurised milk, see: "Rural infections series: Investigating and managing people with diarrhoea", Best Tests (Feb, 2014). For information on *Listeria*, also a milk contaminant, see below.

A focus on *Listeria*

Listeria monocytogenes is a foodborne pathogen found in unpasteurised milk or unpasteurised milk products (e.g. cheeses), and also in items such as processed meat products (e.g. salami, paté), cold pre-cooked meats, uncooked seafood and raw vegetables, e.g. stored salads. *L. monocytogenes* can survive and multiply in food items at standard refrigeration temperatures.[4] People may also be exposed to *L. monocytogenes* via contact with potentially infective farm material, such as aborted animal foetuses.[4]

Listeriosis, the illness caused by *L. monocytogenes*, is characterised by diarrhoea, nausea, vomiting, fever, myalgia and fatigue, which typically resolve within one to three days.[5] More severe complications, such as the development of septicaemia or meningoencephalitis, are more likely to occur in vulnerable groups, such as pregnant women, young infants, elderly adults and immunocompromised people. Listeriosis also causes risks to a pregnancy, including miscarriage, premature labour and stillbirth. *Listeria* infection can be transferred to an infant during childbirth, which can result in serious illness and death for the infant.[6] There are approximately 25 notified cases of listeriosis per year in New Zealand (see: "Listeriosis in New Zealand", next page).[4]

Listeriosis is often an unexpected diagnosis and rarely considered before being identified by laboratory testing. The time between exposure and onset of symptoms is variable, with cases being reported between 1 – 70 days after exposure to a contaminated food.[4, 5] It is estimated that the median incubation period of *Listeria* is three weeks.[4] In practice it will be difficult to differentiate listeriosis from other diarrhoeal illnesses caused by pathogens, such as *Giardia*, *Salmonella*, *Campylobacter* and *E. coli*. Laboratory investigation is recommended in patients presenting with persistent diarrhoea and risk factors, e.g. exposure to a rural environment. It can be important to ask people their occupation when they present with persistent diarrhoea as they may live in an urban area, but work in a rural/agricultural environment.

If listeriosis is suspected (e.g. risk factors present and other likely pathogens have been ruled out), this can be discussed with an Infectious Diseases Specialist or Clinical Microbiologist. The best test for *L. monocytogenes* is blood culture; stool culture for *Listeria* is not routinely performed. Listeriosis is a notifiable disease and cases (suspected or confirmed) must be notified to the local Medical Officer of Health.[4]

Management of listeriosis is usually in conjunction with an Infectious Diseases Specialist. Depending on the clinical situation, patients with listeriosis may be managed at home if their signs and symptoms are mild. Patients with severe signs and symptoms, and those most at risk of serious illness are managed in a hospital setting.[4] Antibiotics may be considered for symptomatic and asymptomatic people who are at high risk of complications (e.g. infants, pregnant women, elderly adults, immunocompromised people), if they are known to have ingested a food implicated in an outbreak.[5] Listeriosis is treated with amoxicillin 1 g, three times daily, for 10 – 14 days.[7] Co-trimoxazole is an alternative.[5] Other antibiotic choices for treatment may be considered in a hospital setting.[6]

Patients with listeriosis can remain infectious to others for several months after resolution of symptoms,[4] however, other than transplacental transmission (mother to foetus), there are few, if any, reported cases resulting from person to person transmission.

Eating home-kill and recreational catch meat

In the rural community, many families will consume meat which has been butchered on the farm (home-kill) or hunted (recreational catch). As these methods are not subject to any hygiene or safety regulations, there is a potential for transmission of infectious diseases and toxicity via handling or ingestion of raw or under-cooked meat.

The main risks are:[9]

- Bacterial contamination from the animal via external wounds or contents of the gut or other infected organs
- Bacterial contamination from the environment, e.g. soil, grass, hunting knife
- Chemical contamination via the animal eating pest control poisons or carcasses of poisoned animals, or if transporting the carcass in a vehicle used to carry chemicals, e.g. weed killer or fuel

Bacterial contaminants in home-kill and recreational catch meats include *Salmonella* (particularly birds), *Campylobacter*, *Cryptosporidium* (particularly calves and lambs), *Giardia* and, rarely *Trichinella* (particularly pigs – see over page).

The Ministry for Primary Industries has guidelines on safe practices for home-kill meat. A consumer information brochure can be found here: **www.foodsafety.govt.nz/elibrary/consumer/Homekill-brochure-2012-web.pdf**

And further information found here: **www.foodsmart.govt.nz/food-safety/hunting-collecting-fishing/**

Listeriosis in New Zealand

In New Zealand, epidemiological data on listeriosis is collected by the Institute of Environmental Science and Research Ltd (ESR). In 2012 (latest reported data) there were 25 notified cases of listeriosis (0.6 per 100 000 population). Two of these cases were perinatal, which resulted in death of the foetus. Of the remaining cases most were in people aged 50 years and over (21 cases). The majority (16 cases) also had an underlying co-morbidity, and four cases resulted in death. The 25 notified cases were from nine DHBs, including five from Counties Manukau, five from Bay of Plenty and four from Hawke's Bay. There was one outbreak of listeriosis reported in 2012, linked to an infected ready-to-eat meat product. The notification rate of listeriosis has been relatively stable over the past 15 years, following a peak of cases in 1997 (0.9 per 100 000 population).[8] It is likely that the actual rate of *Listeria* infection in the population is higher than the notified rate, taking into account cases of sub-clinical or mild infection which are not reported.

