African Journal of Emergency Medicine (2013) **3**, 75–82

African Federation for Emergency Medicine

African Journal of Emergency Medicine

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Emergency centre management of paediatric diarrhoea: An overview

Prise en charge dans les centres d'urgences de la diarrhée pédiatrique: Aperçu

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Received 11 February 2012; revised 4 June 2012; accepted 5 June 2012
Available online 20 July 2012

Keywords
Diarrhea;
Pediatric Emergency;
Management;
Gastroenteritis;
Dehydration

Abstract The worldwide burden of diarrhoeal disease results in 1.5 million deaths annually in children under the age of five. Emergency physicians are often the primary medical provider to care for these children, so a firm understanding of diarrhoeal aetiology, microbiology, risk stratification, and treatment options is crucial. By using a focused history and physical exam with appropriately targeted serum investigations and imaging studies, children may be accurately assessed for degree of dehydration. Ultrasound imaging in particular is emerging as a rapid and reliable tool for this purpose. While oral rehydration remains essential first-line treatment, more severe presentations warrant use of intravenous crystalloid for the correction of fluid deficit. A focus on proper patient disposition and discharge instructions is also critical for prevention of further morbidity and to prevent unnecessary emergency centre returns. This overview of recent literature provides the emergency physician with a basic understanding of the evidence supporting management of paediatric diarrhoea.

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Peer review under responsibility of African Federation for Emergency Medicine.

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http://dx.doi.org/10.1016/j.afjem.2012.06.004

Abstract Le fardeau que représente la maladie diarrhéique à l'échelle mondiale entraîne chaque année 1,5 millions de décès chez les enfants de moins de 5 ans. Les urgentistes sont souvent les premiers fournisseurs de soins médicaux pour ces enfants, ainsi une solide compréhension de l'étiologie, de la microbiologie, de la stratification du risque et des possibilités de traitement de la diarrhée est cruciale. En effectuant une anamnèse ciblée et un examen physique avec des recherches de serum ciblées appropriées et des études par imagerie, le degré de déshydratation des enfants peut être évalué avec précision. L'imagerie par ultrasons en particulier apparaît comme un outil rapide et fiable à cette fin. Alors que la réhydratation orale reste essentielle en tant que premier traitement, des affections plus graves justifient l'utilisation de cristalloïdes par intraveineuse afin de corriger le déficit liquidien. Se concentrer sur des instructions de disposition et de sortie du patient correctes est tout aussi fondamental pour la prévention d'une plus grande morbidité et pour prévenir un retour inutile en service d'urgence. Cette présentation des ouvrages récents fournit à l'urgentiste une compréhension de base des faits probants étayant la prise en charge de la diarrhée pédiatrique.

African relevance

- African countries hold 9 of the 10 highest national under-five-year mortality rates.
- Eighty percent of diarrhoea-related mortalities occur within Africa and southern Asia.
- Over the last decade, progress in the treatment of diarrhoea has been slow in Africa.

What's new?

- Aetiology of diarrhoea is largely dependent on geographic and seasonal factors, with important treatment differences in certain situations.
- Physical exam should focus on capillary refill time, mucous membranes, presence/absence of tears, and overall appearance.
- Ultrasound is a new, inexpensive, rapid, and effective imaging modality to assess dehydration.
- Management of dehydration depends on degree of fluid loss, and intravenous fluids should generally only be used in severe cases.
- Effective parental guidance at discharge is important to prevent worsening of dehydration after an emergency centre admission.

Background

Despite a half-century of public health initiatives, treatment advances, and improved diagnostic tools, acute diarrhoeal illness remains one of the two leading causes of death in paediatric populations.[1,2] According to the most recent estimates from the World Health Organization (WHO), there are approximately 2.5 billion cases of diarrhoeal illness each year. The burden of disease is especially severe in the paediatric population, resulting in 1.5 million deaths annually in children under the age of five. Second only to pneumonia, diarrhoea is responsible for 3% of neonatal mortality and 25% of post-natal mortality. These numbers together account for more paediatric deaths than AIDS, measles, and malaria combined.[2]

