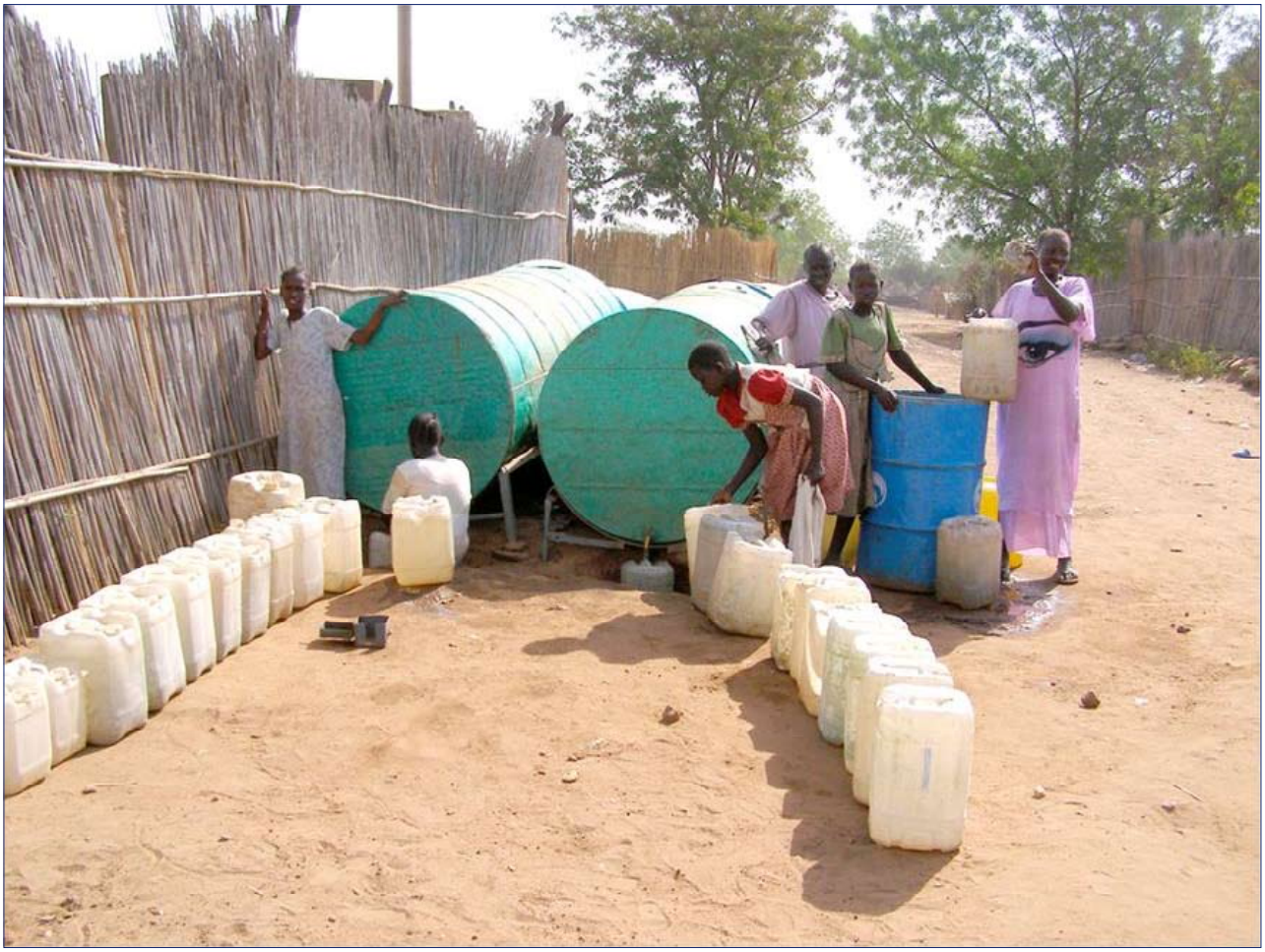
SSMJ
South Sudan Medical Journal

ISSN 2309 - 4605

Volume 7. Number 2. May 2014

www.southsudanmedicaljournal.com



Cholera Outbreak in South Sudan

- What is cholera?
- WASH in South Sudan: what needs to be done
- KAP of tuberculosis patients enrolled on treatment in Juba
- Clostridium difficile
- Hepatitis E Virus

CONTENTS

Cover photo:

Access to water in Juba during 2006. These big barrels are filled with untreated water brought by a water tanker from the River Nile. Women in the residential areas buy the water from the owner of the tank. (credit Victor Vuni Joseph)

SSMJ South Sudan Medical Journal
ISSN 2309 - 4605

Volume 7. No. 2. www.southsudanmedicaljournal.com

A Publication of the South Sudan Doctors' Association

The South Sudan Medical Journal is a quarterly publication intended for Healthcare Professionals, both those working in the South Sudan and those in other parts of the world seeking information on health in South Sudan. The Journal is published in mid-February, May, August and November.

Reviewers are listed on the website

Response to the cholera outbreak in South Sudan

On Thursday, May 15th 2014, the Ministry of Health (MoH) of the Republic of South Sudan declared a cholera outbreak in the capital Juba. As we go to press, the cholera has spread to other parts of the country and the cases are increasing.

In its press statement, the MoH said it had "Reactivated a national emergency taskforce to coordinate the response interventions". This mechanism was set in place to organize the response, coordinate actions of the different partners in curbing the spread of the outbreak.According to WHO reports, a suspected case of cholera was reported on April 29th from a Medicines Sans Frontiers(MSF) clinic in Juba III/UN House Internally Displaced Persons (IDP) camp. A contact in the household had diarrhoea a week earlier. An investigation was done, which confirmed that the diarrhoea was caused by the bacteria Vibrio cholera.

Cholera is a disease of poor sanitation and hygiene, with a short incubation period of two hours to five days. A person dies from severe loss of body fluids as a result of the frequent diarrhoea.The situation is South Sudan had always been seen as a crisis about to happen, where thereis lack of clean drinking water, poor or lack of latrines and good hygiene practices. The last time the country had an outbreak was in 2008, in which more than 6,000 South Sudanese were affected and least 44 died, with more than half dying within the first four weeks.

The MoH had identified several risk factors as drivers of this outbreak:

- Drinking of untreated river water, which in Juba is primarily supplied by water tankers.
- Poor latrine use.
- Eating foods sold on the roadside and at makeshift markets.
- Poor personal hygiene practices (for example, poor hand washing) and community hygiene.
- In addition, not defaecating in a toilet, water from unsafe sources such as surface water-river and ponds, poor community handling of dead bodies and unsupervised burials are other factors that increase the risk of the Juba community to contracting cholera.

In order to address these, a national task force was set up to address issues of case management, surveillance and social mobilization.Cholera Treatment Centers (CTC) have been set up in Juba Teaching Hospital and Gudele area west of Juba to receive the cases and response teams put in place.

Once this outbreak is contained, more work needs to be done to prevent recurrence (see article on page 40). The issue of sanitation in the country, and Juba City in particular must be addressed so that cholera is kept at bay, once and for all. An investment in a good water processing plant and sewage disposal system will go a long way in preventing epidemics of water borne diseases in the future.

Dr. Edward Eremugo Luka

Editor-in-Chief

Printing of this journal is made possible by the support of the American People through the United States Agency for International Development (USAID) through MCHIP, under Cooperative Agreement Number AID-668-LA-12-00003, Leader with Associates Agreement GHS-A-00-08-00002-00. The contents of this journal are the sole responsibility of the Doctors' Association and do not necessarily reflect the views of USAID or the United States Government.

USAID
FROM THE AMERICAN PEOPLE

MCHIP Maternal and Child Health Integrated Program
South Sudan Integrated Service Delivery Program

Knowledge, attitude and practice(KAP) of tuberculosis patients enrolled on treatment in Juba City, South Sudan 2010: a pilot study

Lou Joseph Kenyi[a], Taban Martin[b], Godwin Ohisa[c], Dila Matthew[d], Stephen Macharia[e], and Lasu Joseph[f]

a Deputy Head of Unit, National Tuberculosis, Leprosy and Buruli Ulcer Control Program (NTLBP); Ministry of Health (MoH), Republic of South Sudan, Juba.
b Training Officer, NTLBP, MoH, RSS, Juba, South Sudan
c Training Officer, NTLBP, MoH, RSS, Juba, South Sudan
d Medical Assistant, MoH, Juba Teaching Hospital, Juba, South Sudan
e Country Lead, TB CARE Project, Juba, South Sudan
f Head of Unit, NTLBP; MoH, RSS, Juba, South Sudan

Correspondence to: Lou Joseph Kenyi, C/O. Tuberculosis Control Unit, Ministry of Health, Republic of South Sudan, Juba. P.O.BOX. 80.
Email: lou.joseph16@gmail.com

Abstract

Study setting: Juba Teaching Hospital, Juba city, Republic of South Sudan, 2010.

Objective: To examine, knowledge, attitude and practices of tuberculosis (TB) patients enrolled on tuberculosis treatment, Juba, South Sudan.

Design: Descriptive study

Results: Knowledge in TB: Of the 102 patients interviewed; up to 80.4% were not knowledgeable on cause of TB, 52% did not know correct signs and symptoms of TB, 39.2% did not know overall treatment duration, 54.9% did not know the importance of strict adherence to treatment. Knowledge on correct diagnosis was 87.3% and on correct means of TB transmission was 79.4%.

Practices and Attitudes: On practices; 94.1% respondents were able to perform at least one task to stop spread of disease, access to free TB test occurred in 100% of cases and for free drugs in 99% cases. Health care workers correctly suspected TB on first contact in 95.1% of cases. Patients were offered health education on drug side effects in 93.1% of cases, on HIV testing and counselling in 74.5% of cases. Disclosure of TB diagnosis by patient to family or community did not occur in 91.2% cases. Family, community and employers offered support to patients in 92.2%, 95.1% and 98% of cases respectively.

Conclusion: We found key knowledge gaps among Juba TB patients enrolled on treatment. These knowledge gaps are probably responsible for the high treatment defaulter rates reported in Juba, South Sudan. Tuberculosis patients are still not interested to freely reveal disease diagnosis to members of the family and community at large.

Background

The global burden of tuberculosis (TB) remains enormous according to the World Health Organization (WHO) 2013 report [1]. In this report, there were an estimated 8.6 million incident cases of TB and 1.3 million people died from the disease. Among the deaths were an estimated 170,000 from Multi Drug Resistant Tuberculosis (MDR-TB). Although, South Sudan is not listed among the top 22 high burden countries in the world, the WHO estimates incidence of the disease at 146/100,000 of the population (global report 2013).

The South Sudan National Tuberculosis Programme has documented an increasing trend in TB case notification [2]. The number of cases per 100,000 population was:

50 in 2008;

68 in 2009;

71 in 2010;

84 in 2011; and

96 in 2012.

