Tropical ID

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Tropics

What’s in a Name?
Tropics
Torrid Zone v Temperate Zone
Let’s Call it:

Exotic Infection
Evaluation
Exotic Infection

- Travel history
- At risk exposures
- Prophylaxis
- Incubation periods
- Syndromes
The brave warriors within the trojan horse faced many hardships...giant mythical beasts being one.

Right. Someone please change seats with me.

But it seemed like the safest place at the time
Learn to communicate the important details first!!

…..Help juniors learn
Exotic Infection – The 4 I’s

• **Insect Bites**
  – Mosquitoes, biting flies, midges, sandflies
  – Ticks, mites, bugs

• **Ingestion**
  – GI pathogens
  – Entry point for tissue tropism organisms

• **Inoculation**
  – Skin entry – trauma (major/minor) or not

• **Inhalation**
  – Respiratory pathogens
  – Entry point for tissue tropism organisms
Syndromes

- Fever
- Neurological
- Respiratory
- Gastrointestinal
- Skin & Soft Tissue
- Eosinophilia/Parasitic
- NOT Infection / Exotic
- Combinations of the above
Syndromic Approach Considerations

- Epidemiology, outbreaks, news
- Endemicity (travellers can re-define)
- Immunity – local v. visitor/non-local
- History
- Physical examination
- Adult or Child
- Socio-economic group
- Genetic make-up, pre-existing illness
Illness in Travellers - Incubation Periods

**Less than 21 days**
- Anthrax, Angiostrongylus
- Arbovirus (eg DF, YF)
- Brucellosis
- Enteric fevers (typhoid)
- GIT infections, HAV, HIV
- Leptospirosis, Lyme
- Malaria (all species)
- Meningococcal
- Rabies, RTIs ('flu, LD)
- Schistosomiasis
- Trypanosomiasis, Typhus
- VHF (Ebola, Lassa, CCHF etc)

**> 21 days (m/b years)**
- Amebic Liver Abscess
- Angiostrongylus
- Brucellosis
- Enteric Fevers (typhoid)
- Filariasis
- HAV, HBV, HCV, HEV, HIV
- Histoplasma, HIV, Kala-azar
- Lyme, Malaria, Melioidosis
- Rabies, Schistosomiasis
# Fever in Returned Travellers

<table>
<thead>
<tr>
<th>More Common</th>
<th>Less Common</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria (all species)</td>
<td>Brucellosis, Filariasis</td>
</tr>
<tr>
<td>Dengue</td>
<td>Leptospirosis, Melioidosis</td>
</tr>
<tr>
<td>Viral RTI (influenza)</td>
<td>Pneumonia (incl LD)</td>
</tr>
<tr>
<td>GIT infections</td>
<td>Q fever, Reiters, Fungal</td>
</tr>
<tr>
<td>Enteric fevers (typhoid)</td>
<td>Schistosomiasis, Typhus</td>
</tr>
<tr>
<td>Pyogenic/septicemia</td>
<td>Trypanosomiasis (African)</td>
</tr>
<tr>
<td>Meningococcal</td>
<td>Visceral leishmanianias</td>
</tr>
<tr>
<td>Amoebic liver abscess</td>
<td>Viral hemorrhagic fevers</td>
</tr>
<tr>
<td>Not infection (drug S/E)</td>
<td></td>
</tr>
</tbody>
</table>
### 3. Causes of fever in Canadian and Australian studies of returned travellers

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Percentage of fever cases</th>
<th>Canada $(n=587)^4$</th>
<th>Australia $(n=232)^5$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undiagnosed</td>
<td></td>
<td>25</td>
<td>9</td>
</tr>
<tr>
<td>Malaria</td>
<td></td>
<td>32</td>
<td>27</td>
</tr>
<tr>
<td>Respiratory infection*</td>
<td></td>
<td>11</td>
<td>18</td>
</tr>
<tr>
<td>Hepatitis</td>
<td></td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Diarrhoeal illness</td>
<td></td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td></td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Dengue</td>
<td></td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Enteric fever</td>
<td></td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Epstein–Barr virus infection</td>
<td></td>
<td>2</td>
<td>0.4</td>
</tr>
<tr>
<td>Rickettsial infection</td>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Amoebiasis</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td></td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Acute HIV infection</td>
<td></td>
<td>0.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Other infections</td>
<td></td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Other causes</td>
<td></td>
<td>4</td>
<td>3</td>
</tr>
</tbody>
</table>

* Including upper respiratory tract infection, bronchitis and pneumonia.
Figure 2. Number of cases of selected acute and potentially life-threatening diseases by exposure region among 82,825 ill western travelers to the tropics: data from the GeoSentinel surveillance network, 1996–2011.

Quarantinable Illnesses
<table>
<thead>
<tr>
<th>Disease</th>
<th>Status</th>
<th>Date of last record and notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholera</td>
<td>Free</td>
<td>Small number of cases are reported annually and related to overseas travel or imported food products&lt;sup&gt;24&lt;/sup&gt;</td>
</tr>
<tr>
<td>Plague</td>
<td>Free</td>
<td>Last case recorded in Australia in 1923&lt;sup&gt;25&lt;/sup&gt;</td>
</tr>
<tr>
<td>Rabies</td>
<td>Free</td>
<td>Last case (overseas acquired) recorded in Australia in 1990&lt;sup&gt;26&lt;/sup&gt;</td>
</tr>
<tr>
<td>Smallpox</td>
<td>Free</td>
<td>Last case recorded in Australia in 1938&lt;sup&gt;27&lt;/sup&gt;</td>
</tr>
<tr>
<td>Yellow fever</td>
<td>Free</td>
<td>No cases recorded on shore in Australia – 5 occasions on which vessels arrived in Australian ports 1892–1915&lt;sup&gt;25&lt;/sup&gt;</td>
</tr>
<tr>
<td>SARS</td>
<td>Free</td>
<td>Last case recorded in Australia in 2003&lt;sup&gt;28&lt;/sup&gt;</td>
</tr>
<tr>
<td>HPAIH</td>
<td>Free</td>
<td>No cases recorded&lt;sup&gt;29&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