A focus on Trichinella

Trichinella spiralis is a parasitic round worm that can be found in carnivorous animals, such as feral cats and rats. There have been historical cases of infection among the domestic pig population in New Zealand, from pigs eating carcasses and faeces of infected animals.[10] However, the risk of *T. spirialis* in commercial piggeries in New Zealand is now regarded as very low. Although extremely rare (only three notifications since 1988),[8] infection in humans can occur after ingestion of raw or under-cooked meat, i.e. pork, that contains encysted *Trichinella* larvae. *Trichinella* cannot be transmitted from human to human.[10]

Trichinella can be destroyed by cooking meat until it reaches an internal temperature of ≥ 60°C for at least one minute, or by freezing meat at ≤ −15°C (standard home freezer temperature) for at least 20 days. Curing, salting, smoking or microwave cooking will not destroy *Trichinella*.[10]

Trichinellosis, the illness caused by *T. spiralis*, typically begins one to two days after ingestion of infected meat, with general discomfort, abdominal pain and diarrhoea, lasting up to one week. Headache, fever and excessive sweating may develop three to four days after ingestion. Further systemic features may occur within 8 – 15 days after ingestion (range 5 – 45 days), such as facial oedema (usually periorbital), myalgia (most commonly affecting the trunk and limbs) and severe weakness.[10, 11] Patients with trichinellosis almost always have eosinophilia, which can persist for several weeks to months.[11] Other characteristic laboratory parameters include increased muscle enzymes and increased total IgE. Differential diagnoses of trichinellosis include influenza, infectious diarrhoea and auto-immune disease.[11]

Patients with suspected trichinellosis should be referred to an Infectious Diseases Specialist. Trichinellosis is confirmed by a positive serological test or detection of larvae in muscle tissue biopsy. Treatment usually involves an anthelmintic (e.g. mebendazole), analgesics, corticosteroids and supportive care.[10, 11] Trichinellosis is a notifiable disease so all cases, suspected or confirmed, should be notified to the local Medical Officer of Health.

For further information about trichinellosis, see: FAO/WHO/OIE Guidelines for the surveillance, management, prevention and control of trichinellosis. Available from: **www.trichinellosis.org/uploads/FAO-WHO-OIE_Guidelines.pdf**

Drinking tank water

Using collecting tanks or a natural ground water source for household water supply is common in rural communities in New Zealand. Depending on the source of the collected water, e.g. stream, bore, rainwater, and the household storage and filtering system used, contamination with infectious pathogens, heavy metals, trace elements and agricultural chemicals is possible.

Blastocystis: unknown role in infection

Blastocystis is a protozoan parasite which can be found in the gastrointestinal tract of many animals. Humans may acquire infection from animals (particularly from cattle, pigs or birds) or from person-to-person oral-faecal contact. Whether blastocystis is a cause of human disease is very uncertain. Some people found to have stool carriage of blastocystis are asymptomatic, whereas some have diarrhoea and other gastrointestinal symptoms. It is thought that people who are immunocompromised may be more susceptible to infection.[16] Most mild symptomatic cases are self-limiting; no specific treatment is required. However, in rare cases, gastrointestinal symptoms may be persistent. In these cases, other pathogens, e.g. *Giardia*, should first be ruled out as a cause for the symptoms. If the symptoms appear to be attributable to blastocystis, a course of metronidazole may be trialled. There has been mixed evidence of the success of metronidazole in eradicating infection. If treatment with metronidazole has failed, or is contraindicated, co-trimoxazole is a second-line option.[16]

Human or animal waste is the most likely source of pathogenic micro-organisms in water supplies. Bacteria are also found naturally in ground water and surface water.[12]

Drinking water may be contaminated from seepage from a septic tank, run-off from pastures, heavy rains causing overflowing storm water, animal faeces (e.g. on a roof used for collecting rainwater), or improperly sealed storage tanks or wells.[12]