Treatment success rate has generally been below national and WHO targets of at least 85% [2], that is:

77% in 2007;

78% in 2008;

78% in 2009;

79.4% in 2010; and

78% in 2011.

Treatment defaulting has been the major reason for the low treatment success rate (11.4%) in 2010. The three Tuberculosis Management Units (TBMU) in Juba alone accounted for 46% of all defaulters registered in South Sudan in 2010 Tuberculosis Report [3]. The reasons for the very high defaulter rates have not been systematically

documented. Moreover, interruption of TB treatment is a risk factor for the development of MDR-TB, a disease that is not only difficult and expensive to treat but also carries high mortality rate.

Thus, this study was designed to assess knowledge, attitude and practices of TB patients on treatment and find out if any gaps exists that could explain the high defaulter rates.

Study objective

To examine knowledge, attitude and practices of tuberculosis patients enrolled on tuberculosis treatment.

Materials and methods

Design and setting

This was a descriptive study, conducted from 5th Feb 2010 to 5th March 2010. The study was conducted in the three TBMUs within Juba city: Juba teaching hospital, Kator and Munuki. Juba Teaching Hospital is one of three teaching hospitals in South Sudan, Kator and Munuki are Primary Health Care Centers (PHCCs) located in the suburbs of the city.

Eligibility criteria and sampling:

We sampled consecutively 102 tuberculosis patients from the three TBMUs.

Data collection and management

For each study participant a questionnaire with relevant information was completed. Data collection was done by two doctors who work for the TB programme and one medical assistant who works as a state TB supervisor.

Data entry and analysis

The data was entered (double entry) into EpiData version 3.1 software and exported to SPSS (Statistical Program for Social Sciences) version 17.0 for analysis, while ensuring data quality.

Significance of dichotomous and categorical variables tested using chi-square tests; continuous variables tested using t-tests. An alpha level of <0.05 was considered significant.

Ethical consideration

Authorization to conduct the study was obtained from the Ministry of Health, Directorate of Preventive Health Services. The administration in all three TBMUs was notified and provided approval for the study. Only patients willing to be interviewed participated in the study.

Results

Characteristics of study participants

The total number of respondents was 102, consisting of 52.9% female and 47.1% male. All participants were over 15 years old. Almost seventy percent (69.9%) did not attain formal education.

Participants from the three centres in Juba were comparable except that more patients from Kator TBMU did not know about correct TB symptoms compared to those receiving TB treatment from Juba Teaching Hospital or Munuki TBMU. A greater proportion of patients from Munuki TBMU experienced discrimination from the community due to TB compared to those from Kator of JTH. More patients from JTH visited private practitioners compared to those from Kator or Munuki and more patients from Kator than JTH or Munuki had to remind the HCW to check for TB. The details of the findings is set out in Table 1.

Knowledge of tuberculosis patients on disease TB and its management (see figure 1)

On cause of TB, 80.4% of patients interviewed did not relate causation of TB to a germ but rather to other causes such as cat fur. Eighty one (79.4%) respondents correctly related transmission of TB through coughing. Regarding knowledge of correct disease diagnosis, 87.3% knew they had TB. Regarding symptoms of TB, 52.0% were not knowledgeable of correct symptoms of TB such as prolonged cough. On treatment 39.6% did not know that TB treatment duration is six months. Fifty six respondents (54.9%) did not know the importance of adherence to treatment, that is, cure and or prevention of development of a form of TB that is difficult to treat/resistant TB or more dangerous form of disease.

Attitude of TB patients (see figure 2)

Ninety six (94.1%) respondents believed that TB disease

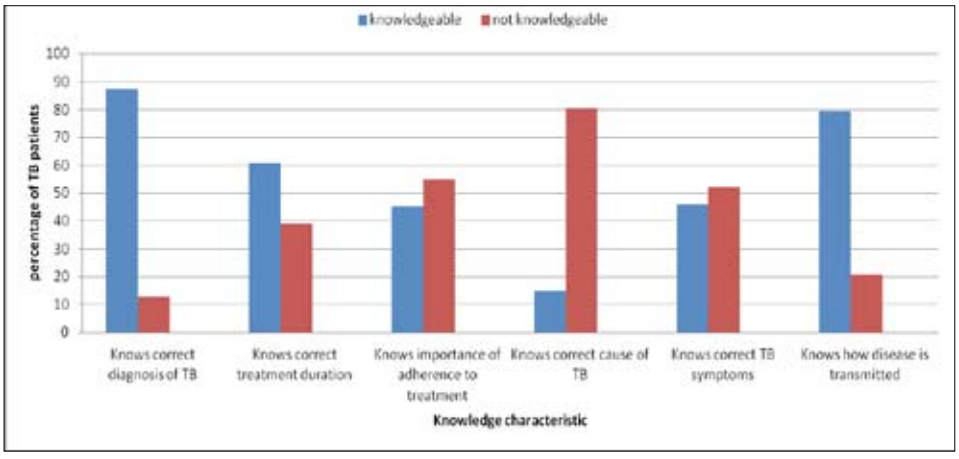


Figure 1. Graphic representation of knowledge of TB patients, Juba, South Sudan, N=102

Table 1 Characteristics of study participants, Juba, South Sudan, N= 102

Characteristic	JTH, N=40 n (%)	Munuki TBMU N=32, n (%)	Kator TBMU N=30, n (%)	P-value
Sex				
F	24 (60.0)	15 (46.9)	15 (50.0)	
M	16 (40.0)	17 (53.1)	15 (50.0)	0.502
Knows correct diagnosis				
Yes	33 (82.5)	28 (87.5)	28 (93.3)	
No	07 (17.5)	04 (12.5)	02 (06.7)	0.404
Knows treatment duration				
Yes	20 (50.0)	23 (61.9)	19 (63.3)	
No	20 (50.0)	09 (28.1)	11 (36.7)	0.158
Knows cause of TB				
Yes	06 (15.0)	06 (18.8)	03 (10.0)	
No	30 (75.0)	25 (78.1)	27 (70.0)	
Undecided	04 (10.0)	01 (03.1)	00 (00.0)	0.275
Knows correct TB symptoms				
Yes	24 (60.0)	16 (50.0)	07 (23.3)	
No	16 (40.0)	14 (43.7)	23 (76.7)	0.050
Knows transmission prevention				
Yes	31 (77.5)	24 (75.0)	26 (86.7)	
No	09 (22.5)	08 (15.0)	04 (13.3)	0.488
Reported contact with a case of TB				
Yes	11 (27.5)	04 (12.5)	05 (16.7)	
No	29 (72.5)	28 (87.5)	25 (83.3)	0.250
Presented Sputum for follow up				
Yes	24 (60.0)	6 (50.5)	14 (70.0)	
No	16 (40.0)	16 (50.0)	16 (30.0)	0.500
Experienced stigma due to TB				
Yes	01 (02.5)	04 (12.5)	00 (0.00)	
No	39 (97.5)	28 (87.5)	30 (100)	0.05
Point of first stop				
Public	30 (75.0)	31 (96.9)	29 (96.7)	
Private modern	08 (20.0)	01 (03.1)	01 (03.3)	
Traditional	02 (05.0)	00 (0.00)	00 (0.00)	0.023
Has at least primary education				
Yes	13 (32.5)	11 (34.4)	07 (23.3)	
No	27 (67.5)	21 (65.6)	23 (76.4)	0.597
Paid for TB test				
Yes	00 (0.0)	0 (0.00)	00 (0.00)	
No	40 (100)	32 (100)	30 (100)	-
Paid for TB drugs				
Yes	01 (02.5)	00 (0.00)	00 (0.00)	
No	39 (97.5)	32 (100)	30 (100)	0.457
TB affected Job				
Yes	11 (27.5)	07 (21.9)	06 (20.0)	
No	29 (72.5)	25 (78.1)	24 (80.0)	0.738
Knows TB is curable				
Yes	37 (92.5)	29 (90.6)	30 (100)	
No	02 (7.5)	03 (09.4)	00 (0.0)	0.344
Received family support				
Yes	36 (90.0)	30 (93.6)	28 (93.3)	
No	04 (10.0)	02 (06.4)	02 (06.7)	0.808
Mobilized community to fight TB				
Yes	37 (92.5)	30 (93.8)	29 (96.7)	
No	03 (07.5)	02 (06.3)	01 (03.3	0.760

Counseled for HIV testing				
Yes	25 (62.5)	25 (83.3)	26 (80.0)	
No	14 (37.5)	07 (16.7)	04 (20.0)	0.172
HCW suspect TB in the first time				
Yes	37 (92.5)	31 (96.9)	29 (96.7)	
No	03 (07.5)	01 (03.1)	01 (03.3)	0.621
Counseled on TB drugs				
Yes	37 (92.5)	30 (93.8)	28 (93.3)	
No	03 (07.5)	02 (6.2)	02 (0.7)	0.977
Disclosed TB to family/friends				
Yes	06 (15.0)	01 (03.1)	02 (06.7)	
No	34 (85.0)	31 (96.9)	28 (93.3)	0.185

is curable and 98% thought that modern health care facilities were the places to seek cure from as opposed to traditional settings.

Support during treatment as viewed by the patient was positive/acceptable from family (92.2%), community (95.1%) and employers (98%).

Practices of TB patients and health workers in TB facilities (see Figure 3)

On prevention on the spread of TB 94.1% respondents were able to perform at least one task to stop spread of disease and 79.4% used a form of personal protective measure like a handkerchief when coughing. Accessing free TB testing was practiced in 100% of cases and on access to free drugs in 99% of cases.

Health care workers correctly suspected TB on first contact in 95.1% of cases. On health education, 93.1% of patients were educated on TB drug side effects, but 24.5% were not educated by the health worker on the need to have a HIV test following TB diagnosis. A total of 93 respondents (91.2%) did not disclose to members of the community or family that they have the disease TB but rather preferred to call their illness other names such as chronic cough or chest disease.

Discussion

Knowledge of TB patients

In this pilot study we documented a pattern showing gaps in tuberculosis patients' knowledge on TB. We found eight in ten of the patients not knowing that TB is caused by a germ or an agent that is transmissible from an infected person to another. They rather related it to community beliefs such as inhalation of fur of cats. However, despite lack of knowledge about the aetiologic agent, nearly 80% had knowledge about transmission of TB, as they were able to relate it to cough. In addition, about 87% still knew that the disease they were suffering from was TB. In a related study carried out in East Shao Zone of Ethiopia during the same year, a smaller number of respondents (69%) did not relate TB to a germ as an aetiologic agent [3]. Knowledge gaps on the cause of TB could be related to low literacy rates in this country. South Sudan has literacy rates of only 27% in those 15 years and older according to the World Bank [4]. Similarly, in this study we also found out that about 70% of the respondents did not attain formal education (not enrolled into primary school).

