MedicineToday 2013; 14(12): 27-38

PEER REVIEWED FEATURE
POINTS: 2 CPD/2 PDP

Fever in the returned child traveller

AMENEH KHATAMI BHB, MB ChB, DipPaeds; **DAVID ISAACS** MD, FRACP, FRCP; **BEN MARAIS** MMed(Paeds), PhD

Fever is common in children after overseas travel. Although usually a symptom of a nonspecific self-limiting condition, it can signify a serious illness such as malaria, dengue fever, enteric fever, rickettsial disease, tuberculosis, yellow fever or hepatitis. A detailed travel history and examination for specific symptoms and signs can help guide investigations and the decision to refer to hospital.

Key points

- **High or persistent fever can be a marker of serious illness in children who have returned from overseas travel, and requires prompt assessment.**
- **A detailed history of travel, immunisations, prophylaxis and exposures, and thorough medical examination will help focus investigations.**
- **First-line investigations include a full blood count, white blood cell differential count, blood and stool cultures, blood films and a rapid antigen test for malaria.**
- **Respiratory and urinary tract infections are common; consider viral studies of a nasopharyngeal aspirate, chest x-ray and urine dipstick or culture.**
- **Malaria should be ruled out in anyone presenting with persistent or recurrent fever after travel to a malaria-endemic region in the previous year.**
- **Other common infections in travellers include dengue fever, typhoid fever and rickettsial infections.**
- **Children who are systemically unwell with a fever require hospital admission.**

Children represent a significant proportion of international travellers, and the current ease and speed of international travel mean that children frequently present to healthcare providers with illness following recent travel. Up to 8% of travellers to developing countries seek medical advice for illness while abroad or after returning home.[1] Fever occurs more commonly in children than in adults, and in particular is most common in children younger than 5 years.[2] Among child and adolescent travellers who seek care, almost a third present with fever.[3] Although many of these children have nonspecific, self-limiting febrile conditions, fever can be a marker of serious illness and requires timely assessment to decide whether admission to hospital is required for further investigations or treatment.[3,4]

The initial assessment of a returned child traveller with a fever requires a detailed history and physical examination to narrow down the

Dr Khatami is a Fellow in Infectious Diseases and Microbiology at The Children's Hospital at Westmead, Sydney.
Professor Isaacs is Senior Staff Specialist in Infectious Diseases and Microbiology at The Children's Hospital at Westmead and Clinical Professor in Paediatric Infectious Diseases, The University of Sydney, Sydney.
Associate Professor Marais is Associate Professor in Paediatrics and Child Health at The Children's Hospital at Westmead, the Marie Bashir Institute for Infectious Diseases and Biosecurity and The University of Sydney, Sydney, NSW.

TABLE 1. COMMON TRAVEL-ASSOCIATED INFECTIONS IN CHILDREN: PREVENTIVE STRATEGIES*

Disease†	Immunisation and chemoprophylaxis‡	Other preventive strategies
Influenza (laboratory-confirmed cases are notifiable)	• Immunisation >6 months of age • Immunisation recommended if travelling during influenza season at destination	• Pay close attention to hand hygiene • Avoid close contact with ill individuals
Malaria (see WHO and CDC websites for country- and region-specific recommendations)	Chemoprophylaxis§ • Chloroquine-susceptible areas: – chloroquine, once weekly from 1 week prior until 4 weeks after travel • Chloroquine-resistant areas: – atovaquone + proguanil daily from 1 to 2 days prior to 7 days after travel, or – doxycycline (children >8 years) daily from 1 to 2 days prior to 4 weeks after travel, or – mefloquine once weekly from 2 to 3 weeks prior to 4 weeks after travel	• Use topical insect repellents (e.g. DEET) • Wear long-sleeved clothing • Avoid outdoor activities at night
Dengue	None	• Use topical insect repellents (e.g. DEET) • Wear long-sleeved clothing • Avoid activities near stagnant water, especially in shaded areas, during morning and late afternoon
Typhoid	• Polysaccharide vaccine >2 years of age • Live oral vaccine >6 years of age • Vaccine recommended for travel to endemic regions	• 'Boil it, cook it, peel it or avoid it' • Pay close attention to hand hygiene • Use bottled water for drinking or brushing teeth • Avoid ice cubes in drinks
Rickettsial infections (*Rickettsia prowazekii* is notifiable)	None	• Use topical insect repellents (e.g. DEET) • Wear long-sleeved clothing during bushwalking or hiking and perform regular self-inspection for ticks

long list of potential investigations (which are often time-consuming and expensive) to the most relevant tests. These should be chosen based on the area and duration of travel, possible exposures and specific signs and symptoms.