**Viral haemorrhagic fevers**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Status</th>
<th>Date of last record and notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ebola</td>
<td>Free</td>
<td>No cases recorded</td>
</tr>
<tr>
<td>Marburg</td>
<td>Free</td>
<td>No cases recorded</td>
</tr>
<tr>
<td>Lassa</td>
<td>Free</td>
<td>No cases recorded</td>
</tr>
<tr>
<td>Crimean–Congo</td>
<td>Free</td>
<td>No cases recorded</td>
</tr>
</tbody>
</table>
Lifelong Risk of Reactivation

**Bacterial**
- Leprosy, TB, Melioidosis
- Syphilis, Lyme
- Typhoid, Q fever
- Louse-borne Typhus

**Viral**
- HIV/AIDS, HTLV I/II
- Hep.B,C,D, HSV 1&2

**Fungal**
- Coccidioidomycosis
- Histoplasmosis
- *Penicillium marneffei*

**Protozoal**
- *P. malariae*, Toxoplasmosis
- Chagas’ Disease
- Amoebic (*E. histolytica*)
- Visceral leishmaniasis

**Worms**
- Hydatid, Cysticercosis
- Strongyloides
- Schistosomiasis
YOU'RE EATING TOO MUCH FIBER!
Enteropathogens and Chronic Illness in Returning Travelers


Figure 1. Relative Risk of Chronic Enteropathogenic Infections Acquired during Travel Abroad.
Data are from Swaminathan et al.9

Table 2  Rates (per 1000 returned unwell travellers) of the most frequently isolated pathogens.

<table>
<thead>
<tr>
<th>Clinically significant IGD pathogen</th>
<th>n</th>
<th>% Total</th>
<th>Rate per 1000 returned travellers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Giardia</td>
<td>810</td>
<td>27.9</td>
<td>31.3</td>
</tr>
<tr>
<td>Campylobacter</td>
<td>384</td>
<td>13.2</td>
<td>14.8</td>
</tr>
<tr>
<td>Entamoeba histolytica</td>
<td>363</td>
<td>12.5</td>
<td>14.0</td>
</tr>
<tr>
<td>Shigella</td>
<td>182</td>
<td>6.3</td>
<td>7.0</td>
</tr>
<tr>
<td>Strongyloides</td>
<td>176</td>
<td>6.1</td>
<td>6.8</td>
</tr>
<tr>
<td>Salmonella spp. other</td>
<td>134</td>
<td>4.6</td>
<td>5.2</td>
</tr>
<tr>
<td>Dientamoeba fragilis</td>
<td>116</td>
<td>4.0</td>
<td>4.5</td>
</tr>
<tr>
<td>Ascaris</td>
<td>110</td>
<td>3.8</td>
<td>4.3</td>
</tr>
<tr>
<td>S. typhi</td>
<td>99</td>
<td>3.4</td>
<td>3.8</td>
</tr>
<tr>
<td>Hookworm</td>
<td>71</td>
<td>2.4</td>
<td>2.7</td>
</tr>
<tr>
<td>Tapeworm</td>
<td>71</td>
<td>2.4</td>
<td>2.7</td>
</tr>
<tr>
<td>Hepatitis A virus</td>
<td>67</td>
<td>2.3</td>
<td>2.6</td>
</tr>
<tr>
<td>Trichuris trichura</td>
<td>52</td>
<td>1.8</td>
<td>2.0</td>
</tr>
<tr>
<td>S. paratyphi</td>
<td>47</td>
<td>1.6</td>
<td>1.8</td>
</tr>
<tr>
<td>C. difficile</td>
<td>38</td>
<td>1.3</td>
<td>1.5</td>
</tr>
<tr>
<td>Enterobius</td>
<td>36</td>
<td>1.2</td>
<td>1.4</td>
</tr>
<tr>
<td>Cryptosporidium</td>
<td>32</td>
<td>1.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Cyclospora</td>
<td>31</td>
<td>1.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Hepatitis E virus</td>
<td>31</td>
<td>1.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Yersinia spp. (non-pestis)</td>
<td>20</td>
<td>0.7</td>
<td>0.8</td>
</tr>
<tr>
<td>Clonorchis</td>
<td>19</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Fasciola</td>
<td>5</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Trichomonas intestinalis</td>
<td>4</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Isospora</td>
<td>3</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>V. cholerae</td>
<td>1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>2902</td>
<td>100</td>
<td><strong>112.2</strong></td>
</tr>
</tbody>
</table>

*From single or multiple regions.

Figure 1  (a) Rates of parasitic pathogens per 1000 returned unwell travellers from a single region, (b) rates of bacterial pathogens per 1000 returned unwell travellers from a single region, (c) rates of viral/protozoal pathogens per 1000 returned unwell travellers from a single region. "Western Industrialized World not shown. "Other" refers to the mean rate of other isolated pathogen/s (where each individual pathogen’s rate is less than 5.5/1000 returned travellers) (note: also relevant for Fig. 1b,c).

Swaminatham A. J. Infection 2009
25,867 international travellers, 7442 ill, 8273 GI infection
Something's just not right -- our air is clean, our water is pure, we all get plenty of exercise, everything we eat is organic and free-range, and yet nobody lives past thirty.

It's infection you fool!

(......& crocodiles)
Tests
<table>
<thead>
<tr>
<th>Fever and rash</th>
<th>Dengue, chikungunya, rickettsial infections, enteric fever (skin lesions may be sparse or absent), acute HIV infection, measles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever and abdominal pain</td>
<td>Enteric fever, amebic liver abscess</td>
</tr>
<tr>
<td>Undifferentiated fever and normal or low white blood cell count</td>
<td>Dengue, chikungunya, zika, malaria, rickettsial infection, enteric fever,</td>
</tr>
<tr>
<td>Fever and haemorrhage</td>
<td>Viral hemorrhagic fevers (dengue and others), meningococcemia, leptospirosis, rickettsial infections</td>
</tr>
<tr>
<td>Fever and eosinophilia</td>
<td>Acute schistosomiasis, drug hypersensitivity reaction, fascioliasis and other parasitic infections (rare)</td>
</tr>
<tr>
<td>Fever and pulmonary infiltrates</td>
<td>Common bacterial and viral pathogens, legionellosis, acute schistosomiasis, Q fever, leptospirosis</td>
</tr>
<tr>
<td>Fever and altered mental status</td>
<td>Cerebral malaria, viral or bacterial meningoencephalitis, African trypanosomiasis, scrub typhus</td>
</tr>
<tr>
<td>Mononucleosis syndrome</td>
<td>Epstein–Barr virus infection, cytomegalovirus infection, toxoplasmosis, acute HIV</td>
</tr>
<tr>
<td>Fever persisting &gt;2 weeks</td>
<td>Malaria, enteric fever, Epstein-Barr virus infection, cytomegalovirus infection, toxoplasmosis, acute HIV, acute schistosomiasis, brucellosis, tuberculosis, Q fever, visceral leishmaniasis (rare)</td>
</tr>
<tr>
<td>Fever with onset &gt;6 weeks after travel</td>
<td>Plasmodium vivax or ovale malaria, acute hepatitis (B, C, or E), tuberculosis, amebic liver abscess</td>
</tr>
</tbody>
</table>
9: Infections in the returned traveller