Juba city accounted for 46% of all defaulters notified to the National Tuberculosis Control Program in the 2010 report [2]. Treatment default in Juba has been linked to the city status of the town with difficult access to the suburbs and the very high number of organized forces who are highly mobile. However, critical gaps in knowledge of patients on treatment may suggest otherwise. In this study we found out that about 40% of the respondents did not know that the duration for TB treatment was at least six months and about 55% did not know the importance of strict adherence to treatment. Key knowledge gaps in TB treatment could be responsible for the high treatment default rates witnessed in Juba prior to the study. Despite the low literacy rates in the country, we suggest that rigorous health education by health care workers on TB during the time of enrollment to treatment may improve patients' knowledge thus improvement in treatment outcomes and overall TB control.

Attitude and practices of TB patients and health workers

Tuberculosis carries a high stigma not only in South Sudan but globally, partly due to its association with HIV/AIDS and the chronic nature of the illness. Anecdotal evidence suggests that in some communities in South Sudan, presence of a TB patient in a family may deter members of the community from marrying from such a family. In this study although, 94% of the respondent believed that TB is curable, up to 91% did not disclose to members of the community or family that they have the disease TB, but rather preferred to call their illness other names such as chronic cough or chest disease. We fell short of exploring further reasons into this diversion, but could be linked to awareness of stigma within the community. Thus, it is not surprising that families, communities and the employers provided sufficient support to the patients

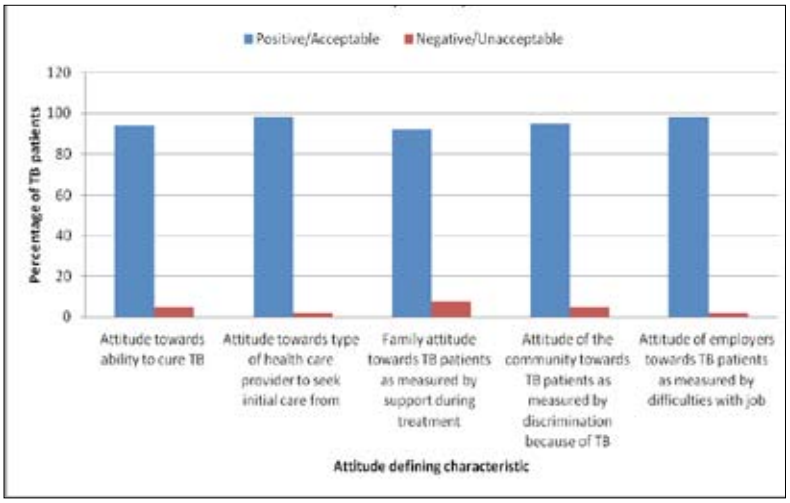


Figure 2. Graphic representation of attitude defining characteristic, Juba, South Sudan, 2010, N=102

during the course of illness [support during treatment as viewed by the patient was positive from family (92.2%), community (95.1%) and employers (98%)].

The National Tuberculosis guidelines South Sudan; recommends health workers in the TB programme provide health education to TB patients on such things as type of disease, cause, transmission, treatment duration, drugs used and side effects and provide HIV/AIDS Provider Initiated Counselling and Testing (PITC). In this study it was evident that patients are being provided services free of charge as 100% of the respondents were not charged for TB test and 99% for treatment. In addition, 94% of the patients performed at least one task to prevent TB spread and 79% used protective items to stop spread of disease. However, one in four were not offered the opportunity to test for HIV by the health worker despite the fact that the guidelines recommends that all patients be offered PITC at enrollment. In the National Program report 2010, only 57% of patients in South Sudan knew their HIV/AIDS status [3]. One of the reasons for the low testing is probably related to health care workers not offering the test to the patients despite other reasons like irregular supply of kits and lack of human resources.

Conclusion

We found key knowledge gaps among Juba TB patients enrolled on treatment. These knowledge gaps are probably responsible for the high treatment defaulter rates reported in Juba, South Sudan. Tuberculosis patients are still not interested to freely reveal disease diagnosis to members of the family and community at large.

Recommendations

- Use of Standard Operating Procedures (SOPs) for nurses in the TB program on health education for TB patients at the three Juba centers.
- Monthly support supervision visits from the central unit of the TB program in the Ministry of Health to the three TB units,
- A Tuberculosis Knowledge Attitude and Practice (KAP) study to be carried out involving the entire country with emphasis on stigma and discrimination.

Acknowledgements

To co-authors Drs. Godwin Ohisa, Taban Martin and Mr. Dila Mathew Loboka for data collection and entry; to Dr. Macharia Stephen for study design and Dr Lasu Joseph for editing the study protocol. Also all the patients who took part in the study, and the health care workers in Juba Teaching Hospital, Munuki PHCC and Kator PHCC who took part in the study.

References

1. World Health Organization. Global Tuberculosis Control: WHO Report, 2013. Geneva, Switzerland: World Health Organization; 2013.
2. Ministry of Health, Republic of South Sudan. National Tuberculosis, Leprosy and Buruli Ulcer Control Program: Tuberculosis Data base, 2012. Juba, Republic of South Sudan (Unpublished).
3. T Kura; T Gedif; Z Tadesse. Ethiopian Pharmaceutical Journal; Assessment of Knowledge, Attitude and Practice about Tuberculosis and its treatment among patients and community in East Shoa Zone, Oromyia Regional State, Ethiopia; 28, 1, 2010.
4. The World Bank. South Sudan Overview, April 2013: http://www.worldbank.org/en/country/southsudan/overview

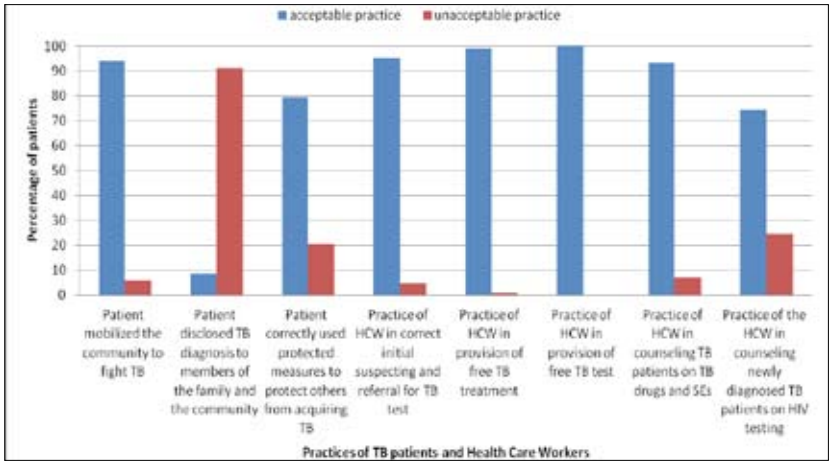


Figure 3. Practices of TB patients, Juba, South Sudan, 2010, N=102

Clostridium difficile

Emily Macnaughton[a]

a Consultant Microbiologist, St Mary's Hospital, Isle of Wight, UK

Correspondence: emily.macnaughton@iow.nhs.uk

Introduction

Clostridium difficile is an anaerobic, spore forming Gram positive bacillus which is a worldwide enteric pathogen. It is a common cause of antibiotic associated diarrhoea and colitis and was identified as the cause of antibiotic associated pseudomembranous colitis in the late 1970s.

Since 2000, *C. difficile* infection (CDI) has had a higher profile following reports of increasing rates, mortality and morbidity, and outbreaks have presented significant challenges to many healthcare facilities. CDI was initially associated with hospital acquired infection but community acquired infections are being increasingly recognised.

Most of the literature surrounding CDI epidemiology relates to the disease in developed countries but it seems unlikely that this pathogen will not have the potential to cause problems in developing countries like South Sudan, particularly on a background of unregulated antibiotic use.

Pathogenesis

Colonization with *C. difficile* occurs following disruption of the normal intestinal flora which usually provides resistance to such colonization, particularly following antibiotic use. *C. difficile* is acquired by the faecal-oral route; the bacterial spores are not destroyed by gastric acid, enabling them to reach the intestines.

The development of active CDI requires toxin production. There are two main toxins (A and B) produced by *C. difficile,* both of which can cause symptomatic disease independently of each other. The toxins disrupt the cytoskeleton of the intestinal mucosal cells, leading to intestinal fluid secretion, damage of the intestinal mucosa and inflammation.

There are many different strains of *C. difficile* and certain strains are associated with higher virulence, particularly the North American type 1 (NAP1)/027 ribotype, first reported in 2003 from Canada [1], which has been linked to more symptomatic disease and more severe presentations.

Epidemiology

C. difficile has been isolated globally from environmental sources both in and outside the hospital. Although most of the research comes from industrialized countries, one study in rural Zimbabwe found toxigenic *C. difficile* in samples of soil, chicken faeces and water [2], highlighting the potential for acquisition of the organism in Africa.

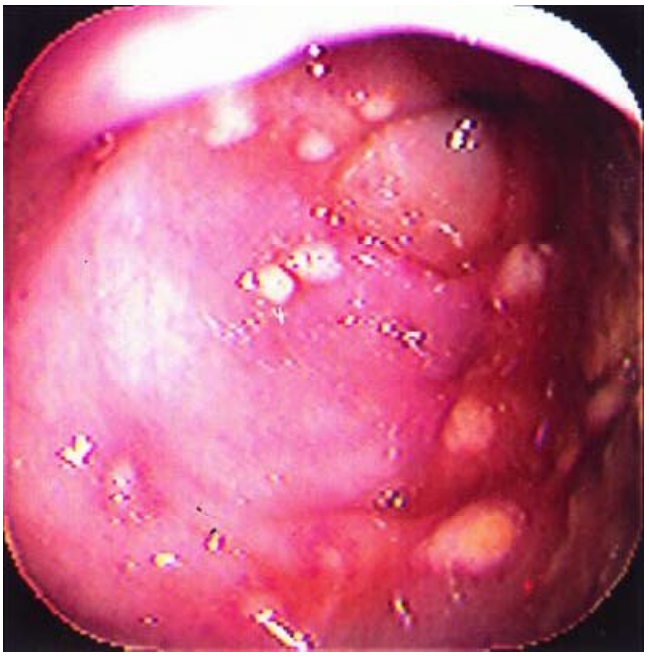


Figure 1. Pseudomembranous colitis at colonoscopy with multiple yellowish patches ("pseudomembranes") and erythematous, friable mucosa. (From Limaye *et al*, 2000].

Asymptomatic colonisation is common in children, reported in up to 70% of healthy neonates [3], compared to around 3% of healthy adults and 20% of hospitalized adults [4]. Some studies have indicated geographical variation in *C. difficile* colonization and infection rates, with some suggesting that the risk in Africa is lower. One study comparing HIV positive adults with diarrhoea in London, UK and Lusaka, Zambia found no patients with CDI in Lusaka, compared to 11% in London [5]. However, *C. difficile* colonisation was detected in 48.8% of Nigerian neonates and children in an earlier study [6], and another more recent Nigerian study of HIV positive patients with diarrhoea demonstrated CDI in 14% of outpatients and 43.5% of inpatients [7].