E. coli is one of the most common infectious pathogens in collected water and is used as a marker of faecal contamination. *Cryptosporidium*, *Giardia*, *Campylobacter*, *Salmonella* and *Shigella* are also common contaminants. Other micro-organisms found in water include helminths (thread worms, tape worms, nematodes) and viruses, such as norovirus, rotaviruses and hepatitis A.[12] These organisms can be found in faecal waste of humans and animals (e.g. pigs, deer, sheep, cows, birds, possums) and also in raw milk.[12] Most of these pathogens cause gastrointestinal illness, and the most susceptible groups are young infants, elderly adults and people who are immunocompromised. In some cases, people who have a prolonged exposure to a pathogen can develop immunity to it. Therefore members of a household with a contaminated water supply may not display and signs and symptoms, but visitors drinking the contaminated supply may become ill.[12]

If a patient presents with persistent diarrhoea and has a history of drinking from a tank water supply, testing for infectious pathogens would be indicated. A faecal sample should be sent for culture (which tests for *Campylobacter*, *Salmonella*, *Yersinia*, *E. coli* (VTEC) and *Shigella*) and antigen testing for *Giardia* and *Cryptosporidium*. Note risk factors and relevant clinical details on the laboratory request form.

It is recommended that home water supplies are frequently tested for *E. coli* (also called faecal coliforms) to monitor faecal contamination. At home kits are available or a sample can be sent to a commercial laboratory. An effective water filtering system, e.g. a UV filter, will help to minimise risk.

For further information on managing diarrhoea in a rural population, see: "Rural infections series: Investigating and managing people with diarrhoea", Best Tests (Feb, 2014).

For further information about drinking water guidelines, see: **www.health.govt.nz/our-work/environmental-health/drinking-water**

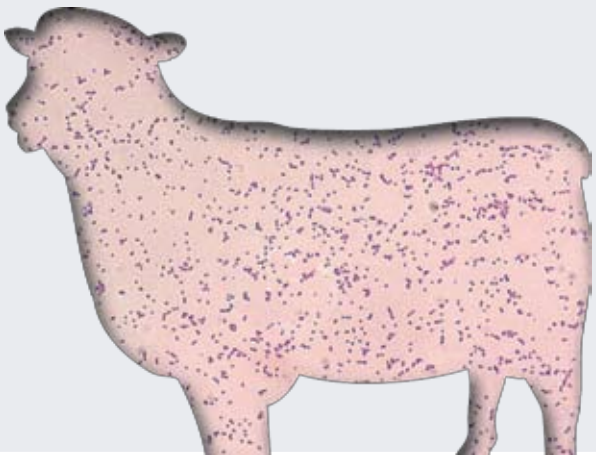
Brucellosis: once endemic in New Zealand but now rare

Brucellosis is a granulomatous infectious disease caused by the ingestion of *Brucella* bacteria in raw milk or meat from infected animals, or through contact with animal faeces or carcasses. Most cases of brucellosis in humans are caused by *B. melitensis*, but *B. abortus*, *B. suis* and *B. canis* can also cause human illness.[13]

Brucellosis is a notifiable disease and between 1997 and 2012, 13 cases were reported in New Zealand.[8] However, these patients are presumed to have acquired the infection in other countries because the only *Brucella* species that remains in New Zealand is *B. ovis*, which infects sheep, but is not pathogenic to humans. *B. abortus* was once endemic in cattle in New Zealand but was eradicated by 1996; since then, there has been no evidence of locally-acquired brucellosis in humans.[14]

People with brucellosis usually present with acute febrile illness, general malaise and respiratory tract symptoms.[15] "Drenching", malodorous perspiration is a characteristic feature.[13] Physical examination is generally nonspecific, however, lymphadenopathy, hepatomegaly or splenomegaly may be present.[13] If untreated, complications can include granulomatous hepatitis, arthritis, spondylitis, anaemia, thrombocytopenia, meningitis, uveitis, optic neuritis, endocarditis and neurological disorders collectively known as neurobrucellosis.[13]

Patients with suspected brucellosis should be referred to an Infectious Diseases Specialist. Laboratory confirmation of brucellosis involves serological testing and culture.



Infections acquired via contact with animals

People with agricultural occupations, such as farmers, dairy workers and meat processors, and people who live on farms, are exposed to a large number of infectious pathogens via contact with animals. For example, leptospirosis, which passes from mammals, such as pigs and cattle, to humans, is the most common occupationally acquired infectious disease in New Zealand.[17]

Animal-to-human contact is associated with respiratory infections, such as tuberculosis, and skin infections, such as pox viruses, dermatophyte and erysipeloid infections and granulomas.

For further information about leptospirosis, see: "Rural infections series: Leptospirosis", Best Tests (Nov, 2013).