HISTORY TAKING

Area of travel and exposures

Likely infections vary according to the area of travel.[1,5] Thus it is important to take a detailed travel history, including areas visited, duration of stay, activities undertaken and potential exposures, in particular bites by insects or animals, injuries, exposures to open water (rivers, lakes, ponds or the sea), animals, unpasteurised milk and sick contacts. It is crucial to construct an accurate timeline that includes the travel itinerary, likely exposures, symptom onset and progression, precautionary measures taken (if any) and treatment received. Comparing this information with the known incubation periods of different infections may help to identify or exclude specific illnesses.

Chemoprophylaxis and immunisations

Children often travel with their families to visit friends and relatives in their parents' country of origin. In this group of travellers, there are low rates of malaria prophylaxis and pre-travel vaccine uptake.[6] Parents are often unaware of risks and may assume a degree of immunity based on

TABLE 1. COMMON TRAVEL-ASSOCIATED INFECTIONS IN CHILDREN: PREVENTIVE STRATEGIES* continued

Disease†	Immunisation and chemoprophylaxis‡	Other preventive strategies
Meningococcal infection	• Quadrivalent meningococcal A, C, W135, Y vaccine for children >9 months of age • Vaccine compulsory for travel to Saudi Arabia for Hajj or Umra, especially recommended for travel to sub-Saharan Africa ('meningitis belt')	• Avoid close contact with ill individuals
Hepatitis A	• Immunisation >1 year of age • Immunisation recommended for travel to areas of moderate or high endemicity (all developing countries)	• 'Boil it, cook it, peel it or avoid it' • Pay close attention to hand hygiene • Use bottled water for drinking, tooth brushing • Avoid ice cubes in drinks
Tuberculosis (TB)	• Chemoprophylaxis: not indicated pre-travel; consider in vulnerable young children (<5 years) if documented TB exposure or infection (in the absence of disease; adult pulmonary TB cases are the most infectious) • Immunisation: BCG recommended (ideally 3 months prior to travel) for children aged <5 years travelling for extended periods to countries with high prevalence of TB	• Avoid close contact with known TB patients and individuals with suggestive symptoms
Japanese encephalitis	• Immunisation >1 year of age • Immunisation recommended for travel for over a month in rural areas in high-risk endemic regions, especially if considerable outdoor activity	• Use topical insect repellents (e.g. DEET) • Wear long-sleeved clothing during bush-walking or hiking
Yellow fever	• Immunisation >9 months of age • Immunisation recommended for travel to endemic areas, required for travel to or from certain countries	• Use topical insect repellents (e.g. DEET) • Wear long-sleeved clothing, especially during bushwalking or hiking
Rabies	• Immunisation at any age • Immunisation recommended based on risk of exposure and access to postexposure prophylaxis	• Avoid close contact with animals, especially dogs • Appropriate wound care of any animal bite

* See *Communicable Diseases Intelligence* for updates on infections in the Australian setting (www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-cdiintro.htm).
† Nationally notifiable diseases unless otherwise stated. Contact the local public health unit or State/Territory authority for information on notification and public health actions required.
‡ Australian Technical Advisory Group on Immunisation. *Australian immunisation handbook*. 10th ed. Canberra: Australian Government Department of Health and Ageing; 2013.[11]
§ Centers for Disease Control and Prevention. Malaria information and prophylaxis, by country. Available online at: www.cdc.gov/malaria/travelers/country_table/a.html (accessed November 2013).[12]

previous travel to the same area.[5,7] This group is at higher risk of acquisition of infections as they are more likely to travel to rural areas, to travel for longer periods, and to eat and drink local food and water.[8,9]

Multiple cultural and socioeconomic factors may also limit access to pre-travel health care.[9] Even when appropriate prophylaxis is prescribed (such as for malaria), adherence may not be complete, particularly in children, so the clinician should ask about the exact type, duration and dosing regimen used. It is important to remember that malaria prophylaxis may delay the onset of symptoms of malaria.[10] Child travellers may also have incomplete routine childhood immunisations and may be at risk of vaccine-preventable infections such as measles.[3,8]

Preventive strategies for common travel-associated conditions are summarised in Table 1, including available chemoprophylaxis and immunisation options.[11,12]