The major risk factor for developing *C. difficile* infection is receiving antibiotic therapy. Broader spectrum antibiotics are associated with a higher risk of *C. difficile,* causing

more disruption of the intestinal flora. The "four Cs" are a particular risk: clindamycin, ciprofloxacin (and other quinolones), cephalosporins and co-amoxiclav. A higher number of antimicrobial agents, doses and duration are all associated with increased CDI risk [8].

The current lack of publications demonstrating a significant CDI problem in many African countries is likely to reflect a lack of detection and reporting rather than a lack of risk, given that the organism is present in the continent and antibiotic use in many African countries is high, with uncontrolled over the counter access without prescription.

Age, co-morbidities, including malignancy and chronic renal disease, and other medications such as chemotherapy agents are also recognised factors increasing the risk of CDI. HIV infection by itself is not thought to increase the risk of CDI, although these patients are more likely to receive antibiotics leading to an increased CDI risk [4].

Presentation

Although many remain asymptomatic with *C. difficile* colonisation, toxin production results in presentations ranging from mild, transient diarrhoea to severe diarrhoea with a pseudomembranous colitis (Figure 1) which can be fatal. Abdominal pain and non-specific signs may be present, including fever, nausea, anorexia and dehydration.

Occasionally, severe disease can present without diarrhoea, with an acute abdomen, peritonitis or toxic megacolon (dilatation of the colon to greater than 6cm without obstruction with systemic signs of toxicity) which is associated with a high mortality rate.

Recurrences are common, occurring in around 20% of patients, and complications include perforation of the colon, transverse colonic volvulus and protein losing enteropathy. Extra-intestinal manifestations of CDI are rare.

Diagnosis of C. difficile

CDI diagnosis involves detecting the organism and its toxin production from diarrhoeal specimens.

One common testing strategy uses a two step algorithm in which an enzyme immune-assay (EIA) for glutamate dehydrogenase (GDH) , an enzyme with a high sensitivity for the presence of C. difficile, is performed first, followed by a *C. difficile* toxin EIA in the GDH positive samples [9). In resource limited settings, such as South Sudan, use of the toxin EIA alone may be more appropriate given the rapid turn around and reagent availability. Other techniques include culture, tissue culture cytotoxicity testing and toxin gene detection by polymerase chain reaction.

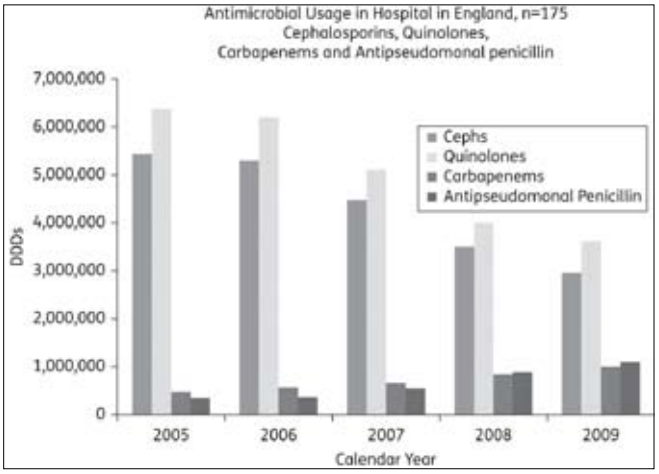


Figure 2: Antimicrobial Usage in Hospital in England 2005-2009, Cephalosporins, Quinolones, Carbapenems and Anti-pseudomonal penicillins. (From: Ashiru-Oredope et al 2012]

DDDs: defined daily doses

Treatment

Treating CDI involves firstly stopping any non-essential systemic antibiotics and secondly using a specific agent against *C. difficile*. The two agents with most evidence for successful treatment of CDI are metronidazole and vancomycin. Metronidazole is used in non-severe cases of infection and is normally administered orally or can be given intravenously if there is a reason for the oral drug not reaching the affected bowel, e.g. intestinal obstruction.

Vancomycin is usually used for more severe infections, although the new agent fidaxomicin is now being used in some centres. Vancomycin is given orally as intravenous vancomycin is not secreted into the intestinal lumen. Patients with unresponsive or recurrent disease can be difficult to treat and approaches include tapering courses of vancomycin, intravenous immunoglobulin, and donor "faecal transplant" therapy, for which there is a growing body of evidence.

Prevention

Strategies to prevent CDI include controlling the spread of *C. difficile*, particularly within healthcare facilities, and minimizing antibiotic exposure, thereby avoiding the disruption of colonic flora which leaves the patient at risk of *C. difficile* colonization. Preventing the spread in hospitals involves side room isolation of patients with suspected or confirmed CDI with barrier nursing, effective cleaning of the patient's environment and careful hand hygiene practices by healthcare workers, with soap and water.

CDI reduction has been a key factor in driving the development of antimicrobial stewardship programmes around the world, along with increasing antimicrobial resistance. Restricting the use of antibiotics, both by minimizing overall antibiotic consumption and by selecting the narrower spectrum lower risk agents is therefore an important method in reducing the problem. In the UK, rates of CDI and mortality from CDI have declined significantly, following improvements in infection control and increased antibiotic stewardship. Figure 2 illustrates the decrease in England in prescription of cephalosporins and quinolones (high risk antibiotics), which is mirrored by a decrease in reported CDI cases over a similar time period (Figure 3).

In summary, CDI is an important cause of diarrhoea, particularly antibiotic associated, which can result in severe disease. Countries without effective control of antibiotic use have a high potential for problems with this organism and it should be considered in the differential diagnosis of patients with diarrhoea.

References

1. Loo et al. A Predominantly Clonal Multi-Institutional Outbreak of Clostridium difficile –Associated Diarrhea with High Morbidity and Mortality. *N Engl J Med* 2005; 353: 2442-9
2. Simango C. Prevalence of Clostridium difficile in the environment in a rural communityin Zimbabwe. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* 2006; 100: 1146—1150
3. Al-Jumaili IJ et al. Incidence and Origin Of Clostridium difficile in Neonates. *J. Clin. Microbiol.* 1984; 19: 177-78
4. Hutin Y et al. Prevalence of and Risk Factors for Clostridium difficile Colonization at Admission to an Infectious Diseases Ward. *Clin Infect Dis* 1997; 24:920-924
5. Zulu I et al. Contrasting incidence of Clostridium difficile and other enteropathogens inAIDS patients in London and Lusaka. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* 2000; 94: 167-8
6. Oguike JU, Emeruwa AC. Incidence of Clostridium difficile in infants in rural and urban areas of Nigeria. *Microbiologica.* 1990 Jul;13(3):267-71.
7. Onwueme K et al. High prevalence of toxinogenic Clostridium difficile in Nigerian adult HIV patients. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* 2011; 105: 667– 669
8. Cohen SH et al. Clinical Practice Guidelines for Clostridium difficile Infection in Adults: 2010 Update by the Society for Healthcare Epidemiology of America (SHEA) and the Infectious Diseases Society of America (IDSA). *Infection Control and Hospital Epidemiology.* 2010; 31(5): 431-455
9. Department of Health. Updated Guidance on the Diagnosis and Reporting of Clostridium Difficile. 2012. Accessed December 2013 at: < https://www.gov.uk/government/publications/updated-guidance-on-the-diagnosis-and-reporting-of-clostridium-difficile

Permission obtained to reproduce all figures. Sources:

- Figure 1. Limaye AP et al. Case reports: Pseudomembranous Colitis Caused by a Toxin A2 B1Strain of Clostridium difficile. *J Clin Microbiol.* 2000; 38(4): 1696–1697.
- Figure 2. Ashiru-Oredope D et al. Improving the quality of antibiotic prescribing in the NHS by developing a new Antimicrobial Stewardship Programme: Start Smart—Then Focus. *J Antimicrob Chemother.* 2012; 67 Suppl 1: i51–i63

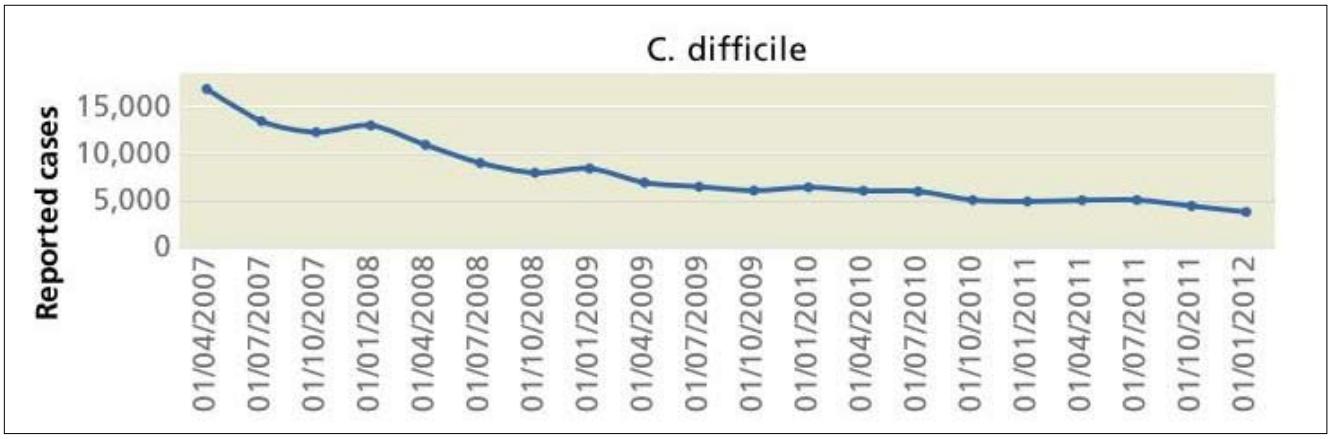


Figure 3: Decline in reported C. difficile cases in England 2007-2012. [From: Davies, SC. Annual report of the chief medical officer. Volume 2, 2011. In: Infections and the rise of antimicrobial resistance. London: DoH, 2013]

Hepatitis E Virus

Christina Levick[a,]

a Post Gastroenterology Specialty Registrar, Queen Alexandra Hospital, Portsmouth, UK

Correspondence: christina.levick@gmail.com

Abstract

Hepatitis E virus (HEV) is the most common cause of acute viral hepatitis in the developing world. It is a waterborne virus that can cause epidemics in the face of overcrowding and poor sanitation. Although the hepatitis illness is usually self-limiting, it has a high mortality in pregnant women and can become a chronic infection in the immunosuppressed. Treatment is mostly supportive and prevention is by good water hygiene.

Introduction

Before the discovery of hepatitis E virus (HEV), many epidemics of hepatitis in the developing world were found to be from a cause other then the known hepatitis A, B and C viruses. HEV was discovered in 1983 in the stool samples of a human volunteer infected with the combined stool samples of patients with non-A, non-B hepatitis (Balayan et al., 1983). HEV is a small single-stranded RNA virus spread by contaminated water.