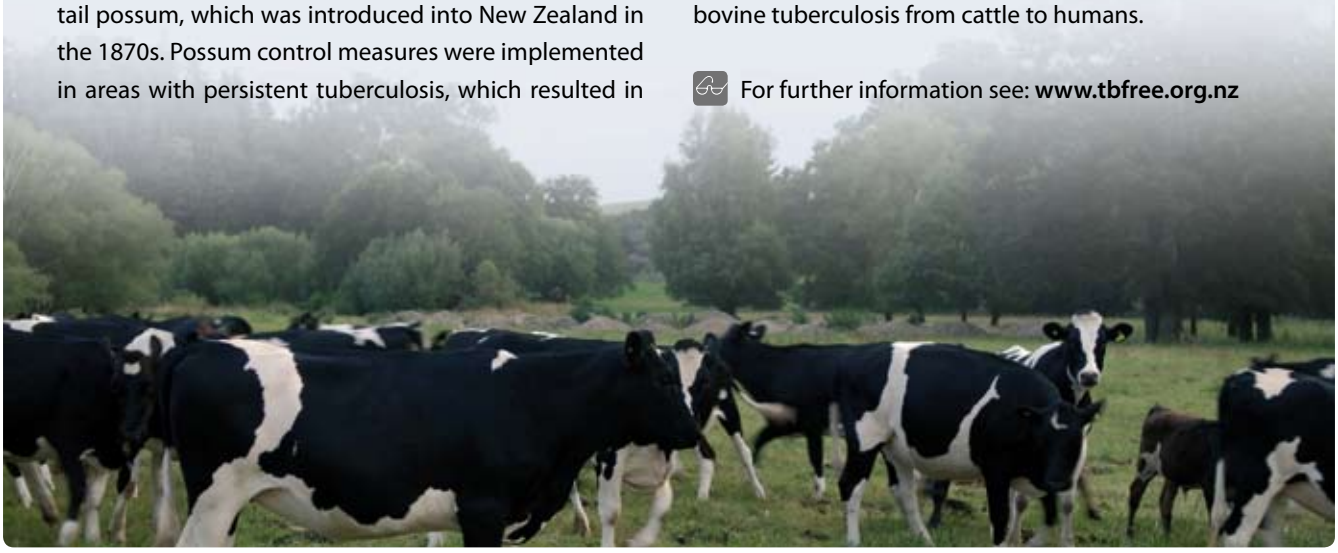
Tuberculosis

In 2013 there were 278 cases of tuberculosis in New Zealand.[18] Tuberculosis is now mostly seen in immigrants and seasonal workers. *Mycobacterium tuberculosis* is the typical bacteria associated with tuberculosis, and is transmitted from human-to-human. Atypical infections with other *Mycobacterium* species also occur. There are multiple causative species, but the most common are *M. kansasii* and *M. avium-intraceullulare*, which can be found in water, milk, bird excrement, soil and house dust. Atypical mycobacterial infections are more commonly seen in children, often presenting as an inflammation of the lymph nodes. Rarely, *M. bovis* (bovine tuberculosis) can be transmitted from infected animals (cattle, deer, possums and ferrets) to humans via handling or ingestion of contaminated animal products, including raw milk, or by airborne droplet spread to people who work closely with animals.[19]

Bovine tuberculosis in New Zealand livestock

It is thought that bovine tuberculosis was first established in New Zealand in the 1800s when cattle and deer were introduced. Control measures were implemented in the mid 1900s and by the 1970s all cattle herds were undergoing regular testing for tuberculosis and post-mortem inspection for disease. Bovine tuberculosis was eradicated in several regions, but there was unexplained disease in some areas, such as the West Coast of the South Island. It was found that livestock were being infected via the Australian brush-tail possum, which was introduced into New Zealand in the 1870s. Possum control measures were implemented in areas with persistent tuberculosis, which resulted in significant declines in livestock infections. When possum control measures were later relaxed in the 1980s, bovine tuberculosis returned, peaking in the mid-1990s at rates much higher than in other developed countries. In the past decade, renewed efforts to control bovine tuberculosis and cooperation between herd owners have resulted in levels which are at an all-time low. It is hoped that in the near future, New Zealand cattle herds will become "TB-free". There have been no reported cases in New Zealand in recent years of transmission of bovine tuberculosis from cattle to humans.

For further information see: **www.tbfree.org.nz**



Symptoms of tuberculosis are dependent on the organ system involved, e.g. pulmonary, intestinal, bone, lymphatic system. Pulmonary symptoms are most common, including dry cough which becomes productive, haemoptysis, pleuritic chest pain and breathlessness, along with anorexia, fatigue, fever and night sweats.[19]

Patients with suspected tuberculosis should be discussed with an Infectious Diseases Specialist. Chest x-ray and sputum culture are usually the initial tests. Further testing, e.g. QuantiFERON Gold assay, may also be required. Tuberculosis is a notifiable disease so all suspected or confirmed cases must be notified to the local Medical Officer of Health.

Combination antibiotic treatment is required for up to one year, or longer in some cases.[19] Tuberculosis can remain latent for many years, and in some cases reactivation may occur years after the original exposure.[19] People with active pulmonary tuberculosis are infective to others for several months to years.[19]

For further information see: "The guidelines for tuberculosis control in New Zealand", available from: **www.health.govt.nz**

Orf

Orf, also referred to as contagious ecthyma, contagious pustular dermatitis or scabby mouth, is a virus that commonly affects sheep (usually lambs) and goats, that can be transferred to humans.[20] It is caused by the parapoxvirus orf virus.[20] Other livestock, such as deer and cattle, are affected by similar poxviruses (see: "Milker's nodules"). Although orf can be a life-threatening disease in sheep and goats, it is a relatively mild and self-limiting condition in humans.