EXAMINATION: SYMPTOMS AND SIGNS

Fever in returned child travellers may be associated with diarrhoea, which is one of the most common symptoms in travellers but is more likely to have a severe or protracted course in young children.[2,5,13] Diarrhoea may be accompanied by blood in the stool. Causes of diarrhoea in returning travellers include infections with viruses, bacteria (commonly *Campylobacter*, *Shigella*, *Escherichia coli* and

TABLE 2. COMMON TRAVEL-ASSOCIATED INFECTIONS IN CHILDREN: INCUBATION PERIODS AND MANAGEMENT

Common infections	Incubation period	Investigations	Treatment options[15]
Malaria			
Plasmodium falciparum	Usually <1 month	• Rapid antigen test • Thick and thin blood films • Full blood count	• *Uncomplicated malaria:* first-line artemether + lumefantrine; or atovaquone + proguanil (if not used for prophylaxis); or oral quinine + doxycycline or clindamycin • *Severe malaria:* IV artesunate or if parenteral artesunate not available, IV quinine (needs loading dose, monitor blood glucose) until stable and able to tolerate oral treatment
P. malariae	Usually <1 month	• Thick and thin blood films • Full blood count	• Oral chloroquine
P. ovale	Up to 1 year	• Thick and thin blood films • Full blood count	• Oral chloroquine, followed by primaquine for 14 days (check for glucose-6-phosphate dehydrogenase (G6PD) deficiency prior to administration)
P. vivax	Up to 1 year	• Thick and thin blood films • Full blood count	• *If acquired outside Indonesia and Pacific Island nations:* oral chloroquine • *If acquired in Indonesia or Pacific Island nations:* atovaquone + proguanil; or mefloquine (if not used for prophylaxis) • All treatment options to be followed by primaquine for at least 14 days (check for G6PD deficiency prior to administration)

nontyphoidal *Salmonella*) and parasitic protozoa (most commonly *Giardia lamblia* and *Entamoeba histolytica*). Sometimes multiple pathogens are isolated and often no pathogen can be identified.[5]

In febrile children returning from the tropics, examination findings are often nonspecific. Hepatomegaly, splenomegaly and jaundice may occur in malaria, hepatitis and enteric fever. However, the absence of these findings does not exclude these conditions; around half of children with malaria may not have hepatomegaly or splenomegaly.[14]

Specific signs that may help identify a tropical infection in a febrile child include:

- rose spots, which may occur in a minority of patients with typhoid fever early in the course of illness
- skin lesions or an eschar, which may be seen in rickettsial infections such as African tick fever and scrub typhus
- a nonblanching rash, which may be associated with meningococcal disease, rickettsial infection or viral haemorrhagic fever.

Signs that require urgent intervention include respiratory distress, hypotension or poor perfusion, haemorrhagic manifestations, confusion, stiff neck or focal neurological signs.

INVESTIGATIONS

All children with a fever after returning from the tropics should have a full blood count, white blood cell differential count, stool and blood cultures and a blood film for malaria if travel occurred in the preceding 12 months.[5] Investigations for respiratory and urinary tract infections should also be undertaken as appropriate for any child who presents with fever, including studies of a nasopharyngeal aspirate, chest x-ray and urine dipstick test and culture.

Table 2 summarises incubation periods and management of some common travel-related infections, including tests to perform and treatment options.[15] Additional

TABLE 2. COMMON TRAVEL-ASSOCIATED INFECTIONS IN CHILDREN: INCUBATION PERIODS AND MANAGEMENT continued

Common infections	Incubation period	Investigations	Treatment options[15]
Schistosomiasis	1 to 2 months or longer	• Full blood count and white cell differential count • Stool and urine examination for ova (depending on species) • Serology	• Praziquantel. Repeat treatment 1 to 2 months after exposure
Hepatitis A	28 to 30 days	• Serology	• Supportive care
Leptospirosis	<30 days (usually 2 to 3 weeks)	• Culture from blood, cerebrospinal fluid or urine • Paired acute and convalescent phase serology • Antigen detection in tissues	• IV penicillin, within 7 days, for severe cases • Oral doxycycline or, in young children, azithromycin, for mild infections
Rickettsial infections	1 to 3 weeks	• Paired acute and convalescent phase serology	• Oral doxycycline (10 days); or in children <8 years, azithromycin (5 days) • Severe disease may require IV tetracycline
Enteric fever	1 to 2 weeks (up to 60 days)	• Blood and stool culture	• Initially, a third-generation cephalosporin until bacteraemia resolved and clinically improving • Completion of 2 to 3 weeks with an oral antibiotic (ideally ciprofloxacin) depending on sensitivity pattern
Dengue fever	4 to 7 days	• Paired acute and convalescent phase serology • RNA or antigen in blood • Full blood count	• Supportive

investigations (e.g. specific nucleic acid amplification tests or serological assays) may be indicated according to the travel and exposure history.