Epidemiology

HEV is the most common cause of clinical hepatitis in many countries in Africa, including South Sudan, and Asia. There are four known genotypes of HEV that can infect humans (Smith et al. 2013). Figure 1 illustrates the prevalence of the different HEV genotypes across the world. It should be noted that there may be some inaccuracy in the epidemiology of HEV due to the variation in the sensitivity of various anti-HEV IgG testing kits and the lack of standardization of testing (Zhou et al., 2013).

Genotypes 1 and 2 affect humans alone. They are transmitted via the faecal-oral route and survive in water. Consequently, the places of highest prevalence are areas of over crowding and poor sanitation (Hazam et al., 2010). They are endemic in the developing world, with genotype 1 being the most common cause of HEV outbreaks. Notable outbreaks include the refugee camps in Maban County, South Sudan, 2012.

Genotypes 3 and 4 affect humans and animals e.g. pigs, game. These genotypes are most commonly seen in the developed world, although low level prevalence of genotype 3 HEV has been found in locally farmed pigs in Cameroon (S de Paula et al., 2013). They are transmitted across species by consuming the raw or undercooked meat of an infected animal (Tei et al., 2003, Emerson et al., 2005). Transmission may also occur rarely between humans. This has been documented by blood transfusions (Matsubayashi, 2004).

A further two more genotypes have been discovered that do not affect humans, but can infect wild boar (Smith et al., 2013).

Acute HEV infection

HEV infection can range from asymptomatic to fulminant hepatitis. The incubation period from infection to symptoms ranges from two to eight weeks (Purcell and Emerson, 2008). Symptoms include fever, fatigue, myalgia, arthralgia, weakness, vomiting, jaundice, pale stools and dark urine. Neurological symptoms, e.g. from peripheral neuropathy,may also occur (Kamar et al, 2011). Blood tests reveal raised liver transaminases, alkaline phosphatase, γ-glutamyltransferase and bilirubin.

Acute HEV infection can be diagnosed by the presence of anti-HEV IgM, whilst IgG suggests past infection. Due to the wide variation in the accuracy of serological testing, viral Polymerase Chain Reaction (PCR) should be used to confirm the diagnosis (Drobenuic et al., 2010).

In HEV endemic areas where HEV genotype 1 predominates, only 20% of HEV infections produce symptomatic hepatitis. HEV infection rarely causes clinical symptoms in children (Buti et al., 2008), in contrast to pregnant women who have the highest risk of symptomatic hepatitis.

Pregnant women are a particularly susceptible group to HEV transmission, symptomatic infection and mortality. Mortality is 2% if not pregnant and 20% if pregnant (Rein et al., 2012). This mortality risk is most pronounced in the 3rd trimester when the mortality approaches 31% (Guthmann et al., 2006). The risk of contracting acute HEV infection is increased by HIV infection in pregnancy (Caron et al., 2012).

Pre-existing liver disease increases the risk of fulminant hepatitis. The mortality from HEV genotype 1 infection as a cause of decompensation of chronic liver disease is almost 70%, which is considerably higher than other causes of decompensation (Kumar et al., 2004).

HEV infection only produces one symptomatic episode

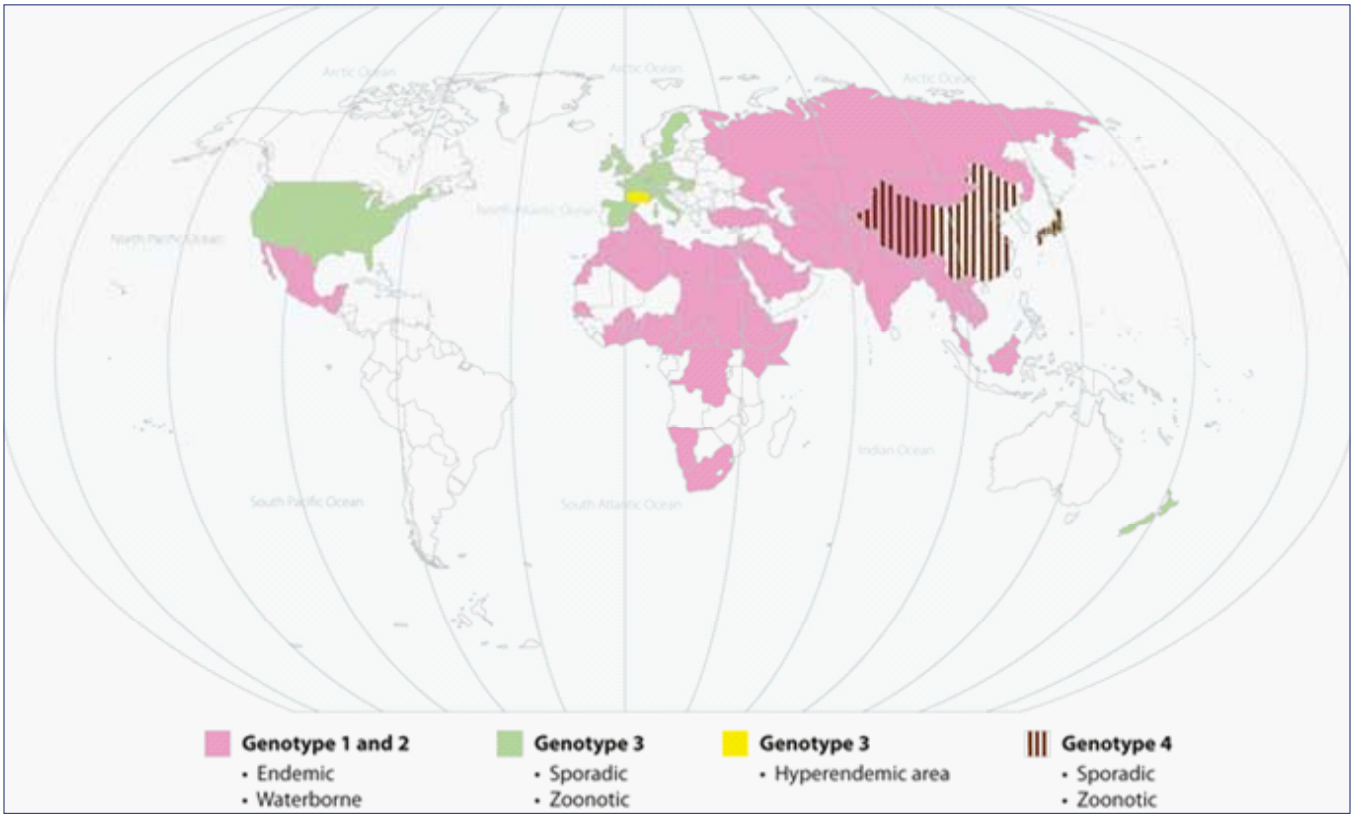


Figure 1. Geographic distribution of human cases of Hepatitis E (Reprinted from The Lancet; 379(9835), Kamar et al., 2477–2488, Hepatitis E, Copyright 2012, with permission from Elsevier)

due to the generation of anti-HEV IgG antibodies.

Chronic HEV

In the majority of cases acute HEV infection will be cleared. An immunocompromised status may lead to the persistence of the virus and chronic HEV infection. This is particularly the case for patients with HIV (Dalton et al., 2009), organ transplants (Kamar et al., 2008) and haematological malignancy. HEV infection in this group is more difficult to confirm as the standard antibody testing for HEV infection is unreliable due to the underlying immunocompromise and so direct molecular assays should be used. To date, only genotype 3 HEV has been shown to cause chronic infection.

Treatment

HEV infection is usually self-limiting and so only supportive treatment is needed. Severe infection can be treated with ribavirin, although ribavirin is contraindicated in pregnancy due to teratogenicity and fetal loss. The approach to treating chronic infection in transplant patients is first to reduce the immunosuppression to allow clearance by the host (Kamar et al., 2010), then to treat with ribavirin and/or pegylated alpha interferon for three months if infection remains (Scobie and Dalton, 2013).

Prevention and Vaccine

The main way to prevent HEV infection in developing countries is by maintaining good hygiene and to supply a safe water source. Chlorination of water has not been shown to inactivate HEV (Guthmann et al., 2006), but boiling water will kill the virus (Emerson et al., 2005).

Human and animal studies have shown the development of antibodies to HEV post infection that protect against re-infection (Bryan et al., 1994, Tsarev et al., 1994). Following subsequent work, two HEV vaccines have been developed.

The HEV 239 vaccine (Hecolin®) was shown to induce immediate immunity to HEV following two doses within one month with 100% efficacy against symptomatic HEV, lasting five months until the third dose (Shrestha et al., 2007). This supports its use in limiting an outbreak of HEV. No adverse effects of the vaccine have been observed in pregnancy (Wu et al., 2012) and its efficacy and safety have been confirmed in a phase III trial (Zhu et al., 2010). Pregnant women or women of childbearing age could be targeted for vaccination in the face of limited resources. The HEV 239 vaccine is currently licensed for use in China, but unfortunately is not part of the WHO priority vaccines.

Comparison with hepatitis A virus

There are many similarities between HEV and hepatitis A virus (HAV). Both HEV and hepatitis A virus (HAV) are enteric viruses spread by contaminated water. HAV is only found to infect human and non-human primates, whereas HEV has a wide variety of zoonotic hosts, depending on its genotype (Purcell et al., 2008). The clinical presentation of HEV is very similar to that of HAV. There are only a few contrasting points. The incubation period of HEV is roughly ten days longer than that of HAV. The severity of disease and mortality tends to increase with age for HAV, whereas age has no bearing on HEV mortality. HEV does however, have a high mortality in pregnant women that is not seen in HAV. Finally, HAV hepatitis can relapse in contrast to HEV, which only causes a single episode of clinical illness.

Conclusion

HEV infection is a major cause of morbidity in areas of over crowding and poor water hygiene. Pregnant women and patients with preexisting liver disease are most likely to develop fulminant hepatitis, with an associated high mortality. They could therefore be targeted for vaccination in endemic areas. Ultimately prevention of transmission by good sanitation and boiling drinking water is the best approach to reduce morbidity and mortality from HEV and a number of other waterborne pathogens.