A disproportionate burden of this morbidity and mortality falls on developing countries, with 80% of diarrhoea-related mortalities occurring within Southern Asia and Africa.[2] Even among these continents, not all nations are equally affected. Six countries alone (India, Nigeria, China, Pakistan, DR Congo, and Ethiopia) account for 50% of worldwide deaths under 5 years of age.[3]

Over the last 30 years the incidence of diarrhoea-related mortality has dramatically decreased. Following introduction of oral rehydration salts (ORS) and educational programs largely spearheaded by the WHO, diarrhoea-related annual mortality dropped from 13.6 to 4.9 deaths per 1000 children under five.[4] Evidence has shown large-scale diarrhoea and dehydration treatment programs to be effective worldwide; Egypt showed a 74% reduction in infant mortality from 1981 to 1990 and Brazil showed a 67% reduction in infant mortality from 1980 to 1989 through improved awareness and use of oral rehydration.[5] But even with the success of these programs, mortality rates remain unacceptably high. Also, over the last 5 years, global diarrhoea-related child mortality has appeared to remain stable.[6]

Emergency physicians find themselves on front lines of public health. It is therefore essential for practitioners in these settings to be able to determine the general aetiology of diarrhoeal illness while identifying and treating fluid or electrolyte disturbances. Also vital to emergency management is patient disposition, with a focus on prevention and education. In some settings, emergency physicians may be the sole medical provider for the patient; in these cases their role becomes even more crucial.

Definitions

Diarrhoea may be divided into three categories: acute watery diarrhoea, acute haemorrhagic/dysenteric diarrhoea, and persistent/chronic diarrhoea. There is a lack of consensus on the precise definition of each of these terms, and on the definition of diarrhoea itself.[7] While the WHO defines diarrhoea as the

passage of three or more loose stools in a 24 h period (with distinct episodes delineated by >2 consecutive days without diarrhoea), the Infectious Disease Society of America (IDSA) considers diarrhoea as "an alteration in a normal bowel movement characterized by an increase in water content, volume, or frequency of stools," and further characterizes infectious diarrhoea as due to any infectious aetiology, "often accompanied by symptoms of nausea, vomiting, or abdominal cramps".[8] These symptoms overlap with the diagnosis of acute gastroenteritis (AGE), an infectious illness characterized by acute diarrhoea, with or without nausea, vomiting, fever, and abdominal pain.[9] A systematic review by Johnston et al. performed in 2010 highlighted 64 unique definitions and 46 unique primary outcomes used in randomized controlled trials involving paediatric diarrhoea.[10] This presents a challenge to clinicians, requiring increased attentiveness when applying evidence-based decision rules to practice. In clinical settings, the terms AGE and acute diarrhoea are frequently used interchangeably, but it is important to consider non-infectious aetiologies including medication effects or systemic conditions.[11]

Persistent diarrhoea – aetiology

This document will focus on acute disease, generally accepted as an episode lasting <14 days duration.[8] Nonetheless, emergency physicians should be cognizant of conditions which may lead to prolonged courses of disease. A recent study in Turkey examined the cases of persistent (>14 days) diarrhoea in children under the age of 2, and found the majority to be suffering from milk protein allergy (54%). Less common diagnoses included coeliac disease (17%) and lymphangiectasia (12%).[12] All patients presenting in areas where HIV is highly endemic must be screened for the disease, as diarrhoea incidence, severity, and mortality are significantly higher in children with HIV/AIDS.[4]

Acute diarrhoea – aetiology

The aetiology of acute diarrhoea is largely dependent on geographic and seasonal factors. The leading cause of severe dehydrating diarrhoea worldwide is rotavirus, responsible for an estimated 16–61% of all cases of AGE, 14–45% of diarrhoeal-associated hospitalizations, 20% of diarrhoeal-associated deaths and approximately 6% of all-cause deaths <5 years of age.[6,13–15] Primarily spread via faecal-oral transmission, rotavirus manifests with watery diarrhoea, vomiting, and low-grade fever, with a peak incidence in the rainy season.[1,11] Other notable viral agents include norovirus, sapovirus, adenovirus, and astrovirus, all of which are likely transmitted faecal-orally and have minor epidemiologic differences that are beyond the scope of this paper. One exception is norovirus, which often presents with worse vomiting than diarrhoea compared to rotavirus and is associated with AGE outbreaks and non-bacterial food-borne illness in developed countries.[11]