Orf is most frequently seen in farmers, shearers, meat processors, veterinarians and people bottle-feeding lambs.[21] Orf is characterised by the development of a 2 – 3 cm tender, flat-topped, red-to-blue papule or pustule on the dorsum of the index finger or hand (less commonly on the forearm or face), approximately one week after contact with an infected animal (Figures 1 and 2).[20, 21] The lesion will eventually crust over and resolve within two months. Usually only one lesion develops, but in some cases there may be multiple lesions.[21] Lymphadenopathy may be present, along with red streaks marking the lymph channels.[21] In some cases, patients may develop erythema multiforme, which is a secondary rash on distal limbs, characterised by target lesions with central blistering. The rash may persist for two to three weeks. Orf lesions may be more progressive and destructive in patients who are immunocompromised.

Orf can be diagnosed based on the appearance of the lesion and a history of contact with animals; laboratory investigation is not usually required. Standard microbiology culture will be negative. Skin biopsy typically shows ballooning of keratinocytes, necrosis and inclusion bodies.

No specific treatment is indicated, unless secondary bacterial infection is present; staphylococcal infection is most likely, which would be treated with flucloxacillin or cephalexin (see New Zealand Formulary or bpac^{nz} antibiotic guide for further details). Lesions can be covered to prevent cross-contamination. Patients with large lesions may require shave

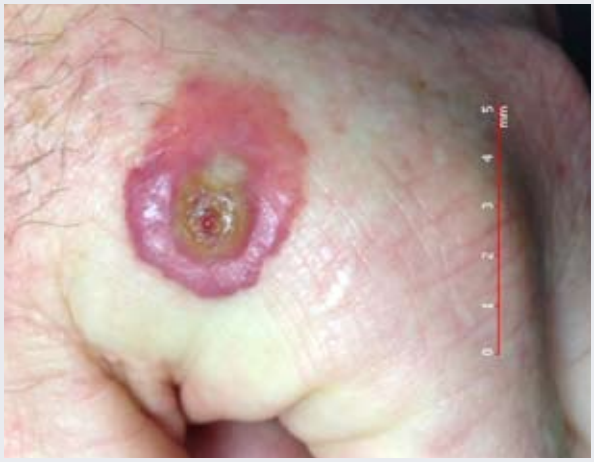


Figure 1: Typical orf lesion. Image provided by DermNet NZ (Courtesy of Dr Bert Rauber)

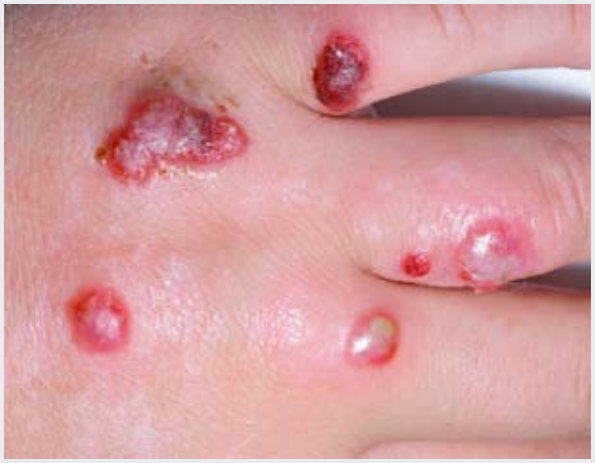


Figure 2: Multiple orf lesions. Image provided by DermNet NZ

excision, and should be referred to a Dermatologist.[21] There is some evidence that imiquimod cream is effective in treating orf,[22] however, this is an off-label use of this medicine and would not meet Special Authority criteria for subsidy.

For further information on orf and other parapox viruses, see Dermnet: **www.dermnetnz.org**

Milker's nodules

Milker's nodules are caused by a parapox virus that affects cattle. Infection is carried on the teats or in the mouth of cows ("ring sores") and can be passed to humans while milking or examining the animal.[23] It is sometimes referred to as "cowpock" and is often confused with cowpox, which is a viral skin infection caused by the vaccina-type cowpox virus (part of the family of viruses that also includes smallpox).[24] Cowpox is extremely rare and unlikely to be seen in New Zealand.

Milker's nodules develop 5 – 14 days after exposure to the virus. They begin as small, red, raised, flat-topped lesions, and over the course of approximately one week, they become red-blue, firm, tender vesicles or nodules, that may develop a greyish skin and small crust. The nodules usually appear on the hands, and less commonly on the face. There may be one or two nodules, or several.[23] As with orf, secondary bacterial infection and erythema multiforme may occur in some cases.