References

1. Balayan MS, Andjaparidze AG, Savinskaya SS, et al. Evidence for a virus in non-A, non-B hepatitis transmitted via the fecal-oral route. *Intervirology* 1983;20:23–31
2. Bryan JP, Tsarev SA, Iqbal M, Ticehurst J, Emerson S, Ahmed A, et al. Epidemic hepatitis E in Pakistan: patterns of serologic response and evidence that antibody to hepatitis E virus protects against disease. *J Infect Dis.* 1994;170(3):517–21.
3. Buti M, Plans P, Dominguez A, et al. Prevalence of hepatitis E virus infection in children in the northeast of Spain. *Clin Vaccine Immu- nol* 2008;15:732–734.
4. Caron M, Bouscaillou J, Kazanji M. Acute risk for hepatitis E virus infection among HIV-1-positive pregnant women in central Africa. *Virol J.* 2012 Oct 31;9:254.
5. Dalton HR, Bendall RP, Keane FE, Tedder RS, Ijaz S. Persistent carriage of hepatitis E virus in patients with HIV infection. *N Engl J Med.* 2009;361(10):1025–7.
6. Drobeniuc J, Meng J, Reuter G et al. Serologic assays specific to immunoglobulin M antibodies against hepatitis E virus: pangenotypic evaluation of performances. *Clin Infect Dis* 2010: 51(3): e24–e27
7. Emerson SU, Arankalle VA, Purcell RH. Thermal stability of hepatitis E virus. *J Infect Dis* 2005;192:930–933.
8. Guthmann JP, Klovstad H, Boccia D, et al. A large outbreak of hepatitis E among a displaced population in Darfur, Sudan, 2004: the role of water treatment methods. *Clin Infect Dis.* 2006;42:1685–91.
9. Hazam RK, Singla R, Kishore J, Singh S, Gupta RK, Kar P. Surveillance of hepatitis E virus in sewage and drinking water in a resettlement colony of Delhi: what has been the experience? *Arch Virol* 2010: 155 (8): 1227–1233.
10. Kamar N, Abravanel F, Selves J et al. Influence of immunosuppressive therapy on the natural history of genotype 3 hepatitis-E virus infection after organ transplantation. *Transplantation* 2010: 89(3): 353–360
11. Kamar N, Bendall R, Legrand-Abravanel F et al. Hepatitis E. *Lancet* 2012; 379(9835): 2477–2488.
12. Kamar N, Bendall R, Peron JM et al, Hepatitis Virus E and Neurologic Disorders. *Emerging Infectious Diseases* (wwwcdc/eid); 2011, 17 (2)).
13. Kamar N, Selves J, Mansuy JM, Ouezzani L, Peron JM, Guitard J, et al. Hepatitis E virus and chronic hepatitis in organ-transplant recipients. *N Engl J Med.* 2008;358(8):811–7
14. Kumar A, Aggarwal R, Naik SR, Saraswat V, Ghoshal UC, Naik S. Hepatiti s E virus is responsible for decompensation of chronic liver disease in an endemic region. *Indian J Gastroenterol* 2004: 23(2): 59–62.
15. Matsubayashi K, Nagaoka Y, Sakata H, et al. Transfusion-trans- mitted hepatitis E caused by apparently indigenous hepatitis E virus strain in Hokkaido, Japan. *Transfusion* 2004;44:934–940.
16. Purcell RH, Emerson SU. Hepatitis E: an emerging awareness of an old disease. *J Hepatol* 2008;48:494–503
17. Rein DB, Stevens GA, Theaker J, Wittenborn JS, Wiersma ST. The global burden of hepatitis E virus genotypes 1 and 2 in 2005. *Hepatology.* 2012;55(4):988–97
18. Scobie L and Dalton HR. Hepatitis E: Source and Route of Infection, Clinical Manifestations and New Developments. *J Viral Hepat.* 2013;20(1):1-11.
19. S de Paula V, Wiele M, Mbunkah AH, Daniel AM, Kingsley MT, Schmidt-Chanasit J. Hepatitis E virus genotype 3 strains in domestic pigs, Cameroon.*Emerg Infect Dis.* 2013 Apr;19(4):666-8.
20. Shrestha MP, Scott RM, Joshi DM, Mammen MP Jr, Thapa GB, Thapa N, et al. Safety and efficacy of a recombinant hepatitis E vaccine. *N Engl J Med.* 2007;356(9):895–903
21. Smith DB, Purdy MA, Simmonds P. Genetic variability and the classification of hepatitis E virus. *J Virol.* 2013 Apr; 87(8):4161-9.
22. Tei S, Kitajima N, Takahashi K, et al. Zoonotic transmission of hepatitis E virus from deer to human beings. *Lance*t

2003;362: 371–373.

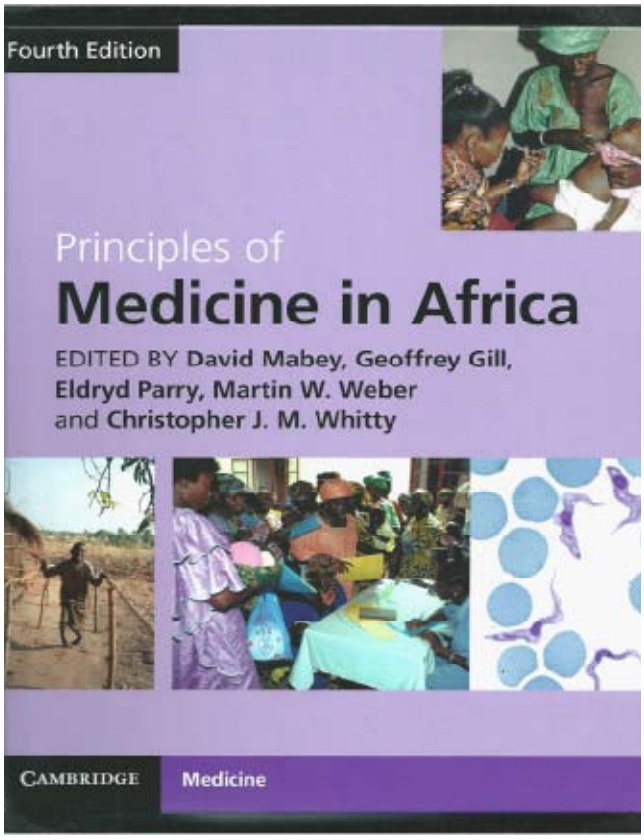
23. Tsarev SA, Tsareva TS, Emerson SU, Govindarajan S, Shapiro M, Gerin JL, et al. Successful passive and active immunization of cynomolgus monkeys against hepatitis *E. Proc Natl Acad Sci U S A.* 1994;91(21):10198–202.

24. Wedemeyer H, Pischk S, Manns M, Pathogenesis and Treatment of Hepatitis E Virus Infection. *Gastroenterology* 2012;142:1388–1397

25. Wu T, Zhu FC, Huang SJ, Zhang XF, Wang ZZ, Zhang J, et al. Safety of the hepatitis E vaccine for pregnant women: a preliminary analysis. *Hepatology.* 2012;55(6):2038

26. Zhao Q, Zhang J, Wu T, Li SW, Ng MH, Xia NS, Shih JW. Antigenic determinants of hepatitis E virus and vaccine-induced immunogenicity and efficacy. *J Gastroenterol.* 2013 Feb;48(2):159-68.

27. Zhu FC, Zhang J, Zhang XF et al. Efficacy and safety of a recombinant hepatitis E vaccine in healthy adults: a large-scale, randomised, doubleblind placebo-controlled, phase 3 trial. *Lancet* 2010: 376(9744): 895– 902.

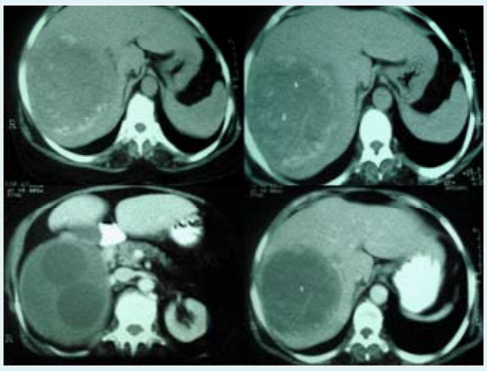


This Book which costs £150 new was donated to the South Sudan Postgraduate Medical Education &Training Library Juba by Dr. Peter Newman, Consultant Neurologist, James Cook University Hospital, Middlesborough. If you have a reference textbook which is less than five years old in Surgery, Medicine, Obstetrics & Gynaecology, Paediatrics & Child Health and Primary Health Care which you wish to donate to this newly established Library please email Dr. Eluzai Hakim with details on eluzaihakim@doctors.org.uk

QUIZ ANSWERS FOR OUR READERS

Lady with fever and hepatomegaly

Case: A 54-year old female from an urban area presented with low grade fever, and vague right upper quadrant abdominal discomfort of one month duration. She was a sweeper by profession and had no close contact with pets. On examination she was febrile; the vital signs were stable and she had no jaundice or other stigmata of chronic liver disease. The liver was palpable to 7cms below the right costal margin and 4cms in the epigastrium with a smooth surface,and was tender to palpation: there were no bruits. Her haemogram and biochemical parameters were normal except for elevated alkaline phosphate levels (323 IU/L). These were the contrast enhanced computerized tomography images (see image).



Questions and answers:

Q1. What is the likely diagnosis?

A. Hydatid cyst of liver.

Q2. What other conditions should be considered?

A. Liver abscess, biliary cystadenoma and hepatoma.

Q3. What is the most likely causative organism?

A. Echinococcusgranulosus.

Q4. What is the most serious complication of this condition?

A. Rupture of a cyst leading to anaphylactic shock.

Q5. Are there any serological tests that would aid diagnosis?

A. Enzyme-linked immunosorbent assay (ELISA): positive in 90%.

Q6. What is the gold standard treatment of the condition?

A. Surgical removal without disruption of cyst contents.

Q7. Which is the preferred drug for the treatment of the condition?

A. Albendazole.

Quiz sent by George Sarin Zacharia

Water, sanitation and hygiene in South Sudan: what needs to be done to bridge the gap?

Victor Vuni Joseph[a]

a Consultant in Public Health, Doncaster Council, England, UK;

Correspondence: vuni.joseph@tinyworld.co.uk

Introduction

Water, and sanitation hygiene (WASH) is a major public health challenge, not only globally, but also in the Republic of South Sudan. It is estimated that 1 in 10 (768 million) of the world's population do not have access to safe drinking water, most of whom are in developing countries, while a third of the world's population (2.5 billion people) do not have access to adequate sanitation [1].

In the developed countries, water and sanitation have been described as one of the first wave of classic public health interventions in the 19th century (1830-1900) [2]. In, England, its importance resulted in the formation of "Health of Towns Association", championed by Mayors of the Cities [3]. In modern day Europe, a new form of Health of Towns Association called Healthy Cities emerged, starting in Liverpool in 1986 [4], and spreading across Europe as "Healthy Cities Network" [5]. This initiative has played an important role in improving the health of the population in those parts of the world. There is, however, a lot for developing countries to do in order to catch up with the challenge of water and sanitation. In addition, there are newer waves of public health interventions to address: biomedical (antibiotics, vaccines), clinical (lifestyle-related diseases), social (social determinants of health) and cultural (culture of health) [2].

In South Sudan, the official statistics indicate that only 15% of household use sanitary means of excreta disposal, and 55% has access to improved drinking water [6]. The picture is likely to be even poorer in rural areas of the country as well as in the overcrowded urban areas of South Sudan (Figure 1).