Important bacterial agents responsible for diarrhoea include *Escherichia coli*, *Shigella species*, *Campylobacter jejuni*, *Vibrio cholerae*, *Salmonella species*, and less commonly *Clostridium difficile*.[1,14,16] Of these, enterotoxigenic *E. coli* (ETEC) is the major cause of paediatric 'traveller's diarrhoea,' followed by *Shigella* and *Salmonella* species.[13] ETEC and *Salmonella* are typically self-limited and often result in a watery diarrhoea, while patients with *Shigella* are more likely to present with haemorrhagic stools. Entero-haemorrhagic/shiga-like toxin producing *E. coli* (EHEC), particularly O157:H7, is associated with haemolytic-uremic syndrome (HUS). While this pathogen is generally found in Europe and parts of North and South America, outbreaks have occurred in southern Africa necessitating awareness worldwide.[1] Local variations in healthcare infrastructure and climate result in variable pathogen prevalence, but some useful information can be gleaned from a large recent prospective cohort study from a United States paediatric emergency centre. Of 1626 patients enrolled, 47% had a pathogen identified. Bacterial cases accounted for 7%, viral 33%, parasitic 1%, and *C. difficile* toxin a surprising 7%.[17] Additional data are required to assess the clinical relevance of this *C. difficile* toxin prevalence but it lays a groundwork of expectations for practitioners in more developed nations.[18]

Protozoan infection is uncommon and should only be suspected if a patient's history directly implicates a parasite: a known contaminated water source, persistent haemorrhagic diarrhoea despite adequate *Shigella* treatment, or microscopic confirmation. Pathogens of note include *Entamoeba histolytica, Isospora belli*, *Giardia*, and *Cryptosporidium*. The latter two are the most common parasitic causes of diarrhoea in developed countries while *Entamoeba histolytica* is more common in developing nations.[14] *Cryptosporidium* is generally self-limited but of concern in immunocompromised or undernourished patients.[1,8] *Isospora belli*, while rare in developed areas, should be considered in severely immunocompromised patients in tropical settings.[19]

Practitioners should also consider non-infectious causes including inflammatory bowel disease, cystic fibrosis, appendicitis, or intussusception.[12,16,20]

History

A thorough history can aid in both diagnosis and disposition planning. Important aspects to discuss with guardians include (1) onset and duration of symptoms; (2) stool characteristics (bloody, watery, presence of mucous); (3) frequency and volume of stool; (4) oral intake of solid food and fluid throughout the period of illness, including breast milk; (5) presence or absence of associated symptoms including nausea, vomiting, abdominal pain, or headaches; (6) presence or absence of infectious signs or symptoms including fever, cough, or known recent infections; (7) symptoms of dehydration such as weight loss, decreased urination, decreased tear production, tachycardia, dry mouth, cool extremities, or orthostasis; and (8) any epidemiologic risk factors, such as recent travel, crowded living conditions or day-care use, raw/undercooked food consumption, animal contacts, sick contacts, recent medication use, underlying conditions such as HIV, and immunization history.[1,8,9]

In addition to aiding in identification of the diarrhoeal pathogen, historical elements can aid in risk-stratifying paediatric patients. A 2003 study by Porter et al. found a high sensitivity (73–100%) for parentally reported data to predict >5% dehydration, with a report of normal fluid intake, normal urine output, and normal tearing all independently reducing the likelihood of significant dehydration.[21]

Physical assessment

The primary goal of the physical exam is to assess the severity of fluid deficit. Secondary to this is identification of complicating conditions such as malnutrition, altered mental status, or co-existing infection. The WHO dehydration classification system (1995) uses a four-item assessment (general appearance, sunken eyes, degree of thirst, and skin turgor), and divides dehydration into three levels, *none, some, and severe*, graded to guide treatment. Mild/no dehydration is defined as a loss of baseline body weight of $< 5\%$, while moderate dehydration falls between 5–10% and severe $> 10\%$.[1] When a patient's baseline weight is unavailable, as it often will be, estimates should be made using ideal weight-for-age, physical exam findings and historical data.

