Enteric adenoviruses (updated report, 2010)

In previous newletters (4 and 5), we have presented the "full story" of adenoviruses. We won't come back on general aspects of adenoviruses but we will focus here on the clinical data of the so-called "enteric adenoviruses" (EAd), responsible for gastrointestinal disease in children. These are adenoviruses of the subgroup F, the well-known types 40 and 41. The other adenoviruses are generally refered as the non-enteric adenoviruses (NEAd).

Although many adenovirus types are found in stool from patients with diarrhea, only EAds have been shown to be the causative agents of gastrointestinal disease. A close comparison of stools from diarrheal and control patients has shown that whereas NEAd particles are shed in stools from both diarrheal and control patients, EAd particles are found almost only in diarhheal stools. As shown by several studies, up to 50% of healty children carry NEAd whereas EAd is rarely present in healthy children. Moreover, EAd carriage is 8 times higher than NEAd carriage in diarrheal stool samples. Moreover, as NEAd viral particle secretions may perists for months (Ad3 is reported to be excreted intermittently for up to 906 days), NEAd presence in stools along with other viruses is common. In these cases, the other viruses (mainly rotavirus) were the causative agents of the observed diarrhea.

The EAd cause a variety of diseases and are prevalent throughout the world. Even in Africa when the EAd was relatively unknown entity in gastroenteritis epidemiology, the recent findings suggest that EAd is probably under-reported due to low or lack of specificity of commercially availables EIA for all circulating EAd strains.

Proportion of EAd from all adenovirus-positive stools is reported to be from 25 to 95%. It is not surprising, since EAd grow poorly in many cell culture (EAd were previously reported as non-cultivable), and as some commercial adenovirus 40/41-specific ELISA as well as electron microscopy observation show poor sensitivity, the EAd prevalence has been initially underestimated.

A survey on more than 4600 children showed that EAd is the second cause of children diarrhea, after the rotavirus. Whereas the rotavirus has its maximal prevalence in the 2nd half of the first year of life, EAd is more prevalent in the firsts 6 months of age, being almost as prevalent as rotavirus in the firsts weeks of age. A case of EAd in an 75 years old adult was reported.

A recent study in UK suggest a great role for 40/41 viruses in community-acquired (CA) acute gastroenteritis (AGE) than has been

previously appreciated and indicate it also play a prominent role in healthcare-associated (HA) AGE.

EAd infections are observed throughout the year, but peaks are often found in autumn and early winter. Many cases of hospital-acquired EAd infections have been reported, sometimes responsible for 50% of observed infections over several years.

Clinical symptoms are somewhat different from what is observed in rotavirus infected children: there is less vomiting and fever, but diarrhea is protracted, with a mean duration of 8 to 12 days and virus excretion for 10 to 14 days. Allmost all EAd-infected young children (< 2 years) have diarrhea, 50% of which are watery. Respiratory problems are infrequent in EAd infections.

In conclusion, among all adenoviruses found in stools from children having gastrointestinal diseases, only the EAd (40/41) are the causative agents of these diseases, NEAd being not diarrhea-specific adenoviruses. EAd are mainly found in very young children. Children under 2 years having diarrhea should be tested for both rotavirus and EAd (40/41).

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