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TRAVEL MEDICINE

CURRENT ISSUES

CONTENT

TRAVEL MEDICINE

Travel medicine is becoming increasingly important within the field of tropical medicine. This is not only because of the increase in the number of people travelling abroad for business or leisure but also because travel destinations have become more exotic and far off and within reach of many. While in many countries excellent pre-travel advice can be obtained, including vaccinations and malaria prophylaxis, many travellers are not well prepared and put themselves at risk. In particular, patients who are immunosuppressed because of their illness or the medication they use are at increased risk and need counselling in specialized centres.

Other conditions are coming closer to home as vectors such as the *Aedes aegypti* mosquito, which can transmit West Nile, Dengue and Chikungunya viruses, have spread to previously non-endemic regions such as southern Europe because of global warming. Mass migration is another important factor, leading to concerns over tuberculosis importation and the spread of antimicrobial resistance. A relatively new category of vulnerable people are Visiting Friends and Relatives (VFRs); these are immigrants who have settled in for instance the Netherlands, and travel back to their home countries for short visits. These travellers rarely seek pre-travel advice and are at risk of malaria or other infectious diseases, as they are not aware that their immunity diminishes over time.

Increased air travel poses another threat, as it facilitates the rapid spread of communicable diseases to any part of the world. Clearly, viral haemorrhagic fevers cause great concern, but these are fortunately uncommon as an imported disease. In the last 25 years, three patients were diagnosed or treated in the Netherlands (one with Marburg fever, one with Lassa fever and one with confirmed Ebola), and 15 patients were admitted suspected of Ebola in 2014-2015 (Leo Visser, personal communication). The recent Ebola outbreak as well as other haemorrhagic fevers are likely to recur as the result of failing public health systems in endemic areas.

While old enemies such as malaria are still around and should be considered in any traveller returning from the tropics with fever, the landscape has changed in the Netherlands and probably in other countries too. One contentious issue is whether it is safe to replace malaria prophylaxis by standby medication for reasons of convenience but also to avoid side-effects.

In contrast, Zika virus infection, which until recently few people had ever heard of, is now a hot topic in the news, causing major concern worldwide. The World Health Organization has been criticized for its handling of the Ebola crisis last year. Zika virus infection is usually not a cause for major concern in terms of the illness itself, but the association with microcephaly in newborns is extremely worrying. Women who are pregnant (or want to become pregnant), and wish to travel to South America or the Caribbean region, are advised to carefully weigh the risks and take the necessary precautions. The economic impact of this outbreak is enormous, and it may affect spectatorship at the Olympic Games, which are coming up in Brazil this summer.

Travel medicine has become an important medical specialty, but it is not the exclusive domain of a few specialized centres: every doctor who sees a patient with symptoms that cannot be immediately explained should ask:

'WHERE HAVE YOU TRAVELLED RECENTLY?'

JAN AUKE DIJKSTRA, ED ZIJLSTRA

Infectious diseases among western travellers and migrants

PHOTO RADIOKAFKA / SHUTTERSTOCK

PHOTO SHUTTERSTOCK

Calcutta, India 2013

In the world's history, migration has been a prominent feature of humans travelling in search of food, escaping inhospitable climatic conditions, and in response to hardships of war, famine, social injustice and poverty. At the end of the 19th and the beginning of the 20th century alone, 60 million people left Europe seeking better lives. Forced migration is still a reality and occurs from and within all continents. As of now, a future in Europe is sought after by unprecedented numbers of refugees from especially Syria and the Horn of Africa, whereas since many decades, Northern America

has experienced a continuous influx of people from Mexico and other parts of Latin America but also from Asia. The health problems of these migrants and refugee populations depend to a large extent on the region of origin and the challenges encountered while travelling. On the other hand, immigration to western countries results in migrants moving back and forth, visiting friends and relatives ('VFRs'), which subsequently leads to import of infectious diseases from the country of origin.

In contrast to migration, which is usually for economic and/or political reasons, tourism is associated with different health risks compared with migrant populations. In the past decades, international travel has shown a marked increase. In 1950 approximately 25 million people from western countries travelled abroad as tourists.

CURRENTLY, EACH YEAR AN ESTIMATED 50 MILLION TRAVELLERS FROM WESTERN COUNTRIES VISIT TROPICAL AREAS OF THE WORLD, AND THESE NUMBERS ARE RAPIDLY GROWING.[1]

Improvements in transportation, changing world economies, increased political stability, the development of tourism as an industry, and increases in travel for business, health related issues and education contribute to this growth.

TRENDS

Impressive examples from history, such as Stanley's trans-Africa expedition around 1870 and the French attempt to construct the Panama Canal around the same time, illustrate the heavy toll on Western travellers taken by malaria, sleeping-sickness, yellow fever and other conditions. Even in the 21st century, despite proper preventive measures whether used appropriately or not, Western travellers may contract tropical diseases that, if left untreated, can be fatal within the first few weeks of onset of symptoms. Given the potentially serious consequences for the patients and, in some cases, their close contacts and healthcare workers, it is important that life-threatening tropical diseases are diagnosed quickly.

Travel, as well as the extensive worldwide migration flows, plays a major role in the globalization of infections. Dengue, chikungunya and the currently fast spreading epidemic of Zika virus infection in the Caribbean and Central and South America, are prominent examples. Transmission of those three diseases takes place through bites by *Aedes* mosquitoes, which are present in all tropical and subtropical regions; except for personal protection with repellents and insecticides, no effective preventive measures exist. The emergence of arbovirus infections in southern Europe and the subsequent autochthonous transmission during the summer (when the vector is active) are increasingly being reported. Northern Europeans visiting the Mediterranean basin are at risk, not only because of these new intruders but also due to endemic vector-borne diseases, such as infections caused by viruses spread by sand flies (e.g. Toscana virus infection), leishmaniasis, rickettsial illnesses and emerging threats such as *Plasmodium vivax* malaria and Crimean Congo haemorrhagic fever. The latter disease is still a rarity in most of Europe, but it is endemic in the most south-eastern tip of Europe.

Highly infectious and easily transmitted diseases, for example airborne conditions such as SARS and MERS CoV or viral haemorrhagic fevers such as Lassa fever or Ebola virus disease, pose an enormous local threat and account for many victims in the respective endemic areas. The frequent travel from these areas to non-endemic countries requires immediate countermeasures so as to prevent their introduction in areas that have so far not been affected and to reduce the chance of further man-to-man transmission.

SURVEILLANCE

Geosentinel is an organisation that has approximately thirty specialized travel medicine clinics on six continents that contribute to clinician-based sentinel surveillance data on travel-related diseases among travellers who became ill after visiting high-risk countries This initiative has enabled monitoring of trends in the occurrence of travel-related illnesses.[2] EuroTravNet, a daughter network of Geosentinel, generates these data from 18 European sites, facilitating rapid communication of detected disease outbreaks among European travellers.

The 2006 Geosentinel report showed that more than 17,000 travellers returned ill from (sub)tropical countries, with significant regional differences in morbidity in most syndromic categories.[2] Systemic febrile illness without clear focus occurred disproportionately among those returning from sub-Saharan Africa or Southeast Asia, while acute diarrhoea was most prevalent among those returning from south central Asia and dermatological problems among travellers returning from the Caribbean or Central or South America. Overall, malaria was the most frequent cause of systemic illness among ill travellers returning from the tropics, predominantly from sub-Saharan Africa, whereas dengue was the most frequent condition for those who visited the Caribbean, South America and Southeast Asia. Typhoid fever was a primary contributor for systemic febrile illness among travellers returning from south central Asia. Rickettsial infection, primarily tick-borne spotted fever, was seen among travellers from sub-Saharan Africa more often than typhoid fever or dengue. Travellers from all regions except Southeast Asia presented more often with parasite-related induced gastrointestinal complaints (overall most frequently giardiasis) than with bacterial diarrhoea. Insect bites were the most common cause of dermatologic problems, followed by cutaneous larva migrans, allergic reactions, and bacterial skin infections including skin abscesses; again with regional differences. Cutaneous leishmaniasis, which is not uncommon, was found mostly

among patients who had travelled to Latin America.