Steiner et al. performed a systematic review of the accuracy and precision of physical exam and laboratory findings to evaluate dehydration in children.[20] Included in this review were only studies comparing weight at presentation to re-hydration weight as a gold standard for fluid deficit. The summary data demonstrated that physical exam findings were often imprecise and varied in their predictive value between examiners. Overall, the three most predictive signs were prolonged capillary refill time (likelihood ratio (LR), 4.1; 95% CI, 1.7–9.8), abnormal skin turgor (LR, 2.5; 95% CI, 1.5–4.2), and abnormal respiratory pattern (LR, 2.0; 95% CI, 1.5–2.7).

Rarely should a clinician use a single exam finding to determine treatment or disposition. Gorelick et al. prospectively evaluated a 10-point scale for predicting paediatric dehydration, defined as a deficit of $> 5\%$ body weight from baseline.[23] Exclusion criteria included malnutrition, prolonged illness, and electrolyte disturbances, limiting the applicability of findings. The signs tested were: decreased skin elasticity, capillary refill time > 2 seconds, general appearance, absence of tears, abnormal respirations, dry mucous membranes, sunken eyes, abnormal radial pulse, tachycardia > 150 bpm, and decreased urine output. While individual factors were non-diagnostic, the presence of 3 or more of these signs had a sensitivity of 87% and specificity of 82% (LR, 4.9; 95% CI, 3.3–7.2). Four selected signs (capillary refill time, dry mucous membranes, absence of tears, and abnormal overall appearance) were tested as subset. The presence of 2 or more of these was also predictive (LR, 6.1; 95% CI, 3.8–9.8).

In the setting of malnutrition, many exam findings for dehydration are confounded. Among these are skin elasticity, sunken eyes, and overall appearance. In these situations, focus should be placed on signs specific to hypovolemia: urine output, tear production, and subjective thirst.[1] Even more challenging is the overlap between shock and dehydration, which both manifest similarly. If a patient appears severely dehydrated but has no history of diarrhoea or meets sepsis criteria, treatment for septic shock should commence.

Role of imaging studies

Imaging has played a very small role in the diagnostic workup of diarrhoeal illness. Abdominal plain films are nonspecific and often show dilated loops of bowel in patients with diarrhoea, while abdominal CT scans rarely lead to changes in therapeutic approach.[16] A notable exception is the patient with an acute abdomen in whom CT imaging may clarify the need for surgical intervention. Examples include intestinal perforation as a complication of Salmonella typhi infection and acute appendicitis, which presents with associated diarrhoea in up to 41% of children less than 3 years old.[20,24]

Each year the body of literature on ultrasound (US) imaging becomes more robust, while access to bedside ultrasonography becomes available to a larger population. In addition to diagnosing intra-abdominal pathology that may be associated with diarrhoea such as appendicitis and intussusception, US has been used in the assessment of dehydration. Levine et al. examined a cohort of children in three rural Rwandan hospitals and found IVC/aorta ratio of 0.82 to be 93% sensitive and 59% specific, with LR+ of 2.3 (95% CI, 1.5–3.5) for detecting dehydration, defined as $\geqslant 10\%$ body weight loss at presentation.[25] IVC compressibility was not a reliable measure of dehydration in this study. A similar trial by Chen et al. performed in an urban US emergency centre found IVC/aorta ratio of 0.8 to be 86% sensitive and 56% specific for detecting dehydration $\geqslant 5\%$.[26]

Role of laboratory testing

Multiple studies have examined laboratory tests for the assessment of dehydration in children, including blood urea nitrogen (BUN), BUN/creatinine ratio, serum electrolytes, and the presence of acidosis (using bicarbonate levels or base deficit measurement).[9,22] Of these, BUN and serum bicarbonate have both been associated with clinically significant dehydration, although evidence is mixed. Depending on cut-off values, elevated BUN levels demonstrate a sensitivity between 38–44% and specificity of 55–99% for $\geqslant 5\%$ dehydration.[22] Vega and Avner found that serum bicarbonate < 17 mEq/L was 94% sensitive for $> 10\%$ dehydration (95% CI, 71–100%).[27] Nagler et al. showed that end-tidal carbon dioxide levels correlate well with serum bicarbonate values in children with AGE, potentially allowing for an accurate, non-invasive proxy measure of dehydration.[28]

