positive effect on opportunistic microsporidiosis and cryptosporidiosis [10]. If these observations are confirmed in a larger patient cohort, the combination of HAART and appropriate antimicrobial treatment might be able to resolve such opportunistic infections, independent of the CD4+ lymphocyte cell count.

Paolo Maggi,1 Angela Maria Vittoria Larocca,2 Nicoletta Ladisa,3 Sergio Carbonara,1 Olga Brandonisio,2 Gioacchino Angarano,1 and Giuseppe Pastore3

Institutes of Infectious Diseases,1 Hygiene, and3 Microbiology, University of Bari, Bari, Italy

References


Desmopressin Treatment for a Case of Dengue Hemorrhagic Fever/ Dengue Shock Syndrome

Sir—Dengue fever presents as a benign viral illness or may progress to dengue hemorrhagic fever/dengue shock syndrome (DHF/DDSS). Since 1988 we have endured 4 epidemics of dengue in French Polynesia that have mainly affected children—the so-called “Southeast Asian” epidemiological pattern. Ten children who were admitted to critical care units for treatment of uncontrolled DHF/DDSS died during the 1989–1990 outbreak. Since then, we have had no more deaths to deplore.

In cases of DHF/DDS, thrombocytopenia is a major concern because of the risk of hemorrhage. Another risk is hypovolemic shock due to increased vascular permeability and massive plasma leakage requiring intensive support [1, 2]. Hypovolemic shock develops in 24 h approximately 5–8 days after the onset of fever; it lasts up to 3 days, a duration that is predictable [3]. If the vital functions of the patient can be sustained during this period and there are no complications, the patient will recover.

For all of our patients, we had to infuse large amounts of fluid for hemodynamic assistance and to maintain diuresis. The infused fluids leaked from the vascular beds and induced generalized edema, causing weight gain that was up to 25%–30% greater than initial body weight; the infused fluids also produced major serious effusions and impaired respiratory function. Therefore, our aim was to restore a functional vascular barrier to avoid these complications and to reduce the risk of thrombocytopenia.

A 7-year-old boy with grade III DHF/DDSS was admitted to the hospital on the fourth day after the onset of symptoms. He was afebrile and had abdominal pain, oral intolerance, and pleural effusion. Laboratory data were as follows: pulse, 130–140 beats/min; blood pressure, 110/90 mm Hg; platelet count, 21 × 10^9 cells/L, with purpura; hematocrit, 52%; albumin, 31 g/L; sodium, 126 mEq/L; and urea, 11.2 mM/L. In addition to administering initial infusions (50 mL/kg over 2 h), we introduced desmopressin (1-deamino-8-D-arginine vasopressin, or DDAVP; 0.3 μg/kg over 30 min). Three hours later, the pulse rate slowed to 80–90 beats/min and the blood pressure differential increased to 100/50 mm Hg and remained normal thereafter. Desmopressin was administered for 3 days (once daily at the same dosage: 0.3 μg/kg), and we monitored the level of sodium and the fluid balance. Daily fluid and caloric maintenance was done with iso-osmotic infusions. Diuresis increased from 500 mL on the fourth day of symptoms (the day of admission) to 1600 mL on the fifth day and 3000 mL on the sixth day. On the eighth day of symptoms, the platelet count was 90 × 10^9 cells/L, the pleural effusion had decreased, and there was no bleeding.

Since we treated this child, 3 more children with DHF/DDSS have been successfully treated in the same manner. One of the children had a digestive tract hemorrhage that stopped after infusion of desmopressin.

It has been proposed that blood products should be infused for bleeding associated with DHF/DDSS, but little attention has been paid to alternative treatments or prevention. Because of its ability to prevent hemorrhages and allow surgical procedures, desmopressin is used to treat some hereditary bleeding disorders, and it also may be used to treat acquired impaired hemostatic conditions [4, 5]. Desmopressin shortens prolonged bleeding times, releases endothelial hemostatic factors, and, in vitro, promotes the adhesion of platelets to the vascular sub-
endothelium. It can be administered for up to 3 or 4 days, after which time its hemostatic benefits decrease [5]. Side effects are rare in children, for whom the drug has been widely used as long-term treatment for enuresis. Desmopressin was controversially reported to worsen thrombocytopenia in patients with von Willebrand disease type 2B [5, 6]. If levels of fluid and salts are not monitored, desmopressin can induce water intoxication [7].