Although the government set a target of reducing the number of people who did not have sustainable access to safe drinking water and sanitation by 50% in three years from 2010, there appears to be neither government policy nor a safe drinking water programme in place to achieve this target. Coupled with the current conflict which started on the night of 15 December 2013 in the country, the little progress that might have been made in relation to improvements in water and sanitation provision, has been halted, if not reversed, in the parts of South Sudan most affected by the conflict.

For South Sudan to make a developmental leap in the water and sanitation front, a radical approach is needed involving all the relevant stakeholders. A report (2010-2012) of official development assistance (ODA) showed that South Sudan spent 3.5 US dollars per person on water and sanitation, significantly lower than other similar developing countries [7]. The report also showed that there is a significant level of inequalities in access to water and sanitation across the world, and it called for efforts to make water and sanitation universally accessible.

Sanitation has been defined as "the safe disposal of human excreta and associated hygiene promotion" [8]. Improved sanitation can be any one of the following types of toilet systems: flushed toilet, piped sewer system, septic tank, flush / pour flush to pit latrine, ventilated improved pit latrine (VIP), pit latrine with slab or composting toilet [8].

Objectives

The objectives of this paper are to review the evidence base for investments in water and sanitation, and to propose some policy recommendations in order to increase access to safe water and sanitation to the people of South Sudan.

The evidence based related water and sanitation

Two major reviews were examined for evidence related to water and sanitation hygiene, along with other web-based resource on the subject. One report was a review, which mapped out the various models available on sanitation from around the world in an attempt to understand better those models in use, and issues related to their sustainability [9]. The report identified 19 different innovative models of sanitation from various parts of the world, mainly in developing countries in Asia, Africa, and South America. The authors identified a number of challenges in implementing various sanitation programmes including poverty, the need for partnership, the role of trained facilitators (educators) in the community, and the role of the government.

Figure 1. Access to water in Juba during 2006. (credit Victor Vuni Joseph)

Key lessons that can be learned from the sanitation programmes in various countries include some of the following [9]:

- Community mobilisation for each household to build their own sanitation (latrine);
- The need for public subsidy to help communities to build latrines, especially for poor households;
- The use of technological innovation in improving sanitation; e.g. attractiveness, reduction in cost, shapes and sizes, and ease of cleaning;
- Involvement of local and national government, communities and external organisations;
- The need for regulation (laws), guidance and best practice in sanitation;

The World Health Organization [10] also described standards for a simple, and basic form of sanitation (latrine) called the ventilated pit latrine (VIP) that could be adopted in rural areas – see Figure 2.

Another systematic review of the evidence of water, sanitation and hygiene (WASH) for UK Department for International Development (DFID) examined the impact of WASH on health, and non-health areas. It also reviewed the principles associated with effective delivery models of sanitation [11]. They found that, globally, around 2.4 million deaths, and 7% of the disease burden could be prevented by having safe access to WASH. The review found good evidence linking WASH with the following:

- diarrhoeal diseases,
- acute respiratory infections,
- undernutrition, and
- soil-transmitted intestinal helminthic infections.

WASH was also linked to non-health impacts, and there were good or suggestive evidence on improving menstrual hygiene in women, violence against women and insecurity, and school attendance, especially among girls [11]. The authors considered that WASH was cost-effective in health, economic and development areas, when compared to other types of interventions.

Although the review [11] could not recommend a particular model of WASH, it identified some key principles for intervention aimed at implementing WASH programmes. These included: market research for behavioral change; targeting of subsidies; toilet designed for particular users, and price; urban on site sanitation systems needing to have mechanisms for emptying; having closer water source to households; and sustainability.

Conclusions

WASH is an important public health problem, both globally and in South Sudan. There are a number of innovative models of sanitation to consider for local adoption. The evidence shows that investment in WASH is a cost-effective intervention, with benefits beyond health to non-health areas such as economy and development of the nation. A number of the principles identified could enable South Sudan to formulate concrete actions to address WASH.

What needs to be done?

In light of the above evidence on WASH, and the current status of South Sudan in relation to WASH, it is necessary to adopt an ambitious programme of actions. The following actions are proposed:

1. The Government need to adopt a vision for WASH. Such a vision should be ambitious – such as "access to WASH for all South Sudanese".
2. Consider adoption of "Healthy Cities", "Healthy Villages", and "Healthy Schools" initiatives. City Mayors and Commissioners of counties in South Sudan are central in driving this initiative. Other African countries have already signed up to such initiatives.
3. The need for a Government level, inter-ministerial task force and resource on WASH in the country. WASH impacts on more than the health sector. The task force should be charged with, among others, the following functions:
    a. Recommending the options for WASH, standards and guidance;

Figure 2. A ventilated pit latrine in Buluk area of Juba, South Sudan (credit Victor Vuni Joseph)

b. Approve government subsidies to target population;

c. Agree a more ambitious target over defined time period (e.g. 3-5 years). Such a target must relate to both access to safe water and improved sanitation.

4. There should be legislation underpinning WASH, such as putting a requirement of any household to have an acceptable sanitation, among others.
5. There needs to be a programme of community mobilisation with sanitary officers, public health officers or community development workers charged with the task of educating the community. Campaigns for safe drinking water and sanitation should be carried out over a specific period such as a month to focus minds.
6. Partnership with NGOs in coordinating the efforts to improve WASH programme to the population of South Sudan.

References

1. WATERAID UK (2014) Sanitation: what would life be like without a toilet? 2.5 billion people know only too well. http://www.wateraid.org/uk/what-we-do/the-crisis/sanitation?gclid=CISK-oi727sCFSvjwgod6isAbw
2. Davies, S., Winpenny, E., Ball, S., Fowler, T., Rubin, J. & Nolte, E. (2014) For debate: a new wave in public health improvement. *The Lancet*, 13, 7.
3. Ashton, J. (2000) Governance, Health and the New Citizenship. Inaugural Lecture, Liverpool, Liverpool John Moores University.
4. Ashton, J. (1995) A Vision of Health for the North West: Inaugral Lecture by Professor John R Ashton, 30 January 1995. Liverpool, Liverpool Public Health Observatory, University of Liverpool.
5. World Health Organization (2009) Phase V (2009-2013) of the WHO European Healthy Cities Network. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0009/100989/E92260.pdf. WHO Europe.
6. Government of the Republic of South Sudan (2011) South Sudan Development Plan 2011-2013: Realizing freedom, equality, justice, peace and prosperity for all. Juba, Government of the Republic of South Sudan.
7. Garret, J., Pankoj, K., White, Z., Koundarjaian, H. & Brewer, T. (2014) Bridging the device: using aid flows to tackle inequalities in water and sanitation access. Briefing paper. WaterAid.
8. Roma, E. & Pugh, I. (Undated) Toilets for Health. A report by the London School of Hygiene and Tropical Medicine in Collaborationwith Domestos. London School of Hygiene and Tropical Medicine in Collaborationwith Domestos.
9. Roma, E., Curtis, V., Jones, C. & Milles, P. (2013) Mapping sanitation solutions: a report in collaboration with London School of Hygiene and Tropical Medicine and Domestos, London School of Hygiene and Tropical Medicine.
10. World Health Organization (Undated) VIP and ROEC latrines: WHO standards for VIP latrines. Fact Sheet 3.5. WHO.
11. Cairncross, S., Cumming, O., Jeandron, A., Rheingans, R., Ensink, J., Brown, J., Cavill, S., Baker, S. & Schmidt, W. (2013) Water, sanitation and hygience: evidence paper. London, Department for International Development.

What is cholera?

Reproduced from MoH Cholera Brochure

Cholera

Cholera is an acute diarrhoeal infection caused by ingestion of food or water contaminated with the bacterium *Vibrio cholerae.*

How is cholera spread?

Cholera is spread through eating food or drinking water contaminated with faeces containing the cholera bacteria. Cholera is closely linked to inadequate environmental management.

Sudden large outbreaks are usually caused by a contaminated water supply. Raw or undercooked food may be a source of infection in areas where cholera is prevalent and sanitation is poor.

The absence or shortage of safe water and sufficient sanitation combined with a generally poor environmental status are the main causes of spread of the disease.

What are the risk factors?

The risk factors for cholera are related to poor sanitation and hygiene conditions. These include;

- Poor use of latrines.
- Inadequate clean water supply.
- Contamination of water due to poor storage.
- Raw vegetable and fruits taken from contaminated water or grown on ground level and irrigated with water containing human waste.
- Dirty homestead.

Incubation period

The short incubation period of two hours to five days, enhances the potentially explosive pattern of outbreaks.

Signs and symptoms of cholera:

A person suffering from cholera can develop the following:

- Passing of frequent watery rice like stool with no smell.
- Vomiting in some patients.
- Thirst.
- Body weakness.

Who is at risk of getting cholera?

Cholera should be suspected in outbreaks of waterborne diseases. Any patient aged 2 years or older is at risk.

How can cholera be prevented?

There is a vaccine available for cholera. However this should always be used as an additional public health tool and should not replace the usually recommended control measures such as improved water supplies, adequate sanitation and health education. The most effective ways to prevent cholera are by:

- Washing your hands with soap and clean water before handling food, after using a latrine and after handling children's faeces.
- Cooking food thoroughly and eating it while still hot.
- Boiling all drinking water or treating it with chlorine and storing it in a clean container (e.g jerrycan) with a cover.
- Disposing of all faeces, including children's, into the latrine
- Washing fruits and vegetables before eating them.
- Maintaining a clean environment around homes.
- Always use long hand containers to always pour water from the storage container for drinking. Never dip the drinking cup into the storage container.

What is your role in preventing cholera?

Everyone has a role to play if cholera is to be prevented. Ensure that

- Your hands are always washed with soap and clean water before the handling food, after using latrine and handling children's faeces.
- Food is thoroughly cooked and eaten while still hot.
- All drinking water is boiled or treated with chlorine and stored in a clean container.
- You always pour water from the storage container for drinking. Never dip the drinking cup into the storage container.
- All faeces, including children's are disposed of in the latrine.
- Fruits and vegetables are thoroughly washed before eating them.
- Personal hygiene is observed and homes are kept clean.
- All sick people are taken to the nearest health unit immediately.

Points to remember:

- Cholera is a killer disease that can be prevented.
- Cholera is a spread through eating food or drinking water contaminated with faeces.
- Even though cholera has a vaccine, prevention is the most effective way of avoiding the disease.
- Everyone is at risk of contracting cholera.
- There is treatment for cholera at the health centre/ unit. It will save your life if you seek medical treatment on time.
- Self medication especially the use of local herbs worsens the condition of a patient with cholera. Seek proper medical help.
- Dispose of all faeces, including children's into the latrine.

Notice: International English language Testing System (IELTS) – volunteer teachers wanted

Passing the IELTS Examination with a minimum of 7 scores out of 10 in the following domains:

- Listening: 40 minutes, 30 minutes for which a recording is played centrally and additional 10 minutes for transferring answers onto the OMR answer sheet.
- Reading: 60 minutes.
- Writing: 60 minutes.
- Speaking: 11–15 minutes.

is a pre requisite for admission to the two year Medical Training Initiative (MTI) run by various Royal Colleges and all Institutions of Higher learning in the United Kingdom for overseas doctors, healthcare professionals allied to medicine, nurses and midwives aspiring to pursue postgraduate training in the UK.