SPECIALIST REFERRAL AND PUBLIC HEALTH NOTIFICATION

Although many fever episodes in returned child travellers are likely to represent non-specific self-limiting illnesses, it is recommended that cases of moderate illness should be discussed with a paediatric infectious disease specialist to ensure that appropriate investigations are carried out. Signs of severe infection (such as respiratory distress, poor perfusion or neurological signs and symptoms) or complications such as bleeding should prompt urgent referral to hospital. Several infections require notification to public health authorities, including malaria, typhoid fever, dengue fever and viral hepatitis. A full list of notifiable diseases can be found at the Australian Government Department of Health website (www.health.gov.au/casedefinitions).

COMMON TRAVEL-ASSOCIATED FEBRILE ILLNESSES

Malaria

Malaria is a global tropical disease caused by *Plasmodium*, a parasitic protozoan that is spread to humans by infected *Anopheles* mosquitoes, which bite from dusk to dawn. In 2010 there were an estimated 220 million cases of malaria and an estimated 660,000 deaths (mainly in African children).[16] In recent years, 300 to 400 cases of imported malaria have been notified annually in Australia, with 9 to 13% involving children aged under 15 years.[17] Among children who present with a febrile illness after recent travel, malaria is the most common specific cause identified, particularly if travel was prolonged and the destination was in sub-Saharan Africa.[5]

Four main *Plasmodium* species cause malaria in humans: *Plasmodium falciparum*, *P. vivax*, *P. malariae* and *P. ovale*.

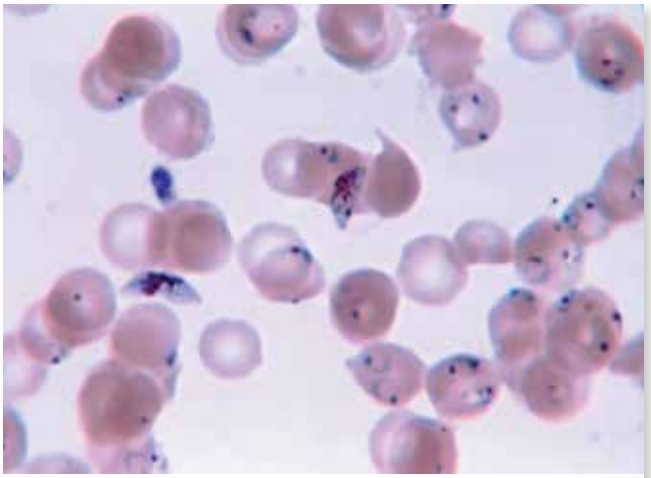

Figure 1. *Plasmodium falciparum* ring forms and gametocytes (thin film Giemsa-stained micrograph).

Courtesy of Centers for Disease Control and Prevention/Steven Glenn, Laboratory & Consultation Division (Public Health Image Library, http://phil.cdc.gov).

Figure 2. A female *Aedes aegypti* mosquito taking a blood meal.

Courtesy of Professor Frank Hadley Collins, Director of the Centre for Global Health and Infectious Diseases, University of Notre Dame, and James Gathany, biomedical photographer, Centers for Disease Control and Prevention (Public Health Image Library, http://phil.cdc.gov).

In a nonimmune individual (young children or adults who have not lived in an endemic area), symptoms usually start one to two weeks after the infective mosquito bite and may initially be nonspecific (fever, headache, chills and vomiting).

The most severe form of malaria is caused by *P. falciparum*, and the progression to severe anaemia, metabolic acidosis, hypoglycaemia, seizures, coma or death can be rapid. Most patients with *P. falciparum* malaria present within one month of exposure, although symptoms may be delayed if malaria prophylaxis is used.

For malaria caused by either *P. vivax* or *P. ovale*, clinical relapses may occur up to a year after the first infection, caused by the release of dormant liver forms of the parasite known as hypnozoites (absent in *P. falciparum* and *P. malariae*).[18] Therefore, a complete travel history should include details of travel to malaria-endemic regions in the previous 12 months.