In dengue with shock, a capillary leak induces plasma drift that is primarily confined to the peritoneum and the pleura. This suggests an initially localized endothelial lesion. Attempts to treat such vascular leakage with carbazochrome sodium sulfonate have produced conflicting results [8, 9]. For our patient, the need for fluid resuscitation was limited to initial infusion and then daily fluid maintenance; we maintained surveillance of diuresis, the patient’s weight, and the levels of salts and fluid. There was prompt clinical improvement, and the need for fluid resuscitation was reduced 10-fold compared with our previous observations. Induced edemas were avoided. We suggest that desmopressin may help reverse the vascular functional deficiency that leads to shock.

We have too few patients to plan a controlled trial of desmopressin in patients with DHF/DSS. However, we recommend that such a controlled trial be undertaken by our colleagues who treat a larger number of patients in a monitored clinical environment.

Addendum. As of 12 September 2001, our unit has admitted 19 patients with grade III or grade IV DHF/DSS. Three of these 19 died. Of the 16 patients who received 1–3 doses of desmopressin, 1 died within 24 hours of admission, and 15 responded favorably.

Laurent Pea,1 Laurent Roda,2 and Fabrice Moll1
1Critical Care Unit and 2Clinical Biology Unit, Centre Hospitalier Territorial, Papeete, Tahiti

References

Aspiration Pneumonia: A Misnomer

SIR—In spite of the fact that the term “aspiration pneumonia” is embedded in the medical parlance used during ward rounds, in the morning report, and in recent review articles [1], its use should be discouraged. Aspiration is a pathogenic mechanism for several inflammatory diseases of the lung, both infectious and non-infectious. Avoiding use of the term “aspiration pneumonia” in the usual sense enables us to distinguish clearly between well-defined pulmonary syndromes, such as chemical pneumonitis, anaerobic pneumonitis, and classical bacterial pneumonitis, that are mediated by aspiration of different noxious mixtures.

Mendelson’s syndrome [2] refers to the inflammatory pulmonary disorder caused by aspiration of gastric content, more aptly called chemical pneumonitis. It produces fever, leukocytosis, purulent sputum, and an infiltrate visible on a radiograph. It is not an infectious but an inflammatory process, because the low gastric pH keeps the gastric contents sterile. If the clinician is sure that the patient vomited and then aspirated, they can follow the patient’s course carefully without administering antibiotics. All these inflammatory changes will start to clear in 24–36 h. There should be a low threshold to determine whether to start administration of antibiotics, because the sloughed pulmonary tissue is primed to become infected. Gastric aspiration can produce an infectious pneumonitis if the gastric pH is increased by antacids, histamine-receptor antagonists, proton-pump inhibitors, or enteral feeding [3–4] or if there is gastroparesis or obstruction of the small bowel.

The disorder that most clinicians associate with the term “aspiration pneumonitis” is better defined by the term “anaerobic pneumonitis” [5]. Normal oral flora is composed of a large variety of microaerophilic and anaerobic organisms of low virulence. For such low-virulence organisms to establish a “beachhead” in the lung parenchyma, they have to be aspirated in rather large volumes. This can occur in aspiration-prone patients, such as those who have frequent episodes of loss of consciousness (e.g., patients prone to seizures or who have alcoholism) or have disorders of the swallowing mechanisms (e.g., patients who have had cerebrovascular accidents). The classic bacterial types of pneumonia are caused by aspiration of small amounts of highly virulent organisms, such as Streptococcus pneumoniae. Use of the term “aspiration pneumonia” to refer to anaerobic pneumonitis blurs the distinction between these different entities, depriving the cli-