Electrolyte measurement is appropriate in patients with altered mental status, moderate/severe dehydration, clinical signs of electrolyte disorders (weakness, cramping, seizures), or in children < 6 months.[9] While resource limitations may not allow for laboratory testing in all situations, Wathen et al. showed that serum electrolyte measurement changed the management in a full 10.4% of 182 patients receiving IV fluid for dehydration in a US paediatric emergency centre, and should at least be considered in all moderate to severe cases.[29] Blood glucose testing of hypoglycaemia should be performed whenever available, as diarrhoea places a child at increased risk and is often clinically undetectable. A study in the US of children with AGE and normal mental status found 9.2% to be hypoglycaemic with a range from 34 to 59 mg/dL.[30]

Stool cultures are rarely indicated in acute diarrhoea. The majority of diarrhoea in developed countries is viral and therefore culture yield is extremely low. While developing countries have higher incidence of bacterial-associated diarrhoea, results rarely impact management.

Acute medical management

The cornerstone of paediatric diarrhoea management is to ensure proper hydration, whether through oral rehydration

therapy (ORT) or intravenous therapy (IVT), and early re-introduction of feeding when appropriate.[2,6] The decision between ORT and IVT depends on the degree of dehydration and the clinical presentation. The WHO recommends that all patients with severe diarrhoea (>10% dehydration) to be treated with rapid IVT while those with mild/moderate diarrhoea be given a trial of ORT.[1] However, all children who are able to drink should be given immediate oral rehydration solution (ORS) regardless of the overall treatment plan. Contraindications to ORT include altered mental status, inability to protect the airway, and suspected ileus; vomiting is *not* a contraindication.[1,9,14] A Cochrane review comparing the two treatment modalities in children with AGE showed no significant differences in weight gain, sodium imbalance, duration of diarrhoea, or total fluid intake at 6 and 24 h between the two groups.[31] Only one in 25 children (95% CI, 14–100) treated with ORT required IVT due to inadequate rehydration.

Reduced osmolarity ORS (245 mOsm/L) has resulted in fewer unanticipated IV fluid infusions, lower stool volume, and less vomiting in hospitalized patients with acute diarrhoea, when compared to the WHO standard formulation (310 mOsm/L).[32,33] Additionally, polymer-based ORS may have some additional advantages although further study is still needed.[34] For patients with *some* dehydration, ORS should be initiated at a rate of 75 ml/kg over the first 4 h.[1,14,35] This rate should be a rough guide for practitioners, with frequent re-assessments and alterations in treatment strategy based on clinical indicators; for example very frequent watery stools may require an increase in total ORS volume. Children who remain thirsty with no signs of over-hydration should be allowed to continue taking ORS. Nasogastric (NG) tube hydration should be considered in children who are not tolerating oral fluids, and as a bridge therapy in children for whom intravenous or intraosseous (IO) access is unavailable. NG hydration is equally as effective as IVT in moderately dehydrated children.[36] Benefits of ORT include natural barriers against over-hydration (thirst decreases with fluid deficit), non-invasive and rapid initiation of treatment, cost-savings over IVT, and less stress for child and guardian.

For the severely dehydrated, obtunded, or hemodynamically unstable patient, IVT should be first-line therapy. In patients with challenging IV access, IO or NG hydration should be started immediately, and all children who can safely drink should take additional ORS. The WHO recommends that infants (< 12 months) receive 30 ml/kg IVT over 1 h followed by 70 ml/kg over 5 h. Older children should receive 30 ml/kg over 30 minutes and 70 ml/kg over the next 2.5 h, with frequent re-assessment.[1] King et al. recommend an initial rate of 20 ml/kg until mental status and vital signs improve, followed by an infusion of twice standard maintenance rates.[35] Normal saline or lactated ringers may be given as an initial bolus, followed by 5% dextrose ½ normal saline for maintenance therapy.[14,35] To replace on-going losses, 20 mEq/L of potassium chloride may be added to maintain fluids.

