per 100 child-years before the pre-cART era to 0.08 per 100 child-years during the cART era (2001–2004).[1] However, *Candida* esophagitis continues to be seen in children who are not responding to antiretroviral therapy (ART).[2,3] Children who develop esophageal candidiasis despite ART may be less likely to have typical symptoms (e.g., odynophagia, retrosternal pain) or have concomitant OPC;[4] during the pre-cART era, concomitant OPC occurred in 94% of children with *Candida* esophagitis.[2] Risk factors for esophageal candidiasis include low CD4 T lymphocyte (CD4) cell count (<100 cells/mm^3), high viral load (>5,000 copies/mL), and neutropenia (absolute neutrophil count [ANC] <500 cells/mm^3).[1-3,5]

Invasive Candidiasis

Invasive candidiasis is less frequent than localized disease in children with HIV infection. However, *Candida* can disseminate from the esophagus, particularly during co-infection with herpes simplex virus (HSV) or cytomegalovirus (CMV).[2,6] Candidemia occurs in up to 12% of children with HIV infection who have chronic indwelling central venous catheters placed for administration of total parenteral nutrition or intravenous (IV) antibiotics.[3,7] While *Candida albicans* remains the most common cause of all candidiasis, approximately 50% of reported cases of *Candida* bloodstream infections in children are caused by non-*albicans Candida* spp. including: *Candida tropicalis*, *Candida kefyr* (*Candida pseudotropicalis*), *Candida parapsilosis*, *Candida glabrata*, *Candida krusei*, and *Candida dubliniensis*. In some settings, non-albicans species cause the majority of blood stream infections. The non-*albicans Candida* species are important to identify because several are resistant to antifungals. In general, *C. krusei* is considered resistant to fluconazole, and *C. glabrata* isolates have an increased rate of resistance to both fluconazole and voriconazole. Recently, an increasing number of *C. glabrata* isolates are also resistant to echinocandins. *C. lusitaniae* is inherently resistant to amphotericin B. Many children who develop candidemia have previously received systemically absorbed oral antifungal azole compounds (e.g., ketoconazole, fluconazole) for control of oral and esophageal candidiasis, which may predispose to resistant isolates.[3] In one study of Cambodian children with HIV infection and on ART who had candidiasis, seven (75%) of nine isolated *C. glabrata* were resistant to fluconazole, and three (40%) of seven *C. parapsilosis* isolated were resistant to >3 azole agents.[8] However, clinicians should be aware of local resistance trends as the epidemiology of species-specific resistance may vary widely by geographic location and hospital.

Clinical Manifestations

Clinical manifestations of OPC vary and include pseudomembranous (thrush), erythematous (atrophic), hyperplastic (hypertrophic), and angular cheilitis presentations. Thrush appears as creamy white, curd-like patches with inflamed underlying mucosa that is exposed after removal of the exudate and can be found on the oropharyngeal mucosa, palate, and tonsils. Erythematous OPC is characterized by flat erythematous lesions on the mucosal surface. Hyperplastic candidiasis presents as raised white plaques on the lower surface of the tongue, palate and buccal mucosa, and cannot be removed. Angular cheilitis presents as red fissured lesions in the corners of the mouth.

Esophageal candidiasis often presents with odynophagia, dysphagia, or retrosternal pain, and children, unlike adults, often experience nausea and vomiting. Therefore, children with esophageal candidiasis may present with dehydration and weight loss. Classic symptoms and signs of OPC may be absent in children with esophageal candidiasis, particularly those receiving ART.

New-onset fever in a child with HIV infection who has advanced disease, a central venous catheter, or both is the most common clinical manifestation of candidemia. Unfortunately, there are limited clinical signs or symptoms to denote dissemination to a particular organ, and detection of end organ involvement is often dependent on radiographic imaging. For example, renal candidiasis can present with candiduria, but ultrasonographic demonstration of renal parenchymal lesions is often not associated with symptoms related to renal disease.[3]