Another study based on Geosentinel data showed that falciparum malaria, mainly from West Africa, was by far the most acute and life-threatening disease in Western travellers, followed by typhoid and paratyphoid fever (south central Asia) and in a minority of cases, but with no less serious a course, leptospirosis and rickettsial diseases.[3]

EuroTravNet travel-associated infection data from the period 2008-2012 show similar results.[4] In its 5-year analysis, increases in vector-borne disease were noticed, particularly falciparum malaria, dengue, and a widening geographic range of acquisition of chikungunya. Acute bacterial and parasitic diarrhoeal illnesses caused high morbidity, similar in magnitude to malaria. Dermatological diagnoses increased over the years, especially of insect bites and animal-related injuries such as dog bites, which may cause the transmission of rabies, while respiratory infection showed an increasing trend, mainly due to the influenza H1N1 pandemic of 2009. Migration was associated with infection of hepatitis B and C, and tuberculosis. Chronic Chagas disease which may be imported by migrants from South America to Europe (mainly Spain) and to North America may be underestimated as it has not only serious consequences for the patients themselves but also for any blood products donated by them, which calls for appropriate screening.[5]

WHAT MORE CAN WE EXPECT?

The above reports and the database are an important tool in travel medicine and are instrumental in designing measures in case of outbreaks, but they do not represent a comprehensive epidemiologic analysis of all illnesses in all travellers. Neither do they provide a representative sample of illnesses in returned travellers, such as those seen at non-specialized primary care centres, where they usually present with mild or self-limited conditions. Diseases with short incubation periods or diseases that seemed not so serious when the person was still travelling are another cause of under-reporting.

Communicable diseases, which often present themselves unexpectedly, will continue to emerge and re-emerge.[6] New infections will emerge, changing patterns in host immunity will occur, and changes in climate and migration will influence their epidemiology. This requires continuous and accurate surveillance so as to reduce the unnecessary burden of morbidity and mortality. Appropriate pre-travel advice and intervention strategies based on the continuum of these surveillance data will help to reduce this burden.

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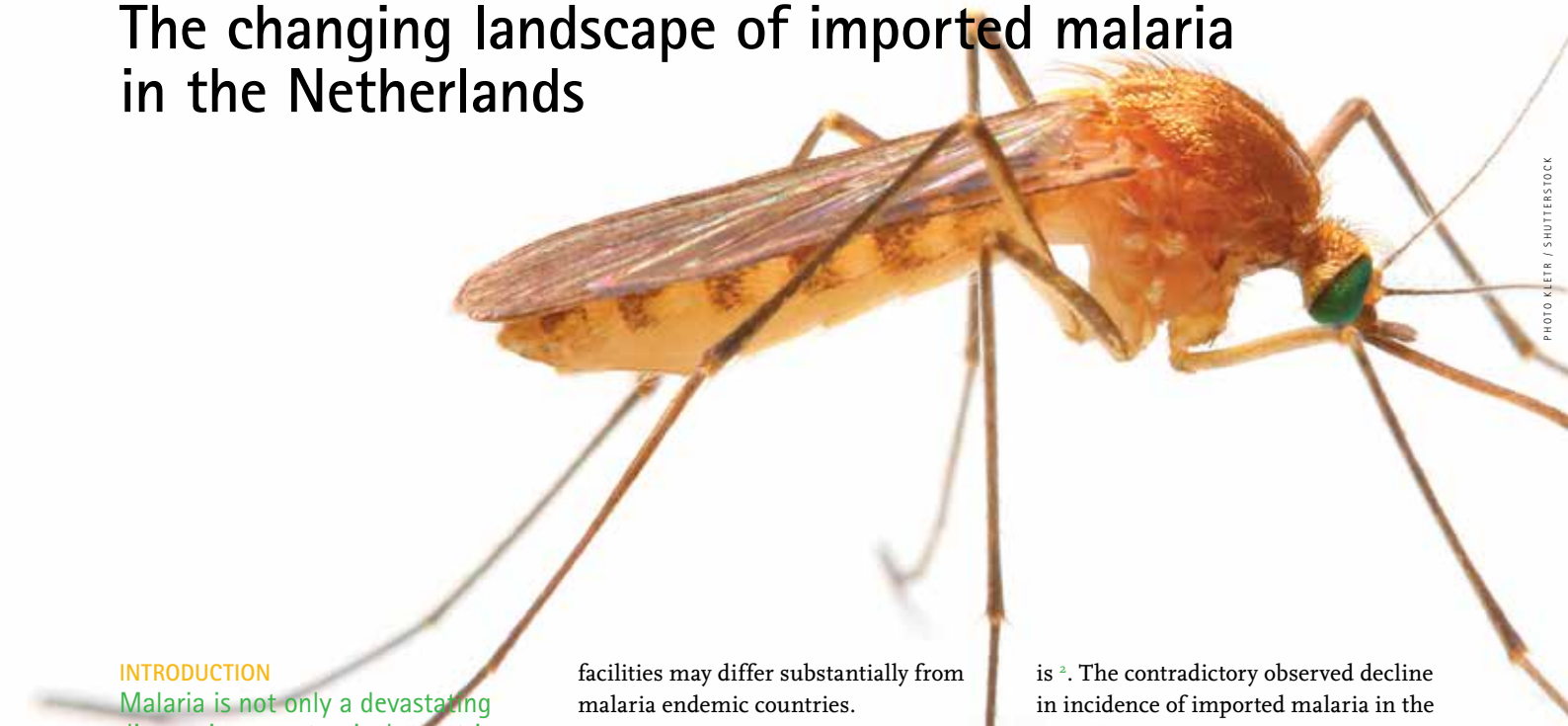
The changing landscape of imported malaria in the Netherlands



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INTRODUCTION

Malaria is not only a devastating disease in many tropical countries, it may also run a complicated course with significant morbidity and even fatalities in non-immune travellers. Most general hospitals in non-endemic countries have limited experience with malaria due to the relatively small number of malaria cases seen in returning travellers. This lack of experience may lead to a substantial delay in making an accurate diagnosis and timely treatment of malaria. This review provides a brief overview of imported malaria in the Netherlands and its changing landscape in terms of epidemiology, diagnosis, treatment and prevention.

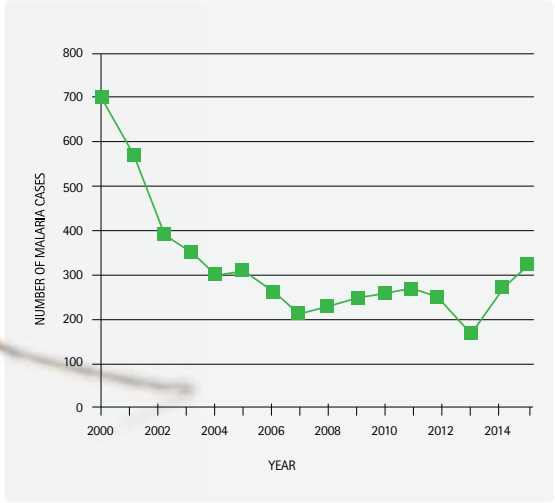
Although the prevalence of malaria is declining worldwide, it is still a devastating disease in many tropical and subtropical areas. According to the World Malaria Report 2015 of the World Health Organisation (WHO), the estimated death toll of malaria worldwide approximated 438,000 deaths in 2014, most of them occurring in children under the age of five and pregnant women in sub-Sahara Africa.[1] Malaria may also occur in non-endemic countries as an imported disease, but clinical manifestation, disease course, and access to adequate diagnostic and treatment facilities may differ substantially from malaria endemic countries.