Both hypo- and hypernatremia may complicate the course of diarrhoeal illness and should prompt hospitalization. Hyponatraemia may be safely and slowly corrected with ORS in most cases, but an important exception is the malnourished/oedematous child for whom ORS provides excessive sodium and requires dilution.[1] Hypernatremia is often secondary to inadequate fluid repletion or repletion with hypertonic fluids. In these children, slow correction of the electrolyte disturbance is crucial, as rapid over-correction may lead to cerebral oedema, seizures, or death. IV fluid repletion places the child at higher risk of over-correction compared to ORT and must be monitored closely, with any changes in mental status prompting immediate re-assessment.

Low-dose zinc supplementation (10–20 mg/day for 10–14 days) is a standard recommendation by the WHO, although in recent years the specific role of zinc has been called into question.[1,7,37] Despite strong evidence from multiple studies that zinc decreases severity and duration of illness, two systematic reviews by Patel et al. highlight heterogeneity of responses to zinc in both acute treatment and preventative therapy.[5,37,38] Also, many studies of diarrhoeal illness are conducted in developing nations where zinc deficiency is more common, obscuring the role of zinc in developed settings.[6] However, while the questions regarding the appropriate target population are being answered through further study, zinc should remain a component of acute diarrhoeal treatment. A systematic review by Walker and Black showed a 23% reduction in mortality and decreased risk of hospitalization in zinc-treated patients with diarrhoea.[33,39]

Recommendations for antibiotics in acute diarrhoea vary widely, largely due to different practice environments and overall goals of care. The WHO, working chiefly to reduce paediatric mortality in developing countries, recommends avoiding routine administration of antibiotics in almost all cases of acute diarrhoea except for notable disease states: severe or epidemic *V. cholerae* infections (treatment should be guided by local antibiogram patterns), laboratory-proven *Giardia* (treated with metronidazole), or cases of haemorrhagic diarrhoea considered possible *Shigella* infections (treated with a fluoroquinolone).[1,8] Holtz et al. discourage the administration of antibiotics to patients with acute haemorrhagic diarrhoea until a clear treatment-responsive pathogen is identified, and recommend hospital admission to allow time for stool cultures to grow and to prevent community spread of bacterial illness.[16] This recommendation is specific for regions with low incidence of shigellosis, as *Shigella* is life-threatening and suspected cases should be treated. The Infectious Disease Society of America further cautions against over-treatment, given worldwide increase in quinolone-resistant *Campylobacter* infections and multi-drug resistant *Salmonella* strains. Antibiotics should be avoided in suspected cases of EHEC, as antimicrobial therapy has not been shown to improve symptoms of O157 infections but is associated with increased risk of HUS.[8] Antibiotic-induced diarrhoea is common, especially in countries where antibiotics are widely available without prescription. In these cases, discontinuation of the antimicrobial agent may be sufficient for treatment. In severe cases, testing for *C. difficile* toxin should be considered.[40]

Adjunct medical interventions have been examined, particularly in developed countries. These ''anti-diarrhoeal'' drugs are contraindicated by the WHO for use in any situation, as no practical improvement in outcomes has yet been associated with these agents. The following medications, if used at all, should only be prescribed with caution by experienced practitioners: Racecadotril (acetorphan), which reduces intestinal secretions, has been shown to decrease stool duration, output, and number in a meta-analysis of children with AGE.[6,11,41]

Diosmectite, an intestinal adsorbent, has been shown to decrease diarrhoeal duration in a meta-analysis with a pooled weighted mean difference of −22.7 h.[6,42] Bismuth subsalicylate has been shown to safely decrease the duration and symptoms of watery diarrhoea, particularly when due to rotavirus.[11,43,44] Probiotics have been shown to be highly effective in limiting viral diarrhoea duration, but efficacy is limited to two strains: *Lactobacillus rhamnosus* GG and *Saccharomyces boulardii*.[45] Other adsorbents and antimotility agents, including loperamide, codeine, diphenoxylate, kaolin, and activated charcoal have limited data or safety concerns and should not be used.[11,43,44]

Disposition planning

Admission to the hospital should be considered for children with severe dehydration or moderate dehydration and inability to take oral fluids, those with serious co-morbidities, haemorrhagic diarrhoea, electrolyte disturbances, or for any child with a poor social situation and low likelihood of proper home therapy. If a child is taking proper oral hydration, appears to be improving in the emergency centre, and has no underlying medical conditions, discharge to home is likely a safe choice. Borderline cases should be referred to paediatric observation units (OU). AGE and dehydration have been specifically examined in these units, with one study of 430 OU admissions resulting in an 81% discharge rate within 24 h.[46] *C. difficile* cases should be isolated, but any admitted diarrhoea case should prompt strict practices to prevent nosocomial infection.