Laboratory investigation is usually not required as milker's nodules can be diagnosed based on the appearance of the lesions and a history of contact with cattle. However, if there is any doubt about the diagnosis, a skin biopsy can be performed.[23]

Management is the same as for orf. Nodules should be covered to prevent contamination, and patients advised to wear gloves if milking. Antibiotic treatment may be required if secondary bacterial infection is present.[23]

See: **www.dermnetnz.org/viral/milkers-nodules.html** for images of milker's nodules

Dermatophyte infections: ringworm

A dermatophyte infection is a skin, nail or hair infection caused by fungi which use keratin for growth. Infections may be acquired from a human (anthropophilic), animal (zoophilic) or soil (geophilic) source. Tinea corporis, known as ringworm, is an example of a dermatophyte infection. The anthropophilic dermatophyte *Trichophyton rubrum* is the most common cause of tinea corporis in New Zealand, and originates from infection in the feet (tinea pedis) or nails (tinea unquium). Tinea corporis caused by *T. rubrum* most often affects people with lowered immunity, e.g. people with diabetes or people treated with oral or topical corticosteroids. It is characterised by annular plaques which expand slowly.

Microsporum canis (from cats and dogs) and *T. verrocosum* (from cattle) are the most commonly implicated zoophilic dermatophyte infections responsible for tinea corporis.[25] Patients with zoophilic (or geophilic) ringworm usually present with single or multiple itchy, inflamed, skin lesions that form irregular expanding rings with a raised, distinct border (Figure 3). There are often scattered follicular pustules and loss of hair within affected areas. The lesions are usually located in exposed areas. Dermatophyte infections rarely occur on or near mucous membranes, helping to differentiate

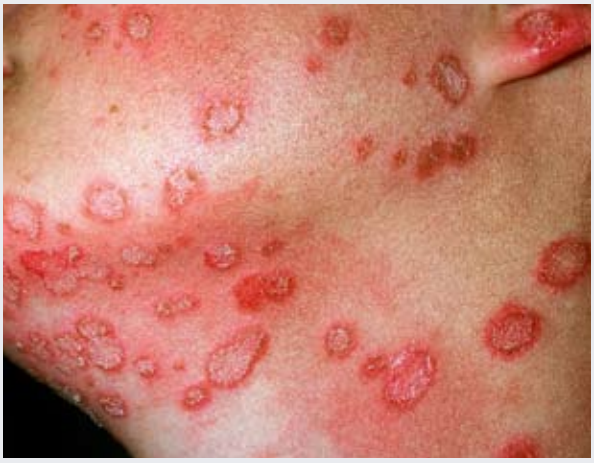


Figure 3: Zoophilic tinea corporis (*M. canis*)
Image provided by DermNet NZ

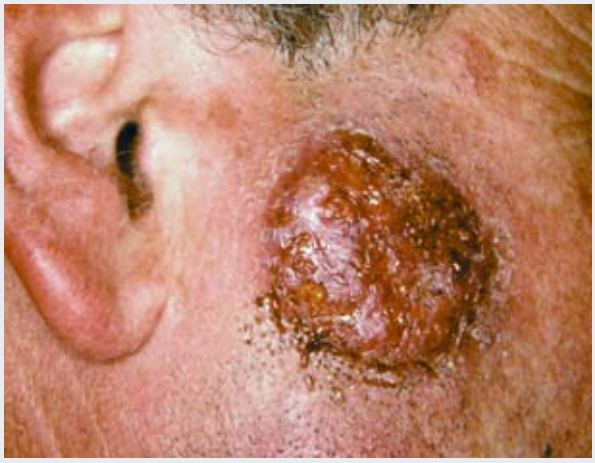


Figure 4: Kerion (*T. verrocosum* – cattle ringworm)
Image provided by DermNet NZ

them from candidal infections.[26] Adults and children in rural areas may present with kerion (fungal abscess – Figure 4).

Diagnosis of tinea corporis can be made by clinical appearance, but should be confirmed by laboratory analysis of skin scrapings and extracted hair shafts. Patients should not use topical anti-fungal medicines for three days prior to a sample being taken as this can prevent identification of the dermatophyte.

Patients with tinea corporis affecting a small area of skin can be treated with topical antifungals (e.g. miconazole or clotrimazole cream). If topical treatment fails, the rash is extensive, there is follicular involvement or the patient has kerion, oral antifungals are appropriate, e.g. terbinafine 250 mg, once daily, for four weeks – sometimes longer.