EPIDEMIOLOGY OF IMPORTED MALARIA: DECLINING INCIDENCE AND CHANGING TRAVELLER RISK PROFILE

Compared to malaria endemic countries, the prevalence of malaria in travellers in the Netherlands is very low, and a significant decrease of incidence has been observed over the past two decades (Figure 1), stabilizing at a plateau phase with a yearly 200 to 300 new cases. *Plasmodium falciparum* remains the most prevalent cause of malaria in travellers accounting for approximately 75% of the cases in the Netherlands, whereas *P. vivax* accounts for approximately 20% of the cases and the other *Plasmodium* species, *P. ovale*, *P. malariae* and the zoonotic monkey malaria *P. knowlesi*, occur relatively infrequently. The majority of *P. falciparum* infections in travellers are acquired in sub-Saharan Africa, whereas *P. vivax* infections in travellers are usually acquired in South-East Asia. In contrast to malaria endemic areas, the clinical picture of imported malaria in the Netherlands is dominated by its occurrence in adults with a clear preponderance for males. Even though effective drugs are available for chemoprophylaxis of malaria and an increasing number of travellers visit malaria-endemic regions, unprotected travellers increasingly outnumber travellers who are prescribed malaria chemoprophylaxis [2]. The contradictory observed decline in incidence of imported malaria in the Netherlands over the past decade was therefore linked to a decreased transmission risk of malaria at the target destination of the travellers. The influx of large numbers of refugees into the Netherlands may also influence the statistics of imported malaria. For example, the observed rise in the number of imported malaria cases in 2014 and 2015 was for the bigger part caused by an increased number of *P. vivax* cases diagnosed in Eritrean refugees.

Additionally, interesting changes have been observed in the travel profile of patients with imported malaria. Where in the past decades cases were almost exclusively seen in non-immune tourists, nowadays Visiting Friends and Relatives (VFRs) dominate the risk profile of imported malaria. VFRs are defined as immigrants, ethnically and racially distinct from the country of residence, who return to their homeland to Visit Friends and/or Relatives. Of note is that the VFR can be either an immigrant who was born in the destination country ('immigrant VFR') or someone not born in the destination country but with close ties, such as an immigrant's child or other close family member ('traveller VFR'). In general, VFRs seek far less pre-travel health advice than tourists or business travellers, frequently associated with insufficient awareness of the

Figure 1 Number of reported malaria cases in the Netherlands from 2000 until 2014



Source of data: Infectious Diseases Bulletin, National Institute for Public Health and the Environment, RIVM, Bilthoven, the Netherlands.

importance of health advice for travel, with the high costs of both consultation and personal protective measures, and with misconceptions about their own presumed immunity towards malaria combined with longer duration of exposure to malaria. VFRs are therefore at risk of contracting malaria again after visiting their homeland because their presumed immunity wanes in the absence of repeated exposure as is the case during their stay in The Netherlands. Even though the disease generally runs a less fulminant course as compared to naïve patients, cases of severe malaria and even fatal disease have also been described in VFRs. Known risk factors for severe malaria in travellers are non-use or inappropriate use of chemoprophylaxis, older age, delay in seeking care or diagnosis, incorrect treatment and non-immunity.[3]

DIAGNOSIS OF IMPORTED MALARIA: THE ADVANCE OF RAPID DIAGNOSTIC TESTS FOR MALARIA

As malaria cannot be diagnosed on clinical grounds, tests are always required to make a proper diagnosis. Most standard blood parameters are non-specific for malaria, but thrombocytopenia, anaemia and elevated plasma LDH levels are frequently seen in malaria patients. In addition, increased plasma lactate was shown to be a useful predictor for disease severity in *P. falciparum malaria*.[4] Microscopic examination of thin and thick blood smears for malaria parasites is still considered the gold standard but increasingly rapid diagnostic tests (RDTs) are used as initial screening method. However, there are a number of important drawbacks to consider with the use of RDTs to diagnose malaria. First, RDTs may result in false-negative results in a substantial number of non-falciparum *Plasmodium* infections (including *P. knowlesi*) or in low-grade *P. falciparum* infections. Second, a positive RDT test result should be confirmed to exclude a false-positive test result, but also to determine the causative *Plasmodium* species and to determine the parasite load in order to assess disease severity.[5] Third, false-negative RDT results may occur in certain genetic polymorphisms of Histidine Rich Protein 2 (HRP-2) variants geographically confined to the Asia-Pacific region and in *P. falciparum* isolates from South America lacking HRP-2. In addition, in some RDTs, false-negative test results may occur at high *P. falciparum* loads due to a so-called prozone effect, defined as false-negative or false-low results in immunological reactions due to an excess of either antigens or antibodies. Finally, it should also be borne in mind that reactivity of the *P. falciparum*-specific protein HRP-2 band together with reactivity of the pan-plasmodium LDH or aldolase band is not only indicative of a monoparasitic *P. falciparum* infection but may also be a feature of a mixed *Plasmodium* infection (for reference, about 0.7% of all *Plasmodium* infections in The Netherlands were attributable to mixtures of species, mostly involving *P. falciparum*).

In addition to RDTs and microscopic examination of stained blood smears, several other sensitive methods are available in health care centres specialized in tropical diseases, such as Quantitative Buffy Coat (QBC) analysis and real-time quantitative PCR. These techniques are not widely used, as QBC analysis requires well-trained technicians and a costly fluorescent microscope and PCR is still a relatively time-consuming method.

TREATMENT OF IMPORTED MALARIA: THE ADVANCE OF ARTEMISININ-BASED TREATMENT REGIMENS

In its essence, the treatment of *P. falciparum* malaria and non-*P. falciparum* malaria does not differ between endemic and non-endemic countries. In the past decade, the artemisinin-combination therapy (ACT) acquired a dominant role in the treatment of uncomplicated (chloroquine resistant) *P. falciparum* malaria, whereas chloroquine is reserved for the treatment of sensitive non-*P. falciparum* strains. Also, in both endemic and non-endemic countries, intravenous artesunate replaced intravenous quinine as the drug of choice for the treatment of severe malaria [6,7] and

contributed significantly to an increased survival of severe malaria cases, with up to 33% relative reduction of mortality in endemic regions. Post-marketing surveillance studies revealed that a delayed haemolysis may occur 2-4 weeks after artesunate treatment of severe malaria. Risk factors include hyperparasitemia and young age.[8] All patients treated with parenteral artesunate should therefore be followed up for at least four weeks after artesunate to detect signs of haemolysis and to allow appropriate symptomatic treatment.[8]

The differences in mortality of severe *P. falciparum* malaria in endemic and non-endemic countries like The Netherlands are conceivably due to differences in availability of proper antimalarials like artesunate and access to supportive or intensive care facilities. In the Netherlands, the mortality of imported severe *P. falciparum* malaria is extremely low because of our well-organized health care system and the availability of artesunate and supportive care measures like mechanical ventilation, renal replacement therapy, vasopressors, fluid resuscitation, restoration of coagulation disorders, broad-spectrum antibiotics, blood transfusion, and sometimes blood exchange transfusion or selective erythrocytapheresis.[9] After the introduction of artesunate treatment for severe malaria almost a decade ago, the additional benefit of exchange transfusion in terms of parasite clearance was shown to be negligible [6], and therefore exchange transfusion has been largely abandoned.

PREVENTION OF IMPORTED MALARIA

Since malaria in travellers can be a fulminant and fatal disease, travellers to malaria endemic areas are advised to adhere to chemoprophylaxis for malaria. Although none of the prophylactic regimens offer 100% protection against malaria (inadequate efficacy especially against non-*P. falciparum* malaria), it does protect against the development of severe *P. falciparum* malaria if taken meticulously.[10] However, the perceived side effects of malaria chemoprophylaxis often cause the traveller to refrain from malaria chemoprophylactic drugs. As a kind of compromise, travel health authorities in the German speaking European countries advise stand-by treatment for travellers to areas with a low prevalence of malaria, i.e. a course of artemether-lumefantrine or atovaquone-proguanil to be used in case malaria is suspected. The results of standby treatment look promising. Active surveillance did not find an increase in the number of malaria cases after implementation of this preventive policy in the German-speaking countries. The Dutch Coordinating Centre for Health Advice is currently considering this policy for implementation in the Netherlands.