Clear and comprehensive discharge instructions are instrumental to ensure optimal treatment, prevent prolonged courses of diarrhoea, and reduce the number of children who fail outpatient management. A survey of parents in the US showed that only 53% were treated for diarrhoea with recommended fluids, while some parents giving fluids high in simple sugars and some giving inappropriate medical treatments.[47] Guardians should be taught to give more fluid than usual. Mothers should be strongly encouraged to continue breastfeeding, as it has been proven protective against diarrhoeal illness.[1] Other appropriate fluids include ORS solution, salty (3 g/l) drinks, soup, or broth, plain clean water, or unsweetened juice.[1] Sweetened drinks should be avoided due to the potential for osmotic diarrhoea and hypernatremia.

Early re-feeding has been shown to be appropriate and beneficial for children with acute diarrhoea, with no increased risk of vomiting, IV fluid requirements, or persistent diarrhoea.[48] Food should be nutritive, and frequent small feeds are better than large meals. The "BRAT" diet (bananas, rice, applesauce, toast) is not associated with any convincing evidence and is low in energy density, protein, and fat. It should not be recommended to parents.[13]

Basic hygiene techniques such as proper water filtration and sourcing should be discussed as is appropriate, given that 88% of deaths due to diarrhoea may be attributable to unsanitary drinking water, inadequate sanitation, and poor hygiene.[3] Medical staff and parents should be strongly encouraged to wash hands often with soap and water to prevent disease transmission, and children of appropriate age should be taught the same.

Finally, the WHO has recommended that rotavirus vaccination should be implemented as a part of all national immunization programs to reduce diarrhoeal mortality.[33] These vaccines reduce diarrhoeal incidence, hospitalization due to diarrhoea, and overall child mortality.[49] Although specific formulations are beyond the scope of this paper, newly developed rotavirus vaccines have been shown to be safe and effective, and should be discussed with the child's guardian as part of prevention planning.[50]

Conclusions

As the WHO stated in their 2009 report on child mortality, "although progress is being made, much more remains to be done." Physicians operating in emergency settings will be frequently faced with the task of treating children with acute diarrhoea and should be prepared to make evidence-based diagnostic and treatment decisions. Using a combination of historical clues, physical exam findings, ultrasound imaging (when available), and laboratory testing, the degree of dehydration can be accurately and rapidly identified. Once risk-stratified, patients should be treated accordingly, with an emphasis on correcting fluid deficit and supplying nutrients/electrolytes. The role of quality discharge planning cannot be understated, as appropriate anticipatory guidance has the potential to decrease the frequency and severity of future episodes.

Appendix A. Short answer questions

1. Which of the following infectious agents is responsible for the greatest majority of morbidity and mortality secondary to diarrhoea worldwide?
   a. Norovirus
   b. *Escherichia coli* O157:H7
   c. Rotavirus
   d. *Campylobacter jejuni*
   e. *Vibrio cholerae*

2. A 20 kg child presents to your emergency centre with 3 days of watery diarrhoea but no vomiting. History is positive for decreased tearing and an estimated 3 kg weight loss. Exam is notable for decreased capillary refill and dry mucous membranes. What fluid repletion strategy is most appropriate?
   a. Management at home, recommend 200 ml intake per watery stool
   b. Oral hydration in the centre, starting with 1.5 l over 4 h
   c. Rapid IVF only, bolus of 400–600 ml followed by twice maintenance rate
   d. Trial of oral hydration while preparing for IVF as above, consider observation unit admission
   e. Immediate admission and inpatient ward management

3. Given the supporting evidence presented in this article, which of the following is not currently recommended for the treatment of acute diarrhoea?
   a. Activated charcoal
   b. Bismuth subsalicylate
   c. Diosmectite
   d. Probiotics
   e. Racecadotril

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