For further information on collecting skin scrapings, see: "Collecting specimens for the investigation of fungal infections", Best Tests (Mar, 2011)

Erysipeloid infection

Erysipeloid is an infection caused by *Erysipelothrix rhusiopathiae*. It is transferred to humans via contact with raw meat, poultry, fish and shellfish, when bacteria enter the skin through an open wound. Farmers, meat processors and veterinarians are most at risk of infection.[27]

Patients with erysipeloid can be affected in three ways: most often they will present with localised skin lesions, in very rare cases a diffuse cutaneous reaction occurs with multiple lesions across the body, and also rarely, a systemic infection affecting multiple organs can occur. Localised lesions are red-purple, with a smooth, shiny surface. The lesions slowly expand over several days, and develop a sharp or curved border, with very small blisters.[27] The lesions may feel warm, and pain, tenderness and a burning sensation may be reported.[27] Most lesions occur on the hands or fingers, but can form on any skin area exposed to the infected meat or animal.[27]

Laboratory investigation is not required; diagnosis is based on clinical examination. Lesions will resolve spontaneously within two to four weeks.[27] Antibiotic treatment can be considered to shorten the healing time. Oral flucloxacillin is an appropriate treatment; erythromycin or doxycycline are alternatives.[27]

Search: **www.google.com/images** for images of Erysipeloid

Foreign body granulomas: wool handlers

A foreign body granuloma is a non-immunological reaction to an exogenous material (e.g. wood or metal fragment, fibres) that has penetrated the skin. The foreign body is encapsulated within granulation tissue (which contains a proliferation of inflammatory and giant cells) and can mimic a soft tissue tumour. In some cases, a sinus is formed, which can result in infection.

Foreign body granulomas have been reported in people who handle sheep, e.g. wool handlers, shearers, pressers and rousies, although there is little published literature on this. When the wool is handled, wool fibres (especially when wet) may penetrate areas of exposed skin, e.g. the limbs and neck. This is also reported to occur in the breast and nipple area, when fibres penetrate through clothing. The resulting painful, swollen lesion is colloquially referred to as a "grease ball". This condition is similar to trichogranulomas that affect hairdressers or dog groomers, when hair penetrates the skin, usually between the fingers, and there is a foreign body reaction to the presence of keratin in the dermis.

A foreign body granuloma can be diagnosed with histopathology (fine needle aspiration or excision biopsy), which will show characteristic cell formation. Foreign bodies can sometimes be detected on ultrasound, but this is unlikely to reveal a wool fibre. Patients with infected lesions may require local incision and drainage, and antibiotics. Historically, topical application of methylated spirits has been used as a treatment for "grease balls". Protective clothing and gloves, and the use of a barrier (moisturising) cream on exposed skin can help to prevent foreign body granulomas from occurring.

Infections acquired via contact with plants or soil

There are many infectious pathogens which pose a risk to people working in outdoor occupations. For example, bacterial or fungal skin infections can occur in crop and field workers, and there is a risk of tetanus being transferred to a wound from soil. Some less common skin and soft-tissue infections are contracted via water-borne microbes through minor abrasions, e.g. *Aeromonas hydrophila*, a rare cause of cellulitis and abscess, and *Mycobacterium marinum*, a cause of chronic granulomatous plaques.

For further information on Aeromonas skin infection see: **www.dermnetnz.org/bacterial/aeromonas.html**

Paronychia

Horticultural workers are at risk of skin infections due to repeated minor trauma, e.g. from thorns and vines. Paronychia is inflammation of the nail folds, caused by bacterial, viral or yeast infection of the fingers or, less commonly, the toes.[28] It occurs when there is penetration between the proximal nail fold and the nail plate, allowing microbial entry. Disruption of the nail seal can also occur due to a contact irritant or excessive moisture.[28]

Paronychia can be acute or chronic. Acute paronychia is caused by bacterial infection, most commonly *Staphylococcus aureus*, and sometimes *Streptococci* and *Pseudomonas* organisms,[28] or by herpes simplex virus. Chronic paronychia is when symptoms have been present for more than six weeks, and is usually due to a fungal infection, e.g. *Candida albicans*. It is more likely in people who have repeated exposure to water containing chemical irritants or exposure to moist environments.[28] Chronic paronychia may also arise as a complication of hand dermatitis.

Patients with acute paronychia (Figure 5) present with localised pain, tenderness and swelling of the perionychium (epidermis bordering the nails). Discharge may be present if an abscess has formed and infection may extend into the nail bed. The nail may be discoloured or distorted.[28] Laboratory investigation is not required unless the infection is severe. If there are signs of significant bacterial infection, oral antibiotic treatment is recommended; flucloxacillin is an appropriate choice. Incision and drainage is recommended if there is an abscess.[28]

In chronic paronychia (Figure 6), several nails and perionychium appear swollen and tender, with "boggy" nail folds. There is thickening, transverse ridging and discolouration of the nail plate, and separation of the nail from the cuticle and nail folds.[28] Microbiological analysis of nail scrapings can be considered to identify the causative agent. Treatment with a combination of topical corticosteroids and a topical antifungal (when yeast infection is present) is usually successful. If symptoms do not resolve, an oral azole antifungal or antibiotic, depending on the microbes present, can be considered. If medical treatment is unsuccessful and the case is severe, surgical intervention may be considered; this may involve removal of the nail.[28]