CONCLUDING REMARKS

In this review, several aspects of imported malaria have been discussed. Due to a decreased transmission risk, a decreasing number of malaria cases have been observed in the past decade, especially in non-immune Dutch tourists. Refugees from malaria endemic regions may cause a resurgence of imported malaria cases in the Netherlands. Diagnostic tests for malaria increasingly rely on rapid diagnostic tests for initial screening, but their use is associated with several important limitations. Intravenous artesunate has replaced quinine as the treatment of choice for imported severe malaria. Given the potentially fatal course in non-immune individuals, malaria chemoprophylaxis is advised for travellers entering a high-risk malaria endemic country, but this advice is being increasingly ignored due to the presumed side effects. Stand-by treatment may provide a valuable alternative policy for prevention in low- to medium-risk malaria regions.

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Quizzz

In the travel clinic

A 27-year-old male traveller was admitted to hospital in the Netherlands because of fever and weight loss. He travelled in Africa from north to south during 9 months. He started in Morocco, crossed the Sahara and continued via Nigeria to Zimbabwe. Four months before admission he developed fever, headache and muscle aches; he went to a local laboratory and was found to have a positive blood film for malaria. He was treated with an antimalarial drug after which the fever disappeared. Two months before admission he had an episode of diarrhoea with some blood admixture; there was no fever. He used some tablets from his travel companion ('against diarrhoea') and improved. One month before admission he developed fever and muscle aches that continued until admission; later there was also right upper quadrant abdominal pain. He had been adequately vaccinated for diphtheria, tetanus, poliomyelitis (DTP -booster), typhoid fever, yellow fever, hepatitis A and B and rabies; he used mefloquine as malaria prophylaxis, albeit intermittently.
After returning home, he had persistent intermittent fever, abdominal pain, right shoulder pain and generalized weakness. There was slight non-productive cough. He had lost 5 kg of weight since the start of the fever. He was admitted to hospital.

PHYSICAL EXAMINATION

Ill looking; not jaundiced; not pale.
Vital signs: blood pressure 130/85 mm Hg; pulse rate 95/min, regular; respiratory rate 24/min; temperature 39.5 C.
Head and neck: no abnormalities.
Lungs: dullness right lower zone; normal breath sounds.
Heart: apex beat not displaced, not enlarged on percussion; heart sounds S1 S2, no pericardial rub.
Abdomen: tenderness right upper quadrant, liver enlarged 3 cm, tender, smooth surface, sharp edge.
Spleen not palpable.
Extremities: no oedema.
Skin: in the anterior axillary line at the upper abdomen/lower chest: swelling, redness and tenderness with peau d'orange appearance.

LABORATORY RESULTS

ESR 60 mm/hr
Hb 8.1 mmol/L
TWC 13.3 x 10^9/L; differential count: 80% neutrophils
Platelet count: 173 x 10^9/L
Bilirubin 16 mmol/L
AST 14 U/L
ALT 23 U/L
Alkaline Phosphatase 110 U/L (n< 75)

Questions

1. What is the differential diagnosis?
2. What diagnostic procedures would you do?
3. What is the likely diagnosis?
4. What is the preferred treatment?

Answers on page 15

REVIEW

Zika virus hitting the headlines

EPIDEMIOLOGY

In May 2015, locally acquired cases of Zika virus infection were confirmed in Brazil.[1] Since then, the virus has spread to more than 25 countries in the Caribbean, North and South America. WHO/ PAHO have warned that the virus is likely to spread across nearly all of the Americas due to a lack of any natural immunity. They added that the vector, Aedes mosquitoes, is present in all countries of the region, except in Chili and in Canada.

A decade ago, confirmed cases of Zika virus infection from Africa and Southeast Asia were rare, and until recently there has not been much published on the virus. In 2007 the virus hit Yap in Micronesia, and via French Polynesia and other Pacific islands in 2013 it arrived – most probably via Easter Island – in Brazil in 2014, and in 2015 in Colombia and in Surinam.[2,3,4,5] Cape Verde in Africa reported a Zika virus outbreak in 2015 as well.

Infection with Zika virus has been linked to newborn babies with congenital microcephaly. It is presumed to be responsible for Guillain-Barré syndrome and other neurologic conditions such as 73 cases in a population of 270,000 in a French Polynesian epidemic in 2013.[6,7]

ZIKA VIRUS

The Zika virus is an emerging mosquito-borne arbovirus belonging to the family of flaviviridae, native to Africa or Asia, and transmitted via Aedes mosquitoes, such as *A. aegypti* – the same mosquito that transmits dengue, chikungunya, West Nile virus and yellow fever.[8] The virus was first isolated from a rhesus monkey in Uganda in the Zika forest in 1947, and it was first described in 1952 by Dick et al in the Transactions of the Royal Society of Tropical Medicine & Hygiene.[9] The infection has a short incubation time of a few days and causes mild fever, conjunctivitis, skin rash and headache (sometimes called dengue-light). These symptoms normally last for 2-7 days. Around 80% of Zika virus infections in individuals are asymptomatic. Usually hospitalization is not necessary and mortality is extremely rare.

TRANSMISSION

Aedes mosquitoes transmit Zika virus; evidence about other transmission routes is limited. Spread of the virus occurs through blood transfusion, but cases of transmission through sexual contact have also been reported.[10] Zika virus has been isolated in human semen, and one case of possible person-to-person sexual transmission has been described. Standard precautions that are already in place for ensuring safe blood donations and transfusions should be followed.

Evidence on mother-to-child transmission of Zika virus during pregnancy or childbirth is limited. A mother already infected with Zika virus near the time of delivery can pass the virus on to her newborn around the time of birth, but this is rare. It is possible that Zika virus could be passed from a mother to her baby during pregnancy. Research is needed to generate more evidence regarding perinatal transmission and to better understand how the virus affects babies. There is currently no evidence that Zika virus can be transmitted to babies through breast milk. Mothers in areas with Zika virus are advised to follow the PAHO/ WHO recommendations on breastfeeding: exclusive breastfeeding for the first 6 months, followed by continued breastfeeding with complementary foods up to 2 years or beyond.

DIAGNOSIS, PREVENTION AND TREATMENT

Zika virus is diagnosed through PCR (polymerase chain reaction) and virus isolation from blood samples. Diagnosis by serology can be difficult as the virus can cross-react with other flaviviruses such as the ones responsible for Japanese encephalitis, dengue, West Nile virus infection and yellow fever.

The most effective forms of prevention are reducing mosquito populations by eliminating their potential breeding sites, especially containers and other items such as discarded tires that can collect water in and around households and in particular using personal protection measures to prevent mosquito bites.

Zika virus disease is usually relatively mild and requires no specific treatment. People sick with Zika virus should get plenty of rest, drink enough fluids, and treat pain and fever with common medicines. If symptoms worsen, they should seek medical care and advice.

INTERNATIONAL RESPONSE AND IMPLICATIONS

The virus is currently spreading rapidly with more and more countries in the Americas reporting cases. Some countries advise women not to get pregnant, while in others women are advised not to travel to regions or to countries where Zika virus transmission is ongoing. The Americas, Europe and Asia receive the most travellers who depart from Brazilian airports internationally. It is obvious that with no vaccine or antiviral therapy available Zika's rather disturbing march may not stop in the Americas. A recent article in the Lancet highlights the potential of Zika virus to follow dengue's and chikungunya's global spread.[11]

In the Netherlands, Zika virus infection has been diagnosed in people who contracted the virus outside the country.[12] At the time of this writing, a total of twenty-three travellers got infected in Surinam. The spread of Zika virus within the country is most unlikely because of a lack of the Zika virus vector in the Netherlands.