Remember to consider serious yet treatable diseases, such as malaria

Abstract

The usual presentation of a returned traveller is with a particular syndrome — fever, respiratory infection, diarrhoea, eosinophilia, or skin or soft tissue infection — or for screening for asymptomatic infection.

Fever in a returned traveller requires prompt investigation to prevent deaths from malaria; diagnosis of malaria may require up to three blood films over 36–48 hours.

Diarrhoea is the most common health problem in travellers and is caused predominantly by bacteria; persistent diarrhoea is less likely to have an infectious cause, but its prognosis is usually good.

While most travel-related infections present within six months of return, some important chronic infections may present months or years later (eg, strongyloidiasis, schistosomiasis).

Travellers who have been bitten by an animal require evaluation for rabies prophylaxis.

Non-specific

• FBC, CRP, LFTs, other

Pathogen Detection

• Blood Cultures
• Other Cultures
• Antigen Detection
• DNA, RNA detection
• Protein detection
• Malaria slides (?1-2)

Indirect measure of Infection

• Serology

Imaging

• Typical, non-specific
4: Evaluation and initial management of fever in a returned traveller*

Suspected febrile illness in a returned traveller

Confirm fever

Severe sepsis
(confusion, collapse, cyanosis, tachypnoea, hypotension, neck stiffness, peritonism or digital gangrene)

No features of severe sepsis

Resuscitation if shocked
Blood cultures
Malaria films
Penicillin or ceftriaxone
(if meningococcal disease likely)

History: Travel and fever onset (compare with typical incubation periods)
Pattern of fever: Occasionally helpful (e.g., second-daily paroxysm in vivax malaria)
Focal features: Neck stiffness, cellulitis, abdominal tenderness, pulmonary consolidation
Investigations: Full blood count, liver function tests, blood cultures (two), chest x-ray, urine microscopy and culture, baseline serological tests, specific investigations for focal disease

Malaria possible†

Thick and thin malaria films (if initially negative, repeat 3 times)

Plasmodium vivax, ovale or malariae

Plasmodium falciparum

Urgent hospital transfer

Rash

Consider dengue or rickettsial disease
• Serological tests
• Consider empirical doxycycline for rickettsia

Respiratory symptoms

Return within 3 days from country with acute influenza

Consider unusual causes of pneumonia (e.g., Legionnaire’s disease, melioidosis)
• If severe, give empirical treatment, including macrolide or quinolone

Pulmonary consolidation

Fever >7 days, malaria ruled out

Consider enteric fever
• Blood, stool and urine cultures
• Consider empirical quinolone or third-generation cephalosporin

Jaundice

Consider
• Acute hepatitis (e.g., A, B, C, E, Epstein–Barr virus, dengue, Q fever): serological tests
• Acute cholangitis (stones, liver fluke): blood cultures, ultrasound and stool examination
• Liver abscess (amoebic, pyogenic): blood cultures, serological tests

PCR = polymerase chain reaction.
*Evaluation should also include the differential diagnoses that would be considered in a non-traveller with fever.
†Travel to high-risk area, rural or prolonged travel, non-compliance with prophylaxis.
Febrile Traveller - Treatment

• Get tests off - ?empiric treatment

• Likely diagnosis malaria
  – RDT -?Pf (NPV good), not so for others

• Others
  – ID - Intuition & Doxycycline (typhus, leptospirosis)
  – ?Typhoid – Azithromycin, Ceftriaxone
  – Dengue – viremia helps early diagnosis; Avoid Aspirin, NSAIDs

• Follow up tests including serology
Don’t be afraid to look it up!

"If you want a second opinion, I'll ask my computer."
No, please go on. I’m sure that your internet forum has access to more medical literature and has studied it more than I have.
Should I Stay?

or

Should I Go?
**Tafenoquine** – long acting 8 aminoquinoline
## Malaria – The Disease

Protozoan genus *Plasmodium*: 4–6 species infect humans:

<table>
<thead>
<tr>
<th>Species</th>
<th>Predominance</th>
<th>Disease</th>
<th>Prevalence globally</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>P. falciparum</em></td>
<td>Africa</td>
<td>Most severe</td>
<td>Highest</td>
<td>Highest mortality</td>
</tr>
<tr>
<td><em>P. vivax</em></td>
<td>Asia</td>
<td>Severe</td>
<td>High (esp. in Australian cases)</td>
<td>Relapse cycles</td>
</tr>
<tr>
<td><em>P. ovale</em></td>
<td>Africa</td>
<td>?Severe</td>
<td>Low</td>
<td>?Relapse cycles</td>
</tr>
<tr>
<td><em>P. malariae</em></td>
<td>Global</td>
<td>Mild</td>
<td>Low</td>
<td>Long dormant periods</td>
</tr>
<tr>
<td><em>P. knowlesi</em></td>
<td>South-East Asia</td>
<td>Severe</td>
<td>Normally found in macaques</td>
<td>Can infect humans</td>
</tr>
<tr>
<td><em>P. cynomolgi</em></td>
<td>Sarawak</td>
<td>?</td>
<td>Also simian malaria</td>
<td>Can infect humans</td>
</tr>
</tbody>
</table>

- Transmitted by bite of infected female *Anopheles* mosquitoes
- Differentiation is by molecular methods (PCR) – challenges old ideas
A systematic review of the clinical presentation, treatment and relapse characteristics of human *Plasmodium ovale* malaria