Diagnosis of malaria is based on detection of malaria antigen (highly reliable for *P. falciparum* but not for other *Plasmodium* species) or parasites in the peripheral blood (Figure 1). Any patient who has spent time in a malaria-endemic region and has a history of prolonged or recurrent fever should be tested for malaria, even if they are afebrile at the time of review. Thick blood films are more sensitive for diagnosing malaria, whereas thin films are more useful for identifying the parasite species. If results are initially negative, blood films need to be repeated at least twice, ideally during fever episodes, before malaria can be excluded with confidence in a febrile child who has returned from any tropical area. Thrombocytopenia is common, and a platelet count above 190 x 10^9/L makes malaria less likely (negative predictive value of 97%).[6]

Resistance patterns to antimalarial treatment are variable, and up-to-date information for the area of travel can be obtained from the US Centers for Disease Control and Prevention.[12] In Australia, current recommendations for treatment are given in *Therapeutic Guidelines: Antibiotic*.[15] Severe malaria requires intravenous treatment in hospital, but initial hospitalisation is recommended even for uncomplicated *P. falciparum* malaria and also for severe cases of nonfalciparum malaria. In addition, eradication of liver hypnozoites is required for *P. vivax* and *P. ovale* malaria; glucose-6-phosphate dehydrogenase deficiency should be excluded before primaquine treatment to avoid haemolysis.

Dengue fever

Dengue fever is caused by a flavivirus transmitted by the bite of *Aedes aegypti* mosquitoes (Figure 2). These mosquitoes bite during the day, mostly in the morning and afternoon, in cooler shaded areas and around stagnant water. Infection risk is highest in the rainy season. In the past five years, more than 1000 cases of dengue fever have been notified annually in Australia, with around 5% in children. Dengue is endemic in most tropical regions and has important public health implications for Australia. The mosquito vector is present in northern Queensland, and local outbreaks have been attributed to travellers returning with the disease from South-East Asia.

The incubation period of dengue is short (four to seven days), and patients usually present within the first week after travel to an endemic area. Four serotypes of dengue virus can infect humans, and prior infection with one serotype can cause subsequent infections to be more severe. Symptoms may range from a mild influenza-like illness with fever, headache and myalgia, often associated with a maculopapular rash, to haemorrhagic complications or vascular leakage with shock, which can be fatal.

Initial diagnosis is clinical, with

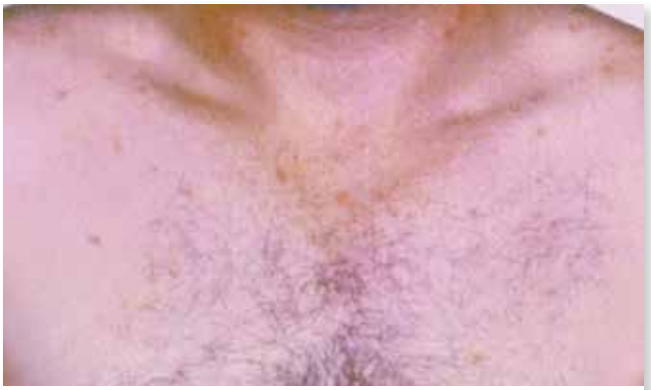

Figure 3. Rose spots on the chest of an adult patient with typhoid fever.

Courtesy of Charles N. Farmer, Armed Forces Institute of Pathology; through the Centers for Disease Control and Prevention (Public Health Image Library, http://philcdc.gov).

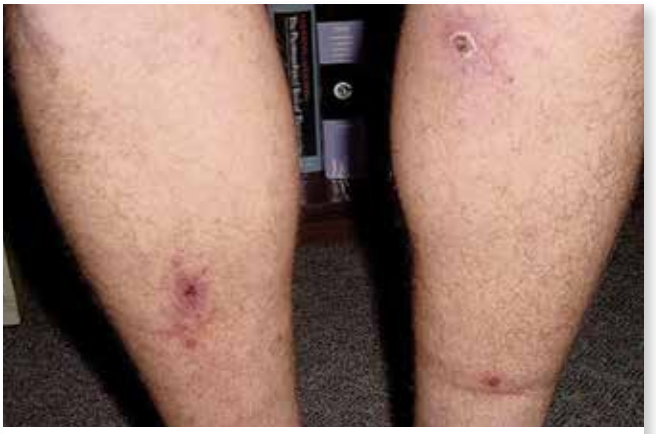

Figure 4. Eschars associated with the sites of tick bites in an adult patient with rickettsial infection.

Courtesy of Centers for Disease Control and Prevention (www.cdc.gov).

confirmation based on serological assays or detection of dengue virus RNA or antigen in the blood; however, serology results are often negative during the acute illness and other results may not be available immediately. Treatment is supportive and mild cases resolve spontaneously, but vigilance is required for potential danger signs (severe abdominal pain, persistent vomiting, blood in vomit, bleeding gums, rapid breathing, fatigue, restlessness). Patients with complications need to be closely monitored in hospital, with special attention to shock and fluid management.