PHOTO KATERYNA KON / SHUTTERSTOCK

The WHO, through its emergency committee on Zika virus, has declared the virus a public health emergency of international concern since a causal relationship between Zika virus during pregnancy and microcephaly is strongly suspected although not yet proven. WHO urges better coordination of the international efforts to investigate and thus understand the relationship better. Improving surveillance and diagnosis of infections is needed to detect congenital malformations and neurological complications earlier. Furthermore, stricter control of mosquito populations is needed, as well as the development of diagnostic tests and vaccines for protection of non-immune individuals.[13] With the summer Olympics in Rio fast approaching, lessons from countries with success in controlling pandemics should be learned, for example Saudi Arabia's success with hosting Umrah and Hajj and others with controlling outbreaks of influenza A H1N1, MERS, and Ebola. In the months ahead, Brazilian and Saudi authorities will have the opportunity to review emerging research findings on the natural history of Zika virus. Proactive planning and preparedness will mitigate the effect of Zika virus infection on mass gatherings, participants, and their home and host countries, so that the events can be held with a sense of confidence among all those involved, including organizers as well as participants and the global community.

Based on the available evidence, WHO is not recommending any travel or trade restrictions related to Zika virus disease. Women who are pregnant or planning to become pregnant must determine the level of risk they wish to take with regard to Zika and plan accordingly. In particular, they should:

- stay informed about Zika virus and other mosquito-transmitted diseases;
- consider delaying travel to any area where locally acquired Zika infection is occurring;
- protect themselves from mosquito bites (see above);
- consult their doctor or local health authorities if travelling to an area where Zika virus is present;
- mention their planned travel during their antenatal check-ups; and, upon return, consult with their healthcare provider for close monitoring of their pregnancy.

Until more is known about the risk of sexual transmission, all men and women returning from an area where Zika virus is circulating - especially pregnant women and their partners - should practice safe sex, including the correct and consistent use of condoms.[14]

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Ebola, lessons from an emerging viral haemorrhagic infection in a failing public health infrastructure

Ebola virus has all the elements required for an emerging infectious disease with epidemic potential: a zoonotic bat virus, high host plasticity, and a receptive ecological and 'socio-political landscape'.[1,2,3] During recent times, the Zaire Ebola virus, carried by its presumed fruit bat reservoir, has covered thousands of miles across Central Africa to end up in the Guinea forest region.[4] Systematic deforestation, poverty-driven game hunting, neglected and collapsed health care systems, distrust in an inefficient and poorly resourced government response, and fluid borders with migrating people displaced in a war-torn region have fuelled the outbreak which spread with unprecedented speed.[3] Population growth, increased urbanization, and better mobility and connectivity further had a profound impact on the dispersion of Ebola cases. Within a few months, the outbreak had reached the capital Conakry and the neighbouring countries of Sierra Leone, Liberia, Nigeria, Senegal, and Mali.

In fact, we were lucky. Thanks to the clever and prompt application of the polio eradication infrastructure to contain the outbreak in Lagos and Port Harcourt, the Nigerian authorities were able to avert a truly apocalyptic scenario of Ebola virus disease ravaging Lagos and its 21 million inhabitants in July 2014.[5]

The 2014 Ebola outbreak has demonstrated at least two things: 1) outbreaks like these are very likely to occur again, and 2) there is a pressing need to build resilient public health systems in the weakest countries if we want to halt future infectious health security threats.

The aim of this contribution is to assess the risk of spill-over of Ebola virus, to describe the clinical lessons learned from the current Ebola outbreak, to address the problem of persistence on future transmission, and to review how well the world is prepared for the next epidemic.

THE ZOONOTIC NICHE OF EBOLA VIRUS

The mapping of the zoonotic niche of Ebola virus, based on reported Ebola outbreaks in humans and Ebola infections in animals, has identified moist tropical forests across 22 countries of West and Central Africa as the predicted suitable environment for zoonotic transmission of Ebola virus.[6] Twenty-two million people are estimated to live in these mostly rural areas. The Democratic Republic of Congo, Guinea, Uganda, Nigeria, Cameroon and Central African Republic belong to the top 10 countries where the population is most at risk. The rapidly increasing human population in these areas and reliance on bush meat for protein may further accelerate the risk of spill-over of zoonotic viruses.[7] In addition, the increased international connectivity by air travel, often within the sub-Saharan African region, carries the potential for international spread, especially to countries with weak health care systems. During the outbreak, 60% of the travellers departing from Guinea, Liberia and Sierra Leone were estimated to be travelling to countries with inadequate medical and public health infrastructure.[8] One of the key messages in a Lancet paper on Global Health Security was, 'The epidemic has shown that we are only as safe as the most fragile states and it is a reminder that improvement of the capacity of every country to find, stop, and prevent health threats is both in the world's self-interest, and a moral imperative'.[9]

CLINICAL CHARACTERISTICS AND CASE DEFINITION

Already early on in the outbreak, it became clear that haemorrhage was a late and uncommon although unfavourable sign, presenting in only 18% of the patients.[10] In contrast, gastrointestinal symptoms were frequent and could be life threatening.[11] Therefore, the name of the disease was changed from Ebola haemorrhagic fever to Ebola virus disease (EVD). The most commonly reported symptoms at presentation are fever, intense fatigue, loss of appetite, vomiting, headache and abdominal pain.[10,12] Unfortunately, these clinical features are too unspecific to discriminate infected from non-infected individuals. For example, in a retrospective cohort study in Sierra Leone, presentation with three or more symptoms like intense fatigue, confusion, conjunctivitis, hiccups, vomiting or diarrhoea increased the odds of EVD by 3.2 (95% CI, 2.3 - 4.4).[12] However, the sensitivity and specificity for identification of clini-

PHOTO OLIVIER LE QUEINEC / SHUTTERSTOCK

cal cases was only 57.8% and 70.8%, respectively. In addition, there is an intense debate about fever as a criterion of the case definition. Around 13% of patients did not present with fever.[10,12] Similar observations were made by local physicians (personal communication). Non-reported over-the-counter use of antipyretics may be one explanation. In addition, non-contact infrared thermometers (NCIT) often perform poorly. In a review on the accuracy of hand-held devices to detect fever by scanning the forehead, inner eye corner or auricular meatus, the sensitivity and specificity ranged from 82% - 89% and 75% - 99%, respectively.[13] These studies suggest that the risk of missing febrile individuals (100% minus sensitivity) would be between 11% and 18%. Excessive perspiration and surrounding ventilation systems may have a cooling effect, further compromising the performance of NCIT as a screening tool for fever. Therefore, a high level of suspicion and rapid point-of-care laboratory diagnosis remain essential not to miss patients with EVD.

On December 29, 2015, Liberia, Sierra Leone and Guinea had successfully interrupted human-to-human transmission linked to the original outbreak, which started two years earlier. Up to then, over 28,000 cases had been reported[14], with an estimated 17,000 survivors, of whom 6,869 are officially registered. Apart from the psychological trauma of having survived a deadly disease in frightening circumstances in a treatment unit, survivors suffer from stigma, and social disruption because of loss of family and friends to Ebola. In addition, according to a systematic survey in a Sierra Leone EVD survivor care clinic, during the first five months after discharge, 76% of the survivors suffers from arthralgia, 24% from auditory symptoms such as tinnitus or hearing loss, and 60% from eye complaints.[15] Eighteen percent of the survivors developed at times sight-threatening uveitis, possibly because of viral persistence in the ocular chamber. Ebola virus can also persist in the central nervous system and cause encephalitis even after clearance of the virus from the blood.[16] These findings underscore the need for clinical follow-up and care of all survivors and emphasize the need to strengthen universal access to ocular care.

Ebola virus can also persist in semen and has been linked to sexual transmission of Ebola virus in Liberia after the outbreak had ended.[17] It is not yet known how long semen remains infectious, although viral RNA has been found up to nine months after the onset of EVD.[18] Transmission through sexual contact is a rare event, but it may be responsible for late EVD cases, well past the incubation period of 21 days. It is therefore of paramount importance to include the sexual history in the medical evaluation of patients returning with fever from former Ebola epidemic regions.

To effectively manage the residual risk of re-emergence of EVD from reservoirs of viral persistence, the WHO has designed a phase-3 Ebola response framework.[14] This framework consists of 3 pillars: 1) expanding the network of clinical services to all survivors and offering voluntary semen screening and counselling programs for male survivors to protect close contacts (PREVENT); 2) implementing a nationwide surveillance strategy to screen all living or deceased individuals with symptoms compatible with EVD (DETECT); 3) deploying rapid-response teams following detection of a new confirmed case (RESPOND). Progress in the key performance indicators by the different countries can be followed on the WHO Ebola situation report website.