Mirjam Groger\(^1,2\), Hannah S. Fischer\(^1\), Luzia Veletzky\(^1,2\), Albert Lalremruata\(^3\) and Michael Ramharter\(^1,2,3\)*

**Abstract**

**Background:** Despite increased efforts to control and ultimately eradicate human malaria, *Plasmodium ovale* malaria is for the most part outside the focus of research or public health programmes. Importantly, the understanding of *P. ovale*—nowadays regarded as the two distinct species *P. ovale wallikeri* and *P. ovale curtisi*—largely stems from case reports and case series lacking study designs providing high quality evidence. Consequently, there is a lack of systematic evaluation of the clinical presentation, appropriate treatment and relapse characteristics of *P. ovale* malaria. The aim of this systematic review is to provide a systematic appraisal of the current evidence for severe manifestations, relapse characteristics and treatment options for human *P. ovale* malaria.

**Methods and results:** This systematic review was performed according to the PRISMA guidelines and registered in the international prospective register for systematic reviews (PROSPERO 2016:CRD42016039214). *P. ovale* mono-infection was a strict inclusion criterion. Of 3454 articles identified by the literature search, 33 articles published between 1922 and 2015 met the inclusion criteria. These articles did not include randomized controlled trials. Five prospective uncontrolled clinical trials were performed on a total of 58 participants. *P. ovale* was sensitive to all tested drugs within the follow-up periods and on interpretable in vitro assays. Since its first description in 1922, only 18 relapsing cases of *P. ovale* with a total of 28 relapse events were identified in the scientific literature. There was however no molecular evidence for a causal relationship between dormant liver stages and subsequent relapses. A total of 22 severe cases of *P. ovale* malaria were published out of which five were fatal. Additionally, two cases of congenital *P. ovale* malaria were reported.

**Conclusions:** Current knowledge of *P. ovale* malaria is based on small trials with minor impact, case reports and clinical observations. This systematic review highlights that *P. ovale* is capable of causing severe disease, severe congenital malaria and may even lead to death. Evidence for relapses in patients with *P. ovale* malaria adds up to only a handful of cases. Nearly 100 years after *P. ovale*’s first description by Stephens the evidence for the clinical characteristics, relapse potential and optimal treatments for *P. ovale* malaria is still scarce.

**Keywords:** *Plasmodium ovale*, Treatment evaluation, Relapse characteristics, Severe *Plasmodium ovale* malaria, Congenital *Plasmodium ovale* malaria
# Malaria – The disease

## Types of disease
- **Asymptomatic** malaria – circulating parasites, no illness

### UNCOMPPLICATED
- Attacks consisting of cold, hot, sweats stages
- Frequency of attacks depends on the parasite species

**General symptoms:**
- Fever
- Chills
- Sweats
- Headaches
- Nausea, vomiting, diarrhoea
- Body aches
- General malaise

**Other symptoms/signs:**
- Splenomegaly
- Mild jaundice
- Enlarged liver
- Increased respiratory rate

### SEVERE

**Beware intercurrent Gram negative sepsis**
- Infections complicated by serious organ failures or abnormalities
- Severe disease symptoms often are medical emergencies

**Manifestations include:**
- Cerebral malaria
- Abnormal behaviour
- Seizures
- Coma
- Severe anemia
- Hemoglobinuria
- Acute respiratory distress syndrome
- Low blood pressure due to cardiovascular collapse
- Acute kidney failure

**High risk groups to develop severe disease when infected:**
- Children
- Pregnant women
- Elderly
- Travellers

## Placental malaria – parasites in placenta > poor fetal +/- maternal outcome

Adapted from the Centers for Disease Control and Prevention (CDC) : Malaria Disease [http://www.cdc.gov/malaria/about/disease.html](http://www.cdc.gov/malaria/about/disease.html)
<table>
<thead>
<tr>
<th>Test</th>
<th>Limit of Detection* (Parasitaemia or Parasites/microlitre)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCR</td>
<td>0.00002% [1]</td>
<td>Most sensitive test</td>
</tr>
<tr>
<td>Microscopy (thick film)</td>
<td>0.0001% [5]</td>
<td>Current Gold Standard</td>
</tr>
<tr>
<td>Enzymeimmunoassay (EIA) for P.falciparum</td>
<td>0.0004% [20]</td>
<td>RDT is an adaptation of EIA but is less sensitive</td>
</tr>
<tr>
<td>Rapid Diagnostic Test</td>
<td>0.002% [100]</td>
<td>Patients may be symptomatic below this level of parasitaemia</td>
</tr>
</tbody>
</table>