Enteric fevers (typhoid and paratyphoid)

Typhoid fever is caused by infection with the bacterium *Salmonella enterica* serovar Typhi (*S. typhi*). With the exception of Western Europe, North America, Japan, Australia and New Zealand, *S. typhi* is endemic worldwide and recent data suggest that the worldwide incidence of typhoid fever is increasing.[19,20]

S. typhi infects only humans and transmission is via the faecal–oral route. The incubation period is usually one to two weeks but can be as long as 60 days. Symptoms are generally nonspecific. Fever is the most common symptom, but others include cough, headache, stomach pain, constipation or diarrhoea, lethargy, and muscle and joint pains. In the youngest age groups, signs and symptoms are highly variable and nonspecific, making diagnosis particularly challenging. Hepatomegaly and/or splenomegaly is a helpful sign, but relative bradycardia is usually not seen in young children. Rose spots (2 to 4 mm blanching erythematous maculopapular lesions) may occur on the abdomen and chest in 5 to 30% of patients (Figure 3).[21] The most serious complication is gastrointestinal perforation, which occurs in 1 to 2% of hospitalised patients. Chronic carriage occurs in 1 to 4% of those infected, with the risk increased by pre-existing gallbladder disease.

Diagnosis of typhoid is based on culturing the organism from blood, stool or bone marrow, but sensitivity is generally poor (60% and 30% for blood and stool, respectively, and up to 90% for bone marrow).[21] Samples from multiple sites should be taken as soon as possible after presentation to optimise the yield. The Widal (serology) test performs poorly and should not be used in children.

Treatment with fluoroquinolones is highly effective and safe in all age groups. It is associated with the highest cure rates (lower rates of relapse or chronic carriage) if the organism is sensitive, and the fastest time to resolution of fever compared with other antibiotics.[21] However, reduced susceptibility to fluoroquinolones is an emerging problem in Asia, and initial empiric treatment with an intravenous third-generation cephalosporin in hospital is recommended until the organism's antibiotic sensitivity profile is available.[22]

Immunisation provides some protection against typhoid fever and should be offered to travellers to areas of high endemicity for periods of travel of 10 to 14 days or longer. An oral live attenuated vaccine is licensed for use in children over the age of 6 years, and a parenteral polysaccharide vaccine is licensed for use in children over the age of 2 years. Both of these vaccines are around 60% effective.[23,24]

Paratyphoid fever is caused by other *S. enterica* serovars (Paratyphi A, B and C). The clinical syndrome is indistinguishable from typhoid fever, but typhoid vaccines do not protect against paratyphoid infection.

Rickettsial diseases

Rickettsial diseases are transmitted by arthropods (usually ticks). Camping and hiking in endemic areas are particular risk factors for infection. This group of infections includes Rocky Mountain spotted fever (transmitted by brown dog ticks), epidemic typhus (transmitted by body lice), scrub typhus, murine typhus (transmitted to humans by rat or cat fleas) and African

tick fever (associated with tick bites). Rickettsial infections are a common cause of fever in travellers who have gone bushwalking in sub-Saharan Africa.[1]

The classic symptoms of rickettsial infection comprise fever, rash (involving the palms of the hands), headache and myalgia. An eschar may be present at the bite site (Figure 4). Diagnosis can be confirmed by serological testing of paired acute and convalescent phase blood samples, but if infection is suspected clinically, empiric treatment may be started. First-line treatment is usually doxycycline. Azithromycin can be used for young children. Specialist advice should be sought for severe cases.[15]

Tuberculosis

Mycobacterium tuberculosis infection (TB) has high prevalence in some parts of the world, with the risk of infection related to the proximity and duration of exposure to people who are potentially infectious. Children, especially infants, are more susceptible than adults. Investigations for TB should be carried out in any child who has stayed in areas of high endemicity and who presents with any symptom or sign compatible with TB, including fever, weight loss, lethargy, persistent lymph node enlargement or chronic cough. Discussion with a paediatric infectious disease specialist is recommended to ensure appropriate testing and management.

Antibiotic-resistant organisms

Globally the incidence of antibiotic-resistant organisms is increasing. In recent years, a rapid rise in infections caused by multidrug-resistant Gram-negative bacteria has been noted in almost every country. This is of particular concern in India and other Asian countries where widespread use of non-prescription antibiotics has led to significant selection pressure. The increase in resistance in Gram-negative bacteria is mainly due to mobile genes on plasmids that can be easily passed between bacterial populations. Increasing human migration and international travel allow selected bacterial clones and plasmids to be rapidly transported between countries.