NEED FOR MAJOR REFORMS

Every outbreak with pandemic potential is a game-changer in how the world prepares for the next serious epidemic. Outbreaks like SARS and pandemic H1N1 identified weaknesses within our ability to address global health emergencies and resulted in new approaches such as the revised International Health Regulations (IHR, 2005) and the Pandemic Influenza Preparedness Framework (PIPF, 2011). However, most countries failed to meet the 2012 deadline for fulfilling the obligations on national surveillance and rapid response capacities as stipulated in the IHR. Especially, low-income countries in the epidemic hot zone like Guinea, Liberia and Sierra Leone lacked

infrastructure, strategy and funding to comply with the IHR obligations.[9]

In the Ebola outbreak, there were failures at almost every level. Therefore, major reforms and recommendations have been formulated by independent bodies such as the Independent Panel on the Global Response to Ebola and the Commission on a Global Health Risk Framework for the Future.[18,19] Taken together, these recommendations revolve around four themes. First, 'All countries need a minimum level of core capacity to detect, report, and respond rapidly to outbreaks'.[18] However, it needs to be stressed that it is equally important to include promotion of individual access to safe and effective health services, products and technologies aimed at gaining public trust, and securing community engagement.[9] Second, the WHO needs 'to meet its responsibility for responding to major disease outbreaks and to alert the global community'.[18] Third, 'Rapid knowledge production and dissemination are essential for outbreak prevention and response'.[18] And finally, 'An effective worldwide response needs leadership, clarity about roles and responsibilities, and robust measures of accountability'.[18] The recommendations will substantially reform existing bodies and create new processes and financial arrangements. Hopefully, we will act now 'before the opportunity is lost as global attention moves on'.[5] The next serious outbreak will be as unexpected as Ebola, and it will thrive on our failure to implement the lessons we have learned from the past.

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Quizzzz

In the travel clinic - Answers

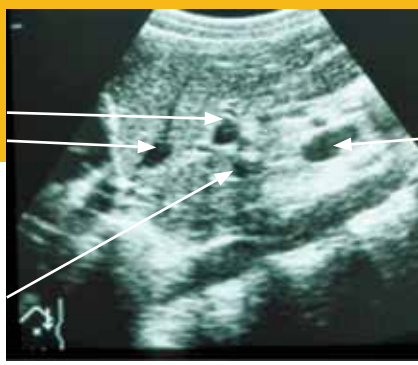
Answers

1 Amoebic liver abscess, pyogenic liver abscess, hepatocellular carcinoma, hydatic cysts; misdiagnosis as cholecystitis or appendicitis occurs.

2 Blood cultures *(these were negative)*, rapid diagnostic test and blood film for malaria *(negative)*. Stool examination for parasites and eggs (showed *E. histolytica* cysts). The chest x-ray showed a raised hemidiaphragm with clear lung fields (the normal breath sounds already suggested that there was no intrapulmonary problem). An ultrasound examination showed four liver abscesses (Figure). A serological test for amoebiasis was positive.

3 Amoebic liver abscess with extension to the skin and right hemidiaphragm leading to shoulder pain.

4 A tissue amoebicidal drug – tinidazole or metronidazole – followed by a contact amoebicidal drug – paromomycin, clioquinol or diloxanide furoate – for elimination of luminal cysts, even if cysts are not found in the stool examination.



Ultrasound of the liver with arrows indicating at least 4 areas suspect for abscesses

Amoebiasis has a world-wide distribution and it is estimated that 10% of the world population is infected with the causative protozoal agent *Entamoeba (E.) histolytica*. Most people are asymptomatic (up to 90%). The transmission is faecal-oral and hence it is most common in unhygienic conditions. The most common clinical manifestation is amoebic dysentery characterized by slow onset of bloody and mucoid diarrhoea with abdominal pain; fever is not a prominent feature but sometimes there may be mildly raised temperature.

Only 10% of *Entamoeba histolytica* parasites are potentially invasive from the gastrointestinal tract. Their microscopic appearance is identical to the non-pathogenic *Entamoeba dispar*, but they can be differentiated by PCR. When invasive (extra-intestinal *amoebiasis*), the liver becomes infected through the portal vein. A liver abscess may develop that may be single or multiple; large abscess may become confluent. Clinically, the patient has fever, chills, right upper quadrant pain with right or left shoulder pain through irritation of the diaphragm depending on the localization in the liver. Over time, anaemia and weight loss may also occur. A superficial abscess in the right liver lobe may irritate the overlying skin with subsequent infiltration resulting in the 'peau d'orange' appearance; this may indicate imminent rupture. From the liver, the abscess may spread into the pleural or pericardial space, or rarely metastasize to e.g. the brain.

Diagnosis requires appropriate exposure in an endemic area, an ultrasound, and a serological test such as the immunofluorescence test; the sensitivity is >95%, one week after onset. There is no need for an aspirate as the parasites are at the edge of the abscess and difficult to target by the needle. In addition, there is a risk of introducing secondary bacterial infection. The white cell count is typically raised as are the liver enzymes, in particular alkaline phosphatase. The stool examination may or may not show *E. histolytica* cysts. A chest x-ray may show a raised hemidiaphragm with or without pleural effusion.

The treatment response is good and the clinical response (disappearance of fever), normalization of the ESR, white cell count and liver enzymes may be used as parameters for cure. On the ultrasound, the abscesses may disappear or persist for months.

Drainage may be indicated in large left lobe abscesses that potentially could perforate to the pericardium, or in severely ill patients with imminent rupture of the abscess, or in case of unresponsiveness to drug treatment. Drainage is also indicated in case of an uncertain diagnosis, in particular with a differential diagnosis of pyogenic abscess where drainage is usually necessary. It is important to eradicate the parasites in the gastrointestinal tract with a contact amoebicidal drug to avoid recurrence of the liver abscess.

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JACTATIO CAPITIS: *IS YOUR CHILD A HEAD BANGER?*

Figure 1

Figure 2

SETTING

This case is set in the Lion Heart Medical Centre in Yele, a rural hospital in the middle of Sierra Leone. The hospital is run by local staff and three tropical doctors trained in the Netherlands. They have access to a laboratory, ultrasound, and an X-ray machine (which is currently out of order). Furthermore, the hospital has an operating theatre.

CASE

A 13-year-old boy presented at the out-patient department with two dark, elevated areas on the forehead (Figure 1 and 2). The onset was spontaneous without previous trauma. According to the patient and his parents, the discolouration occasionally resolved and returned. The lesions did not cause him any pain. The patient was convinced he was 'strange' and 'different', leading to absence from school. He had been treated with betamethasone cream without effect.

On physical examination, he was a healthy, well-nourished boy with two round, hyperpigmented masses on the forehead which were hard on palpation, giving the impression of bony tissue. The tumours were not attached to the skin and did not fluctuate. The skin was normal; there was no wound, lesion or scarring. There was no pain on palpation.

SPECIALIST ADVICE

The dermatologists and paediatricians were consulted regarding this case. Within two days, there were three replies with unanimous advice. The specialists suggested post-inflammatory hyperpigmentation as the underlying disorder, commonly caused by friction or rubbing. They proposed that nightly head banging (jactatio capitis nocturna) could lead to lesions in this particular area, by subconsciously hitting the head against a wall or bed during sleep. The tumours on the forehead could be hematomas, which may be hard on palpation. It was also proposed that, in case the patient was a Muslim, the lesions might be caused by repeatedly touching the floor with the forehead during prayer. They recommended taking a thorough history to ascertain the underlying inflammatory process.

TREATMENT

Unfortunately, no follow-up is available regarding this case as the patient did not return to the hospital for further treatment and evaluation. A further history as to whether this child was indeed a 'head banger' could therefore not be obtained.