*Approximate

References

Acquisition of anti-malaria immunity in a stable *P. falciparum* zone

Malaria (Pf) all year round kills children aged 6mths – 5yrs

www.malariaimpact.com
<table>
<thead>
<tr>
<th>MALARIA</th>
<th>STAY</th>
<th>GO</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.falciparum*</td>
<td>Complicated*</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>P.knowlesi*</td>
<td>Species uncertain</td>
<td>Species known</td>
</tr>
<tr>
<td>P.vivax*</td>
<td>Parasitemia &gt; 1%</td>
<td>Parasitemia &lt; 1%</td>
</tr>
<tr>
<td>P.ovale</td>
<td>Intercurrent illness e.g. Gram negative sepsis, other (PCT)</td>
<td></td>
</tr>
<tr>
<td>P.malariae</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consider</td>
<td>Vomiting</td>
<td>Keeps first dose down</td>
</tr>
<tr>
<td></td>
<td>No-one at home</td>
<td>Someone reliable at home</td>
</tr>
<tr>
<td></td>
<td>No regular GP</td>
<td>Has regular GP</td>
</tr>
<tr>
<td>Treatment</td>
<td>Artemether-lumefantrine</td>
<td>Artemether-lumefantrine</td>
</tr>
<tr>
<td></td>
<td>Atovaquone-Proguanil</td>
<td>Atovaquone-Proguanil</td>
</tr>
<tr>
<td></td>
<td>IV Artesunate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IV Quinine</td>
<td></td>
</tr>
<tr>
<td>Follow-Up</td>
<td>Till negative for asexual</td>
<td>Till negative for asexual</td>
</tr>
<tr>
<td></td>
<td>stages, then</td>
<td>stages, then</td>
</tr>
<tr>
<td></td>
<td>Pf, Pk weekly malaria films</td>
<td>Pf, Pk weekly malaria films</td>
</tr>
<tr>
<td></td>
<td>for 4 weeks</td>
<td>for 4 weeks</td>
</tr>
<tr>
<td>Eradication</td>
<td>P.vivax – primaquine or</td>
<td>P.vivax – primaquine or</td>
</tr>
<tr>
<td>(hypnozoites)</td>
<td>tafenoquine</td>
<td>tafenoquine</td>
</tr>
<tr>
<td></td>
<td>P.ovale – lacks data</td>
<td>P.ovale – lacks data</td>
</tr>
<tr>
<td>Gametocytocidal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Therapy</td>
<td></td>
<td></td>
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<tr>
<td>(Single Dose Primaquine)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parasite clearance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>time (PCT) for</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pfalciparum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Briand V. JTM 2007)</td>
<td></td>
<td>Age 15-64 years 37% could</td>
</tr>
<tr>
<td></td>
<td></td>
<td>have been treated as</td>
</tr>
<tr>
<td></td>
<td></td>
<td>outpatients</td>
</tr>
</tbody>
</table>
AUSTRALIA: MALARIA RECEPTIVE ZONE
NORTH OF 19° S
Dengue
Figure 2.1 The course of dengue illness

Days of illness

1 2 3 4 5 6 7 8 9 10

Temperature

Dehydration

Shock bleeding

Reabsorption

fluid overload

Organ impairment

Potential clinical issues

Laboratory changes

Hematocrit

Platelet

Viraemia

IgM/IgG

Serology and virology

Course of dengue illness:

Febrile

Critical

Recovery phases

1 2 3 4 5 6 7 8 9 14 days

3 months

Years

Isolation of virus

Detection of IgM

Detection of IgG

Onset of symptoms

WHO Dengue Monograph
Figure 1.4 Suggested dengue case classification and levels of severity

**DENGUE ± WARNING SIGNS**

- **with warning signs**
  - Probable dengue
    - live in /travel to dengue endemic area.
    - Fever and 2 of the following criteria:
      - Nausea, vomiting
      - Rash
      - Aches and pains
      - Tourniquet test positive
      - Leukopenia
      - Any warning sign
    - Laboratory-confirmed dengue
      - Important when no sign of plasma leakage

- **without warning signs**

**SEVERE DENGUE**

1. Severe plasma leakage
2. Severe haemorrhage
3. Severe organ impairment

**CRITERIA FOR DENGUE ± WARNING SIGNS**

- Warning signs*
  - Abdominal pain or tenderness
  - Persistent vomiting
  - Clinical fluid accumulation
  - Mucosal bleed
  - Lethargy, restlessness
  - Liver enlargement >2 cm
  - Laboratory: increase in HCT concurrent with rapid decrease in platelet count
  - *(requiring strict observation and medical intervention)*

**CRITERIA FOR SEVERE DENGUE**

- Severe plasma leakage leading to:
  - Shock (DSS)
  - Fluid accumulation with respiratory distress

- Severe bleeding as evaluated by clinician

- Severe organ involvement
  - Liver: AST or ALT >=1000
  - CNS: Impaired consciousness
  - Heart and other organs
Dengue Case Management

**Assessment**

**Presumptive Diagnosis:**
Live in / travel to endemic area plus Fever and two of the following:
- Anorexia and nausea
- Rash
- Aches and pains
- Warning signs
- Leucopenia
- Tourniquet test positive

**Warning signs:**
- Abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation
- Mucosal bleed
- Lethargy, restlessness
- Liver enlargement >2cm
- Laboratory: Increase in HCT concurrent with rapid decrease of platelet count

**Classification**

**Co-existing conditions**
- Social circumstances

**Dengue without warning signs**
- negative

**Dengue with warning signs**
- positive

**Severe Dengue**
- positive

**Group A**
May be sent home

- Patients who do not have warning signs AND who are able:
  - Tolerate adequate volumes of oral fluids
  - Pass urine at least once every 6 hours

**Group B**
Referred for in-hospital care

**Group C**
Require emergency treatment

**WHO Dengue Monograph 2012**
### Dengue without warning signs

**Group A**
- May be sent home
- **Group criteria**
  - Patients who do not have warning signs **AND**
  - who are able:
    - To tolerate adequate volumes of oral fluids
    - To pass urine at least once every 6 hours
- **Laboratory tests**
  - Full blood count (FBC)
  - Hæmatocrit (Hct)
- **Advice for**:
  - Adequate rest
  - Adequate fluid intake
  - Paracetamol, 4 grams max. per day in adults and accordingly in children
- Patients with stable Hct can be sent home

### Dengue with warning signs

**Group B**
- Referred for in-hospital care
- **Group criteria**
  - Patients with any of the following features:
    - C. existing conditions such as pregnancy, infancy, old age, diabetes mellitus
    - S. cial circumstances such as living alone, living far from hospital
- **Laboratory tests**
  - Full blood count (FBC)
  - Hæmatocrit (Hct)
  - Hæmatocrit (low)

### Severe Dengue

**Group C**
- Require emergency treatment
- **Group criteria**
  - Patients with any of the following features:
    - S. evere plasma leakage with shock and/or fluid accumulation with respiratory distress
    - S. evere bleeding
    - S. evere organ impairment
- **Laboratory tests**
  - Full blood count (FBC)
  - Hæmatocrit (Hct)

### Management

#### Treatment
- **Group A**
  - Encourage intake of oral fluids
  - If not tolerated, start intravenous fluid therapy
- **Group B**
  - Give isotonic solutions such as 0.9% saline, Ringer lactate, start with 5-7 ml/kg/hr for 1-2 hours, then reduce to 3-5 ml/kg/hr for 2-4 hours, and then reduce to 2-3 ml/kg/hr or less according to clinical response.
- **Group C**
  - Start I.V. fluid resuscitation with isotonic crystalloid solutions at 5-10 ml/kg/hr over 1 hour