Unfortunately several South Sudanese eligible to pursue further training in the UK through the Royal Colleges and other Institutions of Higher learning have been struggling to gain the minimum scores requiredin the UK to gain admission to suitable courses due to lack of facilities to prepare for the IELTS in South Sudan. This examination is conducted several times a year by the British Council in Juba, the capital of South Sudan and can be taken locally by those well prepared to do so.

The UK South Sudan postgraduate Medical Education and Training steering Group is looking for suitable volunteers with experience in teaching English as a foreign Language to undertake three annual teaching sessions each of which is of three weeks duration in Juba starting in March/April 2015. It is envisaged that two volunteers per visit will be sufficient to train 20 candidates over the three weeks. Funding is currently being sought to support the volunteers with air fares, insurance, subsistence and course materials.

Interested volunteers should contact either Dr. Eluzai Hakim, email eluzaihakim@doctors.org.uk OR Dr. Peter Newman, email dr.p.newman@doctors.org.uk

Peter Newman, Chairman, PGMET Steering Group
Eluzai Hakim, Member PGMET Steering Group

The Gordon Memorial College Trust Fund

What is the GMCTF?

The Gordon Memorial College Trust Fund (www.gmctf.org) was established in 1899 by public subscription to honour the name of General Charles George Gordon of the British army, who was killed during the Mahdi's uprising in 1885. Gordon Memorial College, an educational institution in Sudan, was built between 1899 and 1902 as part of Lord Kitchener's wide-ranging educational reforms, and also named in honour of Gordon. In 1924, the college was merged with the new Khartoum University, as was the Kitchener School of Medicine.

The funds of the Gordon Memorial College Trust Fund are held in Trust and administered by a group of Trustees and an Executive Committee. The purpose of the Fund is to promote educational development in South Sudan and Sudan. Grants are available for educational projects and activities in South Sudan and Sudan and for South Sudanese and Sudanese nationals studying for a postgraduate course in the UK, or in counties neighbouring Sudan and South Sudan, who intend to return to South Sudan or Sudan at the end of their studies. The Trust may also give financial assistance to South Sudanese and Sudanese nationals towards the costs of shorter training programmes, projects and courses in the UK. Please note that at present due to current difficulties in transferring funds to South Sudan and Sudan it is not possible for the Trust to support individual students studying in South Sudan or Sudan.

Ministry of Health Resource Materials on Cholera Health Education and Management

- Treatment flowchart for cholera
- How can you control cholera?
- How can you prevent cholera?
- Five steps to protect yourself from cholera

Treatment Flowchart for Cholera Cases Using Standard Case Definition

Any patient ≥ 2 years presenting with acute watery or rice watery diarrhoea with or without vomiting and with signs of dehydration should be suspected as a case of cholera during an outbreak (*children <2 years can also be affected during an outbreak*). Acute watery diarrhea – passage of watery or liquid stools ≥ 3 times in last 24 hours

Management of patients presenting with acute watery diarrhoea

Patient with acute watery diarrhoea

→ Look for other associated symptoms e.g., swelling of legs, respiratory difficulty, anemia etc.

↓ Urgently Inform Doctor

Assessment of the patient for dehydration

Assess	Condition	Normal	Irritable/Less active*	Lethargic / Comatose*
	Eyes	Normal	Sunken	
	Tongue	Normal	Dry	
	Thirst	Normal	Thirsty (drinks eagerly)*	Unable to drink*
	Skin pinch	Normal	Goes back slowly*	Goes back very slowly*
	Radial pulse	Normal	Reduced	Uncountable or absent*
Diagnosis		**No sign of dehydration**	If at least 2 signs including 1 (*) sign is present, diagnose **Moderate Dehydration**	If moderate dehydration plus 1 of the (*) signs are present, diagnose **Severe Dehydration**
Management		A	B	C

A. No sign of dehydration – ORS

- 50 ml ORS per kg body weight over 6 hours *plus* ongoing losses
- Send patient to home with 4 packets of ORS
- Feeding should be continued
- Return if condition does not improve or deteriorates
- Maintain hydration, replace continuing fluid losses until diarrhoea stops

B. Moderate dehydration – ORS

- 80 ml ORS per kg body weight over 4 – 6 hours *plus* ongoing losses
- Patient should be kept under observation for 6 - 12 hours
- Feeding should be continued
- Reassess the dehydration status frequently - hourly.
- In case of frequent vomiting (>3 times in 1 hour): Treat with IV fluid
- Maintain hydration, replace continuing fluid losses until diarrhoea stops

C. Severe dehydration – IV Sodium, potassium, bicarbonate solution (Ringer's lactate)

- Start IV fluid immediately (100 ml/kg)

Children < 1 year: give 100ml/kg IV in 6 hours, as follows

30 ml / kg in the first 1 hour then

70 ml / kg in the next 5 hours

Adults and Children ≥ 1 year: give 100 ml/kg IV in 3 hours, as follows

30 ml / kg as rapidly as possible within 30mn and then

70 ml / kg in the next 2½ hours

- Monitor regularly and reassess rehydration status
- Encourage the patient to take ORS solution (5ml/kg per hour) as soon as he/she is able to drink
- Start antibiotic after initial rehydration (4-6 hours)
- Maintain hydration, replace continuing fluid losses until diarrhoea stops

Antibiotics in cholera outbreak for South Sudan

- Antibiotics should be given **ONLY** to cases with **SEVERE DEHYDRATION**. This should be done under supervision of a **MEDICAL DOCTOR** in a **HEALTH FACILITY**
- Choice of antibiotics depends on local sensitivity pattern

First line drug (except in pregnancy)

- **For adults:** Ciprofloxacin, 1g (500 mg x 2) – single dose after correction of severe dehydration
- **For children:** Ciprofloxacin susp. 20 mg /kg – single dose after cessation of vomiting (if any)

Second line drug

- **For adults:** Azithromycin, 1g (500 mg x 2) – single dose after correction of severe dehydration
- **For children:** Azithromycin susp. 20 mg /kg – single dose after cessation of vomiting (if any)

Alternative drugs

- Doxycycline, 300 mg (100 mg x 3) – single dose after food (*Adults only, except in pregnancy*)

World Health Organization

HOW CAN YOU CONTROL CHOLERA?

What causes CHOLERA ?

- By consuming contaminated food or water
- Eating food contaminated by flies and dust when left uncovered and by dirty hands.
- Fish/seafood taken from contaminated water, eaten raw or insufficiently cooked.
- Consuming ice made from contaminated water.
- Drinking water polluted by seepage from toilets, open defecation, cut and damaged pipes.
- Drinking water at home polluted by dirty hands, collection and storage in dirty containers.
- Eating unwashed fruits and vegetables.
- Eating fruits and vegetables washed with polluted water.
- Fruit and vegetables grown at or near ground level, irrigated with water containing human waste, or "freshened" with contaminated water, and eaten raw.

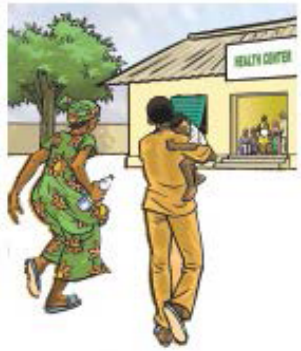
What are the signs and symptoms of CHOLERA?

Passing of frequent watery stool 3 or more times a day

Sometimes with vomiting

Seek medical care immediately

- Cholera can kill. If you pass watery stool 3 or more times and with or without vomiting, you could have Cholera. Immediatey seek treatment from the nearest health facility.
- There is treatment for cholera at Health Facilities. This will save your life if you seek timely medical treatment
- Do not self-medicate from a drug store or a pharmacy.



- Use of local medicine/herbs can worsen the condition of a patient with Cholera. Cholera should only be managed at the health facility.
- Do not touch the dead body of any suspected Cholera or any other diarrheal . case.

World Health Organization

CHOLERA can be treated. Do not panic, but do not wait
Seek EARLY and FREE treatment at the nearest health facility

unicef

HOW CAN YOU PREVENT CHOLERA?

Always wash your hands with soap or ash

- Before and after handling food.
- Before preparation, serving, or eating food. This prevents you from contaminating the food that you consume with germs containing cholera.
- After using the toilet.
- After cleaning your child's bottom.
- After disposing of a child's faeces.
- Before and after eating.
- Before breast-feeding and feeding your child.

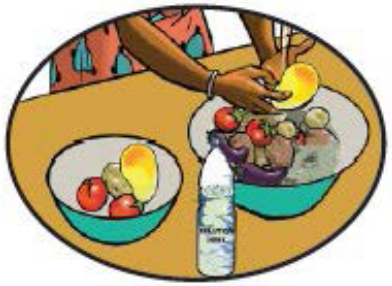
- Wash in between the fingers and under the nails as germs/bacteria can easily hide there.
- Communal washing of hands is extremely dangerous and it should not be done. Pour water for each person separately while washing hands

Always handle your food safely

- Cook food properly.
- Food should be eaten while still hot because the cholera germ cannot survive in very hot and properly cooked food.

- Make sure to separate raw meat and vegetables from cooked food during preparation.
- Keep food in a clean environment and covered at all times. This prevents the contamination of food with the cholera germ and all other germs.
- Avoid drinking, fresh juice and ice. The local brews juice and ice are cold and may be prepared in an unhygienic way and is most likely to have cholera germs.

- Wash all fruits and vegetables thoroughly with chlorine treated water.



- All food handling utensils should be kept clean at all times. Air dry all utensils and avoid using towels or pieces of cloth to dry your utensils.

- Keep your restaurant or kitchen surroundings clean by disposal of left over foods, keep changing your dish washing water.

1. Wash your hands with clean water and soap.

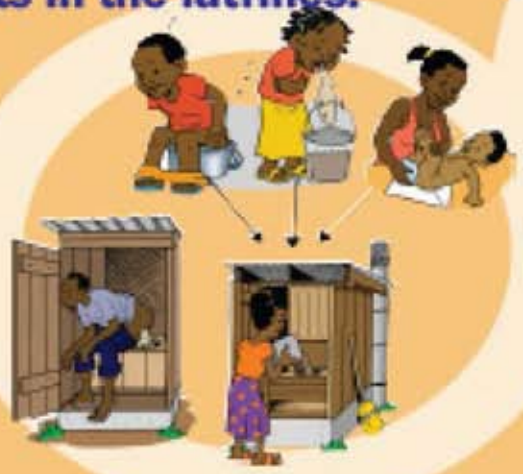
2. Drink purified water.

5 Steps
to protect yourself against
CHOLERA

3. Wash fruits and vegetables with clean water.

4. Cook food properly

5. Dispose faeces and vomits in the latrines.



World Health Organization

unicef