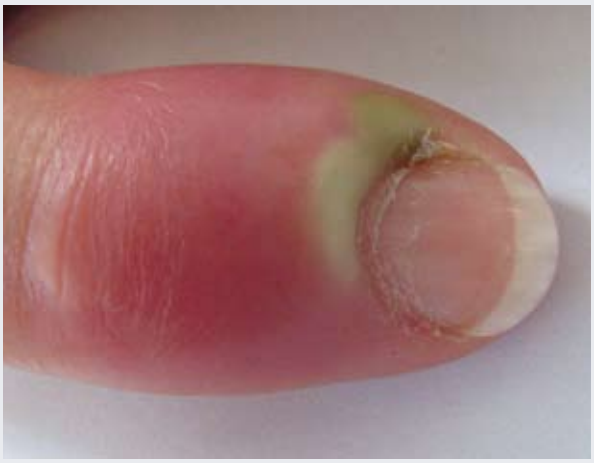


Figure 5: Acute paronychia. Image provided by DermNet NZ

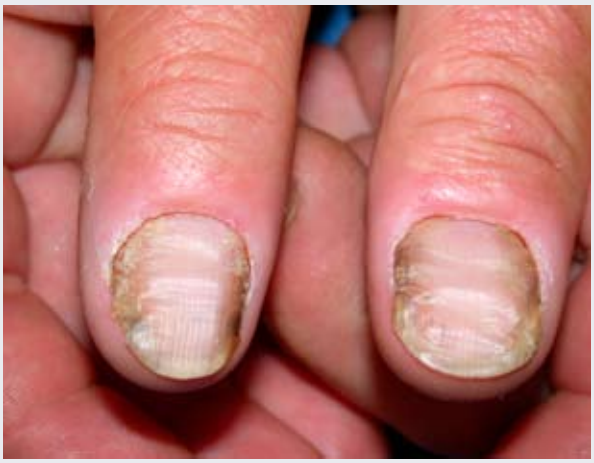


Figure 6: Chronic paronychia. Image provided by DermNet NZ

Tetanus

Clostridium tetani, the causative organism of tetanus, is present in soil, dust and animal faeces. People are at risk of tetanus if infected soil or other matter enters a wound. Once in an anaerobic environment in the wound, *C. tetani* multiplies and releases a toxin which causes the characteristic symptoms of tetanus: muscular rigidity and contraction spasms. Symptoms develop 3 – 21 days after exposure (ten days on average).[29] Initial symptoms include weakness, stiffness or cramps and patients may report difficulty chewing or swallowing food. Muscle spasms usually begin one to four days later. The mortality rate for people with tetanus is approximately 10%, but is higher in older people.[29]

Tetanus is rare in New Zealand due to an effective immunisation programme which was introduced for infants in 1960.[29] Prior to this, only people in the armed forces were likely to have received a primary series of tetanus vaccinations. Most cases of tetanus occur in older people (particularly older women) as they are less likely to have been immunised or to have received booster vaccinations. Between 2000 and 2010, there were 34 people in New Zealand hospitalised with tetanus; 23 of these people were aged over 60 years.[29]

If a patient presents with a tetanus-prone wound, it should be cleaned and dressed, and they should receive a tetanus booster immunisation if they have not had one within the last five to ten years (Table 1). Td (ADT Booster) or Tdap (Boostrix) can be used. Patients with no history of previous tetanus immunisation and a tetanus-prone ("dirty") wound should receive a primary course of tetanus vaccination (three doses) and should also receive tetanus immunoglobulin (TIG). The recommended dose is 250 IU, IM (one ampoule), but this should be increased to 500 IU if the wound occurred more than 24 hours previously or if there is a risk of heavy contamination.[29]

Patients with features suggestive of tetanus should be referred to hospital for further assessment and management.

Table 1: Guide to tetanus prophylaxis in wound management (adapted from Immunisation Handbook, 2011)[29]

Vaccine history	Time since last dose	Type of wound	Tetanus vaccination required?	Tetanus immunoglobulin (TIG) required?
≥ 3 doses	< 5 years	Tetanus-prone	No	No
≥ 3 doses	5 – 10 years	Clean/minor	No	No
≥ 3 doses	5 – 10 years	Tetanus-prone	Booster dose	No
≥ 3 doses	> 10 years	Tetanus prone	Booster dose	No
< 3 doses		Clean/minor	Complete course of three doses	No
< 3 doses		Tetanus-prone	Complete course of three doses	Yes

Assess tetanus status at age 45 and 65 years

The tetanus immunisation status of adults should be reviewed at age 45 and 65 years. If it has been more than ten years since receiving a tetanus vaccination, patients should be offered a booster vaccination: Td (ADT Booster) or Tdap (Boostrix). If they do not have a reliable history of tetanus vaccination a primary course should be given, which is three doses of Td or Tdap, at least four weeks apart. A booster dose is then recommended in ten years.

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