### Treatment of compensated shock:
- **Group A**
  - If Hct remains the same or rises only minimally -> continue with 2-3 ml/kg/hr for another 4 hours
  - If worsening of vital signs and rapidly rising Hct -> increase rate to 5-10 ml/kg/hr for 1-2 hours

### Treatment of hypotensive shock:
- **Other**
  - Blood transfusion
  - Cross-matching
  - Other organ functions, renal profile, liver profile, coagulation profile, as indicated

### Discharge criteria:
- No fever for 48 hours
- Improvement in clinical picture
- No respiratory distress
- Stable haematocrit without intravenous fluids

---

**Notes:**
- Following an episode of severe dengue, patients are at high risk of recurrence.
- Patients should be discharged only when the Hct is below 30% and with no additional complications.
- The treatment should be adjusted according to the patient's condition and the clinical status.
- The guidelines should be followed closely to ensure the best possible outcome for patients.
Management of dengue in Australian travellers: a retrospective multicentre analysis

Alex YC Tai¹, Sarah L McGuinness², Roselle Robosa³, David Turner⁴, G. Khai Lin Huang², Karin Leder⁴,⁵, Tony M Korman³, Irani Thevarajan⁶, Andrew J Stewardson¹, Alexander A Padiglione³, Douglas F Johnson¹,⁶

The known The revised WHO guidelines (2009) are a helpful tool for managing patients with dengue, but among Australian medical practitioners there is a lack of awareness of the warning signs of severe dengue in travellers.

The new Two-fifths of Australian travellers hospitalised for dengue presented with warning signs of severe dengue. Many signs were unrecognised as such, and NSAIDs were prescribed for more than 20% of patients, exposing them to unnecessary risk.

The implications Australian clinicians should be familiar with the clinical manifestations of dengue, especially of dengue with warning signs, and with its management.

Objective: To describe the epidemiology, clinical and laboratory features and outcomes of dengue in returned Australian travellers, applying the revised WHO dengue classification (2009) to this population.

Design, setting and participants: Retrospective case series analysis of confirmed dengue cases hospitalised at one of four Australian tertiary hospitals, January 2012 — May 2015.

Main outcome measures: Clinical features, laboratory findings and outcomes of patients with dengue; dengue classification according to 2009 WHO guidelines.

Results: 208 hospitalised patients (median age, 32 years; range, 4–76 years) were included in the study. Dengue was most frequently acquired in Indonesia (94, 45%) and Thailand (40, 19%). The most common clinical features were fever (98% of patients) and headache (76%). 84 patients (40%) met the WHO criteria for dengue with warning signs, and one the criteria for severe dengue; the most common warning signs were mucosal bleeding (45, 22% of all patients) and abdominal pain (43, 21%). Leukopenia (176 patients, 85%), thrombocytopenia (133, 54%), and elevated liver enzyme levels (154, 76%) were the most common laboratory findings. 46 patients (22%) had serological evidence of previous exposure to dengue virus. WHO guidelines were documented as a management benchmark in ten cases (5%); 46 patients (22%) received non-steroidal anti-inflammatory drugs (NSAIDs).

Conclusions: A significant proportion of returning Australian travellers hospitalised for dengue have unrecognised warning signs of severe disease. Many received NSAIDs, which can increase the risk of haemorrhage in dengue. As travel to Asia from Australia continues to increase, it is vital for averting serious outcomes that clinicians can recognise and manage dengue.
Skin Things
Skin Lesions in Returned Travellers

- Myiasis, jiggers, mites, lice
- “Creeping eruption”
- Cutaneous leishmaniasis
- Pyoderma, stings, “burns”
- Systemic infections
Skin Lesions in Travellers - Tests

- “Spot” diagnosis
- Endemic diseases
- Biopsy
- Special methods
- Other: blood, stools, urine, CXR, imaging, ask for advice!
Jigger – Egg Laden Flea
Jigger – Egg Sac Broken
Jigger – Cleaned Out
Jiggers

- Jiggers
  - Female flea – “Tunga penetrans”
  - Penetrates skin
  - Takes blood from venules
  - Spiracles outside skin
  - Eggs develop & painful lesion develops
  - Risk of skin infection & tetanus
  - Removal with skill and a Swiss Army Penknife
Myiasis

- Cutaneous - Congo Floor Maggot
- “furuncular myiasis”
  - Screw worms (*Cordylobia spp*)
  - Bot fly (*Dermatobia hominis*)
- Dermal “creeping eruption”
  - horse & cattle bot fly larvae
Creeping eruption - DDx

- C.larva migrans - more persistent
  - Ancylostoma

- Transient
  - Strongyloides (larva currens)
  - Gnathostomiasis
  - Fasciola
Gnathostomiasis
Gnathostomiasis, Another Emerging Imported Disease

Joanna S. Herman¹* and Peter L. Chiodini¹,²

Department of Clinical Parasitology, Hospital for Tropical Diseases, 3rd Floor, Mortimer Market, Capper Street, London WC1E 6JB, United Kingdom,¹ and London School of Hygiene and Tropical Medicine, London, United Kingdom²

INTRODUCTION ................................................................. 484
EPIDEMIOLOGY ............................................................... 484
LIFE CYCLE AND MORPHOLOGY .................................. 485
CLINICAL FEATURES ...................................................... 486
Cutaneous Gnathostomiasis ........................................... 487
Visceral Disease ............................................................. 488
  Pulmonary manifestations ........................................ 488
  Gastrointestinal manifestations ............................... 488
  Genitourinary manifestations ................................. 488
  Ocular ............................................................... 488
  Auricular manifestations .................................... 488
  CNS manifestations .............................................. 488
DIAGNOSIS ................................................................. 489
TREATMENT ................................................................. 490
PREVENTION ............................................................... 491
ACKNOWLEDGMENTS ................................................... 491
REFERENCES ............................................................... 491
Gnathostomiasis

Cutaneous
Ocular
Visceral
Neurological

Gnathostoma spinigerum
Domestic and wild felines and canines are the definitive hosts.

Gnathostoma hispidum
Domestic and wild pigs are the definitive host.

Embryonated egg
Egg hatches and releases L1 larva.

First intermediate host
L1 develops into L2.

Copepod
L2 develops into L3.

Second intermediate host
Infected second intermediate host ingested by definitive host.

Paratenic host
L3 develop into adult worms.