Examples of antibiotic-resistant pathogens that may be related to previous travel (or history of travel in close contacts) include fluoroquinolone-resistant *S. typhi* causing typhoid fever, and extended spectrum β-lactamase (ESBL)-producing enterobacteria causing urinary tract infections. It is important to be aware of the potential for drug-resistant pathogens and, where possible, to tailor antibiotic use to culture and sensitivity results.

Other imported febrile illnesses

Yellow fever and other viral haemorrhagic fevers. Yellow fever is caused by a flavivirus transmitted by several mosquito vectors, including *Aedes* species. The yellow fever

vaccine is very effective and is required for travel to most countries in sub-Saharan Africa, and to Central and South America. It is contraindicated in children younger than 9 months of age and in immunocompromised individuals as it is a live virus vaccine. Several other less common forms of viral haemorrhagic fever can occur as outbreaks, usually in Africa. Up-to-date information regarding recent outbreaks can be found on the websites of the Centers for Disease Control and Prevention (www.cdc.gov) and World Health Organization (www.who.int).

Leishmaniasis. The causative protozoan of this disease is transmitted to humans by infected sandflies that typically bite between dusk and dawn. Cutaneous and mucocutaneous forms (ulcers on the skin or mucous membranes) occur more commonly in the Middle East and Central and South America than visceral leishmaniasis (fever, anaemia and hepatosplenomegaly), which is more common in Africa and the Mediterranean region.

Schistosomiasis. This parasitic disease is widespread in Africa and infection with the causative trematodes occurs through exposure to fresh water (e.g. at Lake Malawi). Acute infection (Katayama syndrome) can cause high persistent fevers that may last several weeks. Eosinophilia and urticarial rash are important clues to recent primary infection, although the incubation period is relatively long with acute symptoms starting typically one to two months after exposure.[25]

Hepatitis A and hepatitis E. These viral infections are endemic in most parts of the world and are usually contracted by exposure to contaminated food or water. Both infections can manifest with diarrhoea and jaundice and rarely cause fever. The incubation period of hepatitis A is 28 to 30 days, and infection is often subclinical in young children. Hepatitis A is preventable by a highly effective vaccine.

Leptospirosis. This bacterial infection is typically spread through contact of mucous membranes or broken skin with water contaminated with animal urine. It causes an acute flu-like illness, sometimes associated with aseptic meningitis. The incubation period ranges from one to 30 days (usually two to three weeks), and most infections cause mild symptoms and are self-limited. Rarely, a rapidly progressive, multisystem form (Weil syndrome) occurs, presenting with jaundice, haemorrhage, acute renal failure and/or pulmonary and cardiac involvement.

Chikungunya fever. This disease is caused by an alphavirus transmitted by *Aedes* mosquitoes in tropical residential areas of Africa and Asia. The incubation period is short (one to three days), and infected patients usually present with fever and severe polyarthalgia and arthritis, which can be severe and persistent over several

months. There is no specific treatment and most patients recover completely.

Herpes simian B virus infection. This herpes virus usually infects macaque monkeys but can be transmitted to humans through bites, scratches or mucous membrane exposures. Contact with monkeys around temples in South-East Asia can be a risk factor. The virus causes severe and permanent neurological disease, with case fatality rates up to 80% if untreated. Early treatment with intravenous aciclovir can prevent severe disease.

COMMON CHILDHOOD INFECTIONS

Common infections of childhood, also referred to as cosmopolitan infections, are frequently responsible for fever in returned child travellers, including those returning from tropical locations.[6] Several infections occur more frequently after travel, including respiratory tract infections (e.g. pharyngitis, otitis media and influenza), urinary tract infections, meningococcal disease, glandular fever (caused by Epstein-Barr virus or cytomegalovirus) and staphylococcal skin infections.

CONCLUSION

Fever is a common complaint in children following overseas travel. Prompt evaluation, including a detailed travel history and thorough clinical examination, will help guide investigations. First-line investigations for all febrile children returning from the tropics include a full blood count, stool culture, blood culture and blood films for malaria, as well as urine dipstick and culture and a chest x-ray, depending on signs and symptoms. Signs that require urgent intervention include respiratory distress, hypotension, poor perfusion, haemorrhagic manifestations, confusion, stiff neck or focal neurological signs.

