



ACUTE HEMORRHAGIC CYSTITIS DISEASE IN CHILDREN AND ITS DEVELOPMENT IN THE CHILD'S BODY

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Annotation

Hemorrhagic cystitis is defined as a diffuse inflammatory condition of the urinary bladder due to an infectious or noninfectious etiology resulting in bleeding from the bladder mucosa. The most common cause is bacterial infection that usually responds promptly to treatment. But chronic and recurrent hemorrhagic cystitis often arises from anticancer chemotherapy or radiotherapy for the treatment of pelvic malignancies.

Keywords: acute hemorrhagic cystitis, infection, recurrent, adenovirus, E. coli.

Acute Hemorrhagic Cystitis Acute hemorrhagic cystitis (AHC) is usually a benign, selflimited disorder in healthy children. Adenovirus is the most common cause of viral AHC in children, with E. Coli occasionally involved. In a series of 69 infants and children with AHC, adenovirus 11 was recovered from the urine in 10 (14.5%), adenovirus 21 in 2 (2.9%), and E. coli in 12 (17.4%). However, the remainder (>60%) had no known infectious agents isolated from the urine. Conversely, children presenting with acute adenovirus respiratory infections may develop incidental microscopic or gross hematuria. In healthy children, no antiviral therapy is indicated, because the infection is self-limited. However, radiologic evaluation should be considered to rule out other causes of hematuria. More recently, AHC has been observed with increasing frequency in children undergoing bone marrow transplantation (BMT). AHC can be divided into pre-engraftment and post-engraftment subtypes, each with unique etiologies. The pre-engraftment AHC is a



result of agents known to be urinary tract toxins, such as cyclophosphamide used to condition the recipient before BMT. Hydration and use of 2-mercaptoethan sulfonate (MESNA) may prevent this type of AHC. The post-engraftment subtype is more common, occurs 2 weeks to 4 months after BMT, and can be life-threatening. BK virus, a ubiquitous DNA virus of the polyomavirus genus, has increasingly been implicated as the causative agent for AHC in this population. Whereas asymptomatic BK virus infection is present in more than 50% of children younger than 10 years of age, the BMT host allows the BK virus to replicate unchecked in the urothelium. After engraftment, the host immune response reacts to the virus in the urothelium, resulting in mucosal injury and hemorrhage. Preliminary studies have shown that fluoroquinolones decrease the urinary concentrations of the BK virus when used in a prophylactic fashion, via inhibition of DNA gyrase. However, larger studies demonstrating prevention of AHC are needed. Treatment of AHC in these children is primarily supportive, because no standardized drug regimen has been developed. Mild cases may be treated with aggressive hydration. More severe cases may require continuous bladder irrigation and fulguration. Immunocompromised children are also at risk for adenovirus-associated hemorrhagic cystitis, and intravenous ribavirin has anecdotally been used to treat such an infection. Cidofovir, a purine analogue, was used in series of BMT patients with some success, but use is limited by nephrotoxicity of the agent.

All in all, HC is a common devastating life-threatening complication of bone marrow transplantation and cyclophosphamide-based chemotherapy. Thus, treatment with cyclophosphamide and bone marrow transplantation mandate preventive therapy but physicians should be aware of the fact that despite measures to prevent it, HC might be inevitable. Development of HC mandates immediate reaction and cooperation between the medical services. Bladder perforation is a rare but life-threatening complication, patients with severe HC, especially with recurrent clot retention, and uninhibited bladder contraction and those under continuous bladder irrigation should be monitored meticulously.

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