Unembryonated egg

CDC
http://www.dpd.cdc.gov/dpdx
Safer, Healthier, People™
Gnathostomiasis Distribution

Regions with Endemic *Gnathostomiasis spinigerum*
(Adapted from information in Rusnak and Lucey 1993)
FIG. 1. Map of countries with reported acquisition of gnathostomiasis.
FIG. 3. Photograph of a third-stage larva of *Gnathostoma spinigerum*, showing the entire larva (A) and the head with hooks (B).
FIG. 4. Photograph showing cutaneous larva migrans due to *Gnathostoma spinigerum* on the forehead (A) and shoulder (B). (Reprinted from reference 17.)
Eosinophilic meningitis can be the result of noninfectious causes and infectious agents. Among the infectious agents, *Angiostrongylus cantonensis* and *Gnathostoma spinigerum* are the most common. Although angiostrongyliasis and gnathostomiasis are not common in the United States, international travel and immigration make these diseases clinically relevant. Both *A. cantonensis* and *G. spinigerum* infection can present as severe CNS compromise. Diagnoses of both infections can be challenging and are often clinical because of a paucity of serological assays readily available in the United States. Furthermore, there are conflicting recommendations about treatment for angiostrongyliasis and gnathostomiasis. To further explore the emerging nature of these helminthic infections, a case description and review of *A. cantonensis* and *G. spinigerum* infections are presented. The clinical severity of eosinophilic meningitis and diagnosis of these infections are highlighted.
Table 1. Comparison of features of *Angiostrongylus* and *Gnathostoma* infection with the case description.

<table>
<thead>
<tr>
<th>Variable</th>
<th><em>Angiostrongylus cantonensis</em></th>
<th><em>Gnathostoma spinigerum</em></th>
<th>Case description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure</td>
<td>Consumption of contaminated crustaceans, mollusks, prawns, crabs,</td>
<td>Consumption of contaminated poultry or fish</td>
<td>Consumption of seafood and contaminated water; contact with mosquitoes and bats</td>
</tr>
<tr>
<td></td>
<td>frogs, and/or vegetables; contact with rats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Geographical</td>
<td>South Pacific, Australia, Africa, the Caribbean, Hawaii, and</td>
<td>Southeast Asia, Japan, China, Mexico, Central and South</td>
<td>Fiji, Vanuatu, and Thailand</td>
</tr>
<tr>
<td>distribution</td>
<td>Louisiana</td>
<td>America, Africa, and the Middle East</td>
<td></td>
</tr>
<tr>
<td>Onset of</td>
<td>Days to ~1 month</td>
<td>Days to months</td>
<td>First symptoms 3 days after return to United States</td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of</td>
<td>A few months</td>
<td>10–13 years</td>
<td>&gt;5 months</td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical features</td>
<td>Headaches, photophobia, stiff neck, vomiting, paraesthesias/hyperesthesia, fever, cranial nerve 8 palsy, and ocular involvement</td>
<td>Cutaneous: migrating, panniculitis, eruptions, and pseudofurunculosis; visceral: any organ; CNS: radiculomyelitis, encephalitis, paralysis, hemorrhage, and cranial nerve/ocular involvement</td>
<td>Cutaneous: nonerythematous, nonmigrating, tender nodules; systemic: self-limited fever; CNS: headache, photophobia, and paraesthesias/hyperesthesia</td>
</tr>
<tr>
<td>Laboratory</td>
<td>Peripheral eosinophilia, CSF: involvement, elevated open pressure, elevated WBC count, eosinophilia, elevated protein level, and normal glucose level</td>
<td>Peripheral eosinophilia, CSF: xanthochromia, elevated open pressure, elevated WBC count, eosinophilia, elevated protein level, and normal glucose level</td>
<td>Peripheral eosinophilia, CSF: clear (2 weeks after return to United States); open pressure, 49 mm water; WBC count, 754 cells/μL; 44% eosinophils; protein level, 110 mg/dL; and glucose level, 42mg/dL</td>
</tr>
<tr>
<td>features</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imaging</td>
<td>Head MRI and CT findings usually normal</td>
<td>Head CT and MRI findings: hemorrhage and hydrocephalus</td>
<td>Head and neck MRI findings normal; extremity MRI findings normal</td>
</tr>
<tr>
<td>Serological</td>
<td>Immunoblot</td>
<td>Immunoblot</td>
<td>CSF and/or serum immunoblot assay positive for <em>Angiostrongylus</em> species</td>
</tr>
<tr>
<td>assays</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Serial lumbar punctures and analgesics; consider steroids and antihelminthics</td>
<td>Cutaneous: albendazole, ivermectin; CNS infection: supportive therapy (consider steroids); no evidence for antihelminthics</td>
<td>Serial lumbar puncture, analgesics, steroids, and 28-day course of albendazole</td>
</tr>
</tbody>
</table>
Gnathostomiasis Diagnosis

• Clinical, history of exposure
• Histopathology
• Serology - Immunoblot
  – Specific 24-kDa band
  – Not available in Australia
  – Mahidol, Swiss Tropical Institute, London Hospital for Tropical Diseases
FIG. 6. (A) Hematoxylin- and eosin-stained cross-section of a *Gnathostoma* organism taken from a subcutaneous nodule above the right breast of a patient, showing the esophagus. Note the presence of cuticular spines (arrow). (B) Another hematoxylin- and eosin-stained cross-section of a *Gnathostoma* organism, taken from the same specimen as for panel A, showing the intestinal cells and characteristic large lateral chords (LC). (Panel A is from Diagnostix Pathology Laboratories LTD and the CDC-DPDx and panel B is from the CDC-DPDx [www.dpd.cdc.gov/dpdx/HTML/gnathostomiasis.htm].)
TREATMENT OF CUTANEOUS GNATHOSTOMIASIS WITH IVERMECTIN

KANYARAT KRAIVICHIAN, SURANG NUCHPRAYOON, PRASERT SITICHALERNCHAI, WANPEN CHAICUMPA, AND SUTIN YENTAKAM

Department of Parasitology, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand; Faculty of Allied Health Science, Thammasart University, Pathumthani, Thailand