BACKGROUND OF HEAD BANGING

Jactatio capitis, or head banging, is part of a rhythmic movement disorder (RMD), which also includes headrolling, bodyrocking and bodyrolling.

EPIDEMIOLOGY

RMD is typically seen in the first year of life, resolves before the age of 10 and seldom continues

into adulthood.1 It more commonly affects males and is associated with ADHD, autism or mental retardation in older patients.[1,2] RMD can be aggravated during periods of stress and sleep deprivation.[3]

CLINICAL MANIFESTATIONS AND DIAGNOSIS

RMD is defined by the International Classification of Sleep Disorders as repetitive, stereotyped movements of the large muscles, occurring anywhere in the body.1 The movements last several minutes and typically occur during sleep onset and light non-REM sleep. Patients are often not aware of their condition.

DIAGNOSIS

RMD can be diagnosed by observing nightly behaviour on video. More advanced diagnostic tools include polysomnography and videometry. As the differential diagnosis includes epilepsy, an EEG should be performed.

PROGNOSIS

RMD is a self-limiting disorder and does not influence the development of children. However, adverse effects such as injuries, fractures and even subdural hematoma have been described.

THERAPY

RMD does not need treatment apart from reassurance and patient education. Safety precautions and general sleep hygiene measures are recommended.[3] In severe cases, medical treatment with benzodiazepines can reduce the severity and frequency of RMD. Clonazepam has been reported to be effective.[4,5]

BACKGROUND OF POSTINFLAMMATORY HYPERPIGMENTATION

Postinflammatory hyperpigmentation (PIH) is a reactive hypermelanosis of the skin, occurring as a result of cutaneous inflammation.

EPIDEMIOLOGY

The exact prevalence of PIH is unknown. It can occur in any skin type but is most common in darker skin (Fitzpatrick skin type IV, V and VI: light brown to black).[6,7] It affects males and females equally and can occur at any age.

ETIOLOGY AND PATHOPHYSIOLOGY

PIH can arise from different types of inflammatory processes, which can be divided into two categories: endogenous and exogenous conditions.[6,8] Among the endogenous causes are inherited diseases, cutaneous diseases, systemic diseases and allergic reactions. Exogenous conditions resulting in PIH include mechanical trauma, extremes of temperature, radiation, phototoxic reactions, chemical peels and laser procedures. Common causes include acne vulgaris, eczematous dermatoses and burn injury. In Muslims, a prayer mark can be considered.[9]

Two pathophysiologic mechanisms exist, leading to an excess or abnormal distribution of melanin either in the epidermis or in the dermis, thereby giving rise to two clinical subtypes: epidermal melanosis and dermal melanosis. PIH within the epidermis results from the release of inflammatory substances, stimulating melanocytes to increase production of melanin, with pigment being transferred to keratocytes.[6] The dermal variant is caused by the release of melanin from basal keratinocytes, which have been damaged by an underlying inflammation process. The pigment is then phagocytosed by macrophages in the upper dermis.[8]

CLINICAL MANIFESTATIONS AND DIAGNOSIS

The affected areas correspond with the distribution of the initial inflammatory dermatosis.[6,7] Epidermal melanosis is characterized by light to dark brown lesions which are well-circumscribed. It may take 6-12 months to years to resolve without treatment.[6] When the pigment is in the dermis (dermal melanosis), the lesions are darker blue-grey and poorly circumscribed. This hyperpigmentation can take years to fade to normal.[6]

PIH is a clinical diagnosis, based on the typical hyperpigmentation and a history of a preceding inflammatory process. A Wood's lamp can be used to clinically distinguish dermal from epidermal melanosis.[6] The diagnosis can be confirmed by biopsy, showing melanin in the dermis or epidermis.

TREATMENT

The first step in the treatment of PIH is eliminating the underlying inflammatory process. The patient should be advised to use high-factor sun protection and to avoid cosmetics, manual manipulation and harsh conditions (wind, cold, sunlight).[6] Epidermal melanosis can be treated by topical depigmenting agents, retinoids, corticosteroids, chemical peels and laser modalities. Topical hydroquinone, which reduces melanin, is considered the gold standard therapy.[8] Deeper pigmentation as in dermal melanosis does not respond well to the above mentioned treatment.[6] Patients may benefit from camouflage using cosmetic products.

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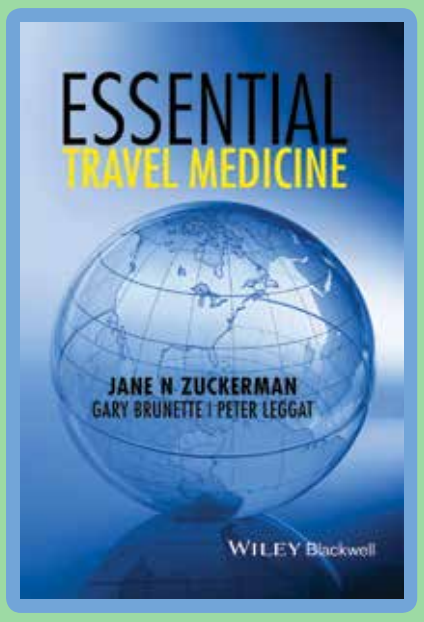
Essential Travel Medicine



Edited by Jane. N. Zuckerman.
Wiley Blackwell, July 2015, 360 pages, €87.80

ESSENTIAL TRAVEL MEDICINE

Most of you are probably frequent travellers rather than students in travel medicine. Or you may be living abroad and visiting home regularly. But if you want to dig a bit deeper into travel medicine, this book could be worth your while! Released last year, this book with contributions from all over the world, aims to support students in travel medicine and stimulate interest and enthusiasm for the discipline.

Starting with some basic epidemiology, the book raises your knowledge to a level beyond just knowing that malaria is mostly acquired in West Africa and enteric fever in the Indian subcontinent. It also emphasizes the risks of non-infectious diseases, illustrating one of the difficulties of travel medicine:

'ONE OF THE MOST SIGNIFICANT CHALLENGES FOR THE TRAVEL HEALTH ADVISOR IS TO CONVEY MESSAGES OF PREVENTION TO A POPULATION THAT IS MORE CONCERNED ABOUT EXPOSURE TO INFECTIOUS DISEASES'

Then it gives a very practical overview of how to set up a travel clinic, referring to more in-depth resources and (international) guidelines.

The clinical part of the book covers most infectious diseases, giving first the expected basics on epidemiology and pathophysiology and then focusing mostly on the clinical presentation in travellers, (chemo)prophylaxis and emergency treatment. It also includes a full chapter on vaccine-preventable diseases with detailed vaccine schedules, precautions, storage information etc.

The next section discusses specific groups of travellers separately: VFRs (people 'visiting friends and relatives' in their country of origin), migrants, students who study abroad and the group that most of you belong to – humanitarian aid workers and missionaries. Not only specific groups of people have special risks and needs but also certain environments. The risks of aviation are mentioned, including the oxygen dissociation curve. And of course expeditions in the wilderness and ventures like mountain climbing and diving have their particular hazards of exposure to heat and direct sunlight, extreme altitudes, etc. Even full-service cruise ships are not without danger; imagine a Noro outbreak on a ship that contains over 6000 passengers with a passenger-to-crew ratio of 2:1!

The last section of the book follows the chronology of travelling and focuses on the returning traveller, with fever and gastro-intestinal complaints as the most common symptoms that may not immediately be recognized by a clinician unless the patient reveals his or her travel history.

MISSION ACCOMPLISHED?

At the end of the journey, the question remains: does the book accomplish its goal? Or a better question to ask might be: after reading this book, have you achieved the goal you had at the start of your trip? If you are looking for a concise, structured guide in travel medicine this is the right book. But, if you are interested in a more specific topic, for example migrant health, it may be too superficial and not very helpful. But that is inevitable in a compact book on a very broad topic. In that sense, it did a great job. The book is well structured, easy-to-read, and where necessary it refers to more in-depth literature. Recommended!