REFERENCES

1. Freedman DO, Weld LH, Kozarsky PE, et al. Spectrum of disease and relation to place of exposure among ill returned travelers. N Engl J Med 2006; 354: 119-130.
2. Newman-Klee C, D'Acremont V, Newman CJ, Gehri M, Genton B. Incidence and types of illness when traveling to the tropics: a prospective controlled study of children and their parents. Am J Trop Med Hyg 2007; 77: 764-769.
3. Wilson ME, Weld LH, Boggild A, et al. Fever in returned travelers: results from the GeoSentinel Surveillance Network. Clin Infect Dis 2007; 44: 1560-1568.
4. Klein JL, Millman GC. Prospective, hospital based study of fever in children in the United Kingdom who had recently spent time in the tropics. BMJ 1998; 316: 1425-1426.
5. Hagmann S, Neugebauer R, Schwartz E, et al. Illness in children after international travel: analysis from the GeoSentinel Surveillance Network. Pediatrics 2010; 125: e1072-1080.
6. West NS, Riordan FA. Fever in returned travellers: a prospective review of hospital admissions for a 2(1/2) year period. Arch Dis Child 2003; 88: 432-434.
7. Bradley D, Warhurst D, Blaze M, Smith V. Malaria imported into the United Kingdom in 1992 and 1993. Commun Dis Rep CDR Rev 1994; 4: R169-R172.
8. Angell SY, Cetron MS. Health disparities among travelers visiting friends and relatives abroad. Ann Intern Med 2005; 142: 67-72.
9. Bacaner N, Stauffer B, Boulware DR, Walker PF, Keystone JS. Travel medicine considerations for North American immigrants visiting friends and relatives. JAMA 2004; 291: 2856-2864.
10. Reyburn H, Behrens RH, Warhurst D, Bradley D. The effect of chemoprophylaxis on the timing of onset of falciparum malaria. Trop Med Int Health 1998; 3: 281-285.
11. Australian Technical Advisory Group on Immunisation. Australian immunisation handbook. 10th ed. Canberra: Australian Government Department of Health and Ageing; 2013.
12. Centers for Disease Control and Prevention. Malaria information and prophylaxis, by country. Available online at: www.cdc.gov/malaria/travelers/country_table/a.html (accessed November 2013).
13. Pitzinger B, Steffen R, Tschopp A. Incidence and clinical features of traveler's diarrhea in infants and children. Pediatr Infect Dis J 1991; 10: 719-723.
14. Ladhani S, Aibara RJ, Riordan FA, Shingadia D. Imported malaria in children: a review of clinical studies. Lancet Infect Dis 2007; 7: 349-357.
15. Antibiotic Expert Group. Therapeutic guidelines: antibiotic. Version 14. Melbourne: Therapeutic Guidelines Limited; 2010.
16. World Health Organization. Malaria factsheet. Available online at: www.who.int/mediacentre/factsheets/fs094/en (accessed November 2013).
17. Australian Government Department of Health and Ageing. National Notifiable Diseases Surveillance System - malaria [updated 20/06/2013]. Available online at: www9.health.gov.au/cda/source/rpt_5_sel.cfm (accessed November 2013).
18. Brabin BJ, Ganley Y. Imported malaria in children in the UK. Arch Dis Child 1997; 77: 76-81.
19. Crump JA, Luby SP, Mintz ED. The global burden of typhoid fever. Bull World Health Organ 2004; 82: 346-353.
20. Buckle GC, Walker CL, Black RE. Typhoid fever and paratyphoid fever: systematic review to estimate global morbidity and mortality for 2010. J Glob Health 2012; 2: 10401.
21. Parry CM, Hien TT, Dougan G, White NJ, Farrar JJ. Typhoid fever. N Engl J Med 2002; 347: 1770-1782.
22. Threlfall EJ, Ward LR, Skinner JA, Smith HR, Lacey S. Ciprofloxacin-resistant Salmonella typhi and treatment failure. Lancet 1999; 353: 1590-1591.
23. Levine MM, Ferreccio C, Black RE, Germanier R. Large-scale field trial of Ty21a live oral typhoid vaccine in enteric-coated capsule formulation. Lancet 1987; 1: 1049-1052.
24. Klugman KP, Gilbertson IT, Koornhof HJ, et al. Protective activity of Vi capsular polysaccharide vaccine against typhoid fever. Lancet 1987; 2: 1165-1169.
25. Ross AG, Vickers D, Olds GR, Shah SM, McManus DP. Katayama syndrome. Lancet Infect Dis 2007; 7: 218-224.

COMPETING INTERESTS: None.