Abstract. In a randomized open study, we compared the efficacy of a single dose of oral ivermectin (200 μg/kg) and oral albendazole (400 mg/day for 21 days) for the treatment of cutaneous gnathostomiasis. Thirty-one patients were randomly assigned to receive ivermectin (n = 17) or albendazole (n = 14). Thirteen of 17 patients who received ivermectin responded, 3 relapsed, and 1 was unresponsive (cure rate = 76%). Thirteen of 14 patients who received albendazole responded very well and did not relapse. Only one patient was unresponsive (cure rate = 92%; P > 0.05). No major side effects were observed in both groups. We concluded that a single dose of ivermectin (200 μg/kg) is less effective than albendazole (400 mg/day for 21 days) for treatment of cutaneous gnathostomiasis, but there was no statistically significant difference (P > 0.05).
Gnathostomiasis Outcome

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Long-term Follow-up of Imported Gnathostomiasis Shows Frequent Treatment Failure

Christophe Strady,* Paron Dekumyoy, Marina Clement-Rigolet, Martin Danis, François Bricaire, and Eric Caumes
Department of infectious and tropical diseases, la Pitié Salpêtrière Hospital, France; Department of Helminthology, Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand

Abstract. Gnathostomiasis is increasingly reported among travelers returning from endemic areas. Between 2000 and 2004, thirteen patients were diagnosed with imported gnathostomiasis and followed for at least 6 months after treatment. Nine patients presented with cutaneous signs, two with gastrointestinal signs, and two with neurological signs. The median age was 38 years and the female/male sex ratio was 1.6. The patients had visited South East Asia or Central America. The median interval between symptom onset and treatment (with albendazole in 12 cases and ivermectin in one case) was 3.5 months. Post-treatment follow-up lasted a median of 15 months. Eight patients relapsed, a median of 2 months (1–7 months) after initial treatment. These eight patients had a total of 13 relapses, the last occurring a median of 16 months (2–26 months) after initial treatment. Thus patients with imported gnathostomiasis should be monitored for at least 6 months to detect late treatment failure.
Cutaneous Larva Migrans - Rx

• Oral therapies
  – Albendazole 400mg bd 5 days
  – Ivermectin (200mcg/kg)

• Topical therapies
  – Thiabendazole (?others) paste
  – Ointment 3x/day 5 or more days (eg steroid ointment)
  – Freezing
Cutaneous larva migrans caused by the larvae of animal hookworms is the most frequent skin disease among travelers returning from tropical countries. Complications (impetigo and allergic reactions), together with the intense pruritus and the significant duration of the disease, make treatment mandatory. Freezing the leading edge of the skin track rarely works. Topical treatment of the affected area with 10%-15% thiabendazole solution or ointment has limited value for multiple lesions and hookworm folliculitis, and requires applications 3 times a day for at least 15 days. Oral thiabendazole is poorly effective when given as a single dose (cure rate, 68%-84%) and is less well tolerated than either albendazole or ivermectin. Treatment with a single 400-mg oral dose of albendazole gives cure rates of 46%-100%; a single 12-mg oral dose of ivermectin gives cure rates of 81%-100%.
Cutaneous Larva Migrans – Rx

if NOT Hookworm

• Strongyloidiasis
  – Albendazole 400mg bd 10 days
  – Ivermectin (200mcg/kg/d) 2 days
    • Repeat in 2 weeks

• Gnathostomiasis
  – Albendazole 400mg bd 21 days
  – Ivermectin (200mcg/kg/d) 2 days
Cutaneous Leishmaniasis
Cutaneous Leishmaniasis

• Bites of “true” sandflies
• Mediterranean, Asia, Africa
• Jungle areas of South & Central America
• Biopsy - pathology, culture
Cutaneous Leishmaniasis - Rx

• Intraleisional Pentostam injection
• Oral Azoles (Itra/fluconazole)
  – 200mg/d Fluconazole 6 weeks
• Antimony compounds (some resistance)
  – IMI/IVI Pentostam 21-28 days
  – IVI Pentamididine 21-28 days
  – NOT with amphotericin
• Oral Miltefosine 2.5mg/kg/d 28 days
• IVI Amphotericin 1mg/kg/d 30 doses
• IVI Liposomal Amphotericin 10mg/kg 2 doses!)
Worms - Miscellaneous
I found a worm in the toilet !!!

• Long white/grey worm in the toilet bowl
  – Most likely *Ascaris lumbricoides*

• Flat segment, moves like a leech
  – Tapeworm (beef or pork)
  – *Taenia saginata* or *Taenia solium*
  – Other

• Not a human pathogen?
  – Send to laboratory for identification
Figure 1. Wood print depicting a man passing a strobila of a broad tapeworm. The caption (not shown) said, “The man ate masu salmon. After a time, a strange object emerged from the anus and was pulled out: it turned out to be 2–3 m long.” From Shinsen Yamaino Soushi, by Daizennosuke Koan (1850).
The first confirmed case of *Diphyllobothrium latum* in Brazil

FLN Santos*/*, LB de Faro

Setor de Parasitologia, Centro de Medicina Laboratorial, Av. Antônio Carlos Magalhães 4009, sala 5, 40280-000 Salvador, BA, Brasil  *Centro de Pesquisa Gonçalo Moniz-Fiocruz, Salvador, BA, Brasil

Diphyllobothriasis is an infection of the small intestine by the broad tapeworm *Diphyllobothrium* sp. The associated symptomatology is nonspecific, but megaloblastic anemia is a well-described complication. Although the infection is common in temperate regions, descriptions in South America have so far been limited to Chile, Peru, and a few cases in Argentina. This paper presents the first confirmed Brazilian case of diphyllobothriasis. A 29-years-old woman living in Salvador (state of Bahia) apparently acquired the infection from eating sushi. The diagnosis was based on fecal examination that revealed a large quantity of operculated eggs. A single dose of praziquantel (600 mg) was sufficient to cure the infection.

US salmon may carry Japanese tapeworm, scientists say

By Susan Scutti, CNN

† Updated 1707 GMT (0107 HKT) January 11, 2017
Prevention
• “Boil it, cook it, peel it, or .. ..........forget it”

Alternative aphorism
• “Don’t get HIT”
• “Don’t get LIT (drunk/drug affected)”
• “Don’t get BIT” and ....
• “Don’t eat SHIT”