JOSEPHINE VAN DE MAAT, PHD STUDENT
ERASMUS MC

News from the NVTG working parties: Working Party on International Safe Motherhood and Reproductive Health

Linking Ethiopian gynaecologists with Dutch colleagues: Request for collaboration with the Working Party on International Safe Motherhood and Reproductive Health

How can one contribute to decreasing the unacceptable health differences around the globe whilst working in a rich country? How to help improve the health of women and children from a distance? In what way can gynaecologists working in the Netherlands assist their peers in underprivileged parts of the world? And how can they, the ones who are interested in collaborating, find us, rather than the other way around? The Working Party on International Safe Motherhood and Reproductive Health (WP) introspectively addressed these questions, looking to renew the purpose for its existence.

Our WP is part of the Netherlands Society for Tropical Medicine and International Health (NVTG) and the Dutch Society of Obstetrics and Gynaecology (NVOG). Until recently, we supported the Association for Gynaecologists and Obstetricians of Tanzania, a twinning which recently ended. Members of the WP expressed their interest in continuing such collaborations as complementary to the work the WP does in the Netherlands. This comment in MTb explores the contours of a possible new collaboration of the WP with colleagues in Gondar, the former capital of Ethiopia.

The formal request for collaboration came from local gynaecologist working at the University Hospital of Gondar. The support requested differed somewhat from what we usually provide, as they were more interested in post-speciality training in uro-gynaecology and gynaecologic oncology rather than capacity building in basic obstetrical and gynaecological skills. Also they expressed interest in operational research and supporting staff in surrounding health centres.

The WP responded positively to the request and, in conjunction with the Liverpool School of Tropical Medicine, delivered training in obstetric emergencies, facilitated by a grant from Share-Net. Also, the urogynaecologist and gynaecologic oncologist conducted surgery with local staff and examined possibilities for post-specialty training. The training was well received, and the team was impressed by the careful selection of patients and the decent level of perioperative care.

This contrasted with the level of obstetric care provided in the surrounding health centres. Basic proactive and supportive management of labour is not always performed; there is reluctance to induce labour, perform artificial rupture of membranes, start oxytocin and do operative vaginal deliveries. And although reliable data are absent, it is clear that peripartum maternal and perinatal mortality and morbidity remain high. Over the past decade, the maternal mortality ratio has decreased from 871 to 676 per 100 000 live births, but Ethiopia remains the fifth contributor to global maternal mortality in absolute numbers. This strengthens our conviction that our obstetric training efforts need to be sustained.

Besides the first set of trainings, we addressed operational research needs. A number of interventions, which have been shown to be effective in high-income settings, are to be tested for their effect on Ethiopian women. Plans to assist in improving the referral system and the functionality of health centres are under way but require local political commitment, for example regarding staffing.

Currently we are identifying specialists, in particular urogynaecologists and gynaecological oncologists, to take part in this collaboration with Gondar. We are sharing our experiences to illustrate the conditions for good partnering, i.e. the action should be initiated and led by the local counterpart and must be carefully planned, with due attention for relevance and sustainability. We look forward to hearing from other NVTG working parties on their experiences.

For more information: secretariaat.wp@gmail.com.

On behalf of the Working Party,

THOMAS VAN DEN AKKER
JELLE STEKELENBURG

Erratum

In the previous edition of MTb (December 2015), in the article The Drugs for Neglected Diseases Initiative by S. Wells, the last sentence before the heading 'Risk sharing' on page 8 should read as follows: Both antimalarial projects, ASAQ and ASMQ, were formally handed over in May 2015 to the Access and Product Management Team of MMV, in order to continue efforts to help maximize patient access.

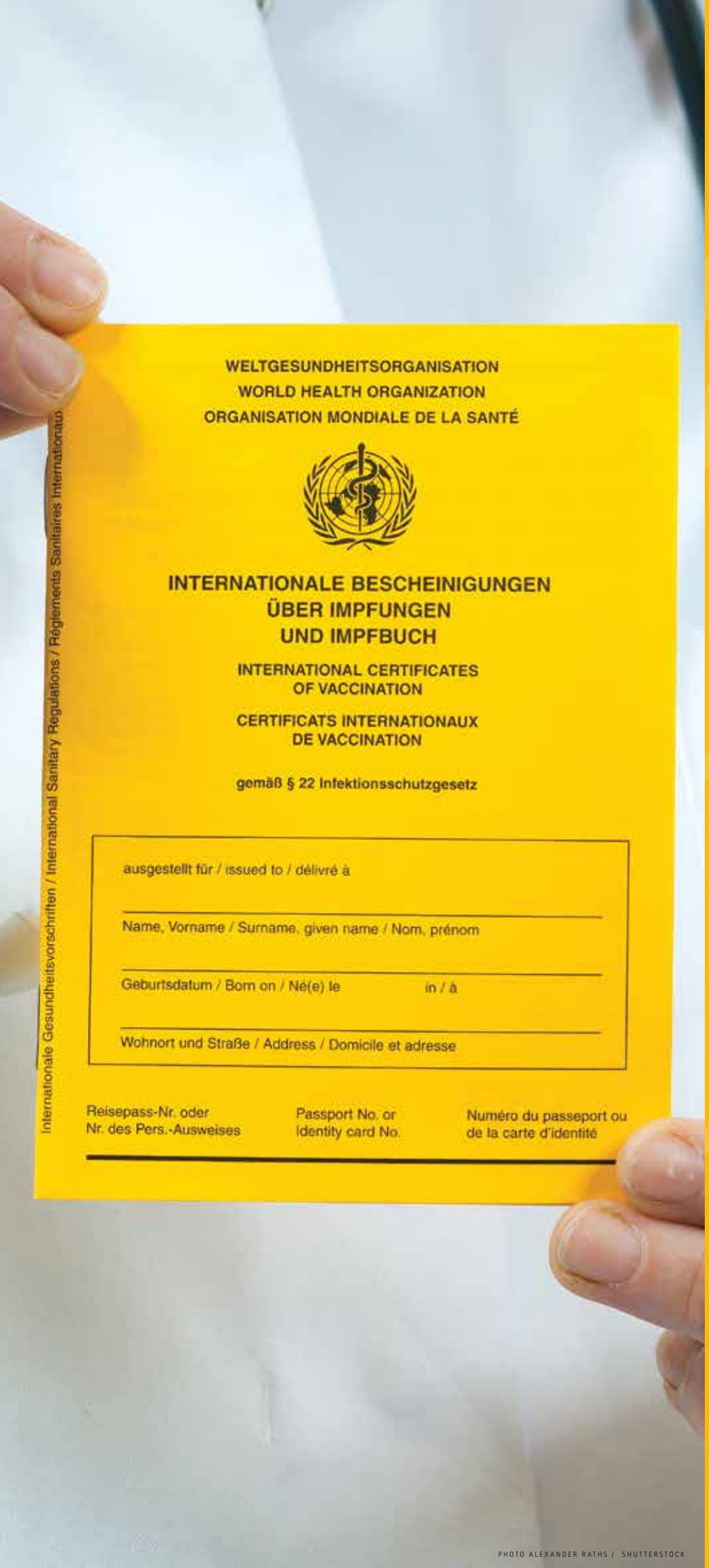


PHOTO ALEXANDER RATHS / SHUTTERSTOCK

NVTG

Membership of the **Netherlands Society for Tropical Medicine and International Health** (NVTG) runs from January 1st to December 31st and may commence at any time. Membership will be renewed automatically unless cancelled in writing before December 31st. Membership includes MT and International Health Alerts. An optional subscription to TM&IH carries an additional cost.

Non NVTG members can subscribe to MT through a student membership of the Society for € 23 per year by sending the registration form through our website **www.nvtg.org/lidworden** or by sending name and postal address by e-mail to **info@nvtg.org or MTredactie@nvtg.org**.

Contributions and announcements should be submitted to the editorial office by e-mail: **info@nvtg.org.** Closing date for N° 02 / June 2016: 22-04-2016.

Disclaimer: all views expressed in this journal are of the authors only and are not necessarily shared by the editors of MT. Letters and articles may be edited for purposes of (clarity and) space.

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