**Candida rugosa: a new fungal pathogen emerging, but from where?**

From the perspective of the clinical mycologist, it is, perhaps, easy to become sceptical about the fashion to place emphasis on the so-called ‘emerging infectious diseases’. Aspergillosis and candidosis continue to be the most important serious fungal infections. Aspergillosis is almost invariably caused by *Aspergillus fumigatus* and candidosis continues to be caused mainly by *Candida albicans* with a minority of other species, mainly *Candida glabrata* and *Candida parapsilosis*, and a smattering of other species, such as *Candida krusei*, *Candida lusitaniae* and *Candida guilliermondii*.

But this of course is a very Eurocentric view. The article by Singh et al. (2011) in this issue of the *Journal of Medical Microbiology* gives a stark reminder that in the world outside Europe and the US, the epidemiology and aetiology of invasive fungal disease are both different and quite possibly more rapidly evolving. Singh et al. (2011) have studied episodes of candidaemia at a level I trauma centre in New Delhi, India. At 27.6 cases per 1000 admissions, the observed prevalence was in the middle of the range observed in different countries for candidaemia generally, though higher than that specifically seen in trauma patients in a recent US study.

One of the most intriguing and perhaps worrying results presented is the fact that in over one-fifth of the cases (19 of 89 patients), the yeast involved was identified as *Candida rugosa*. Indeed, *C. albicans* was seen in only 16.8% of cases and relegated into fourth place behind *Candida tropicalis, C. parapsilosis* and *C. rugosa*. Also curious was the relatively low level of *C. glabrata* at 5.6%. The predominance of *C. tropicalis* in India is a relatively well-described phenomenon (Xess et al., 2007) but why the fuss about *C. rugosa*? Sixteen per cent of all *C. rugosa* isolates were resistant to fluconazole compared to 12.5% of *C. glabrata* isolates and fewer than 10% of *C. tropicalis* and *C. parapsilosis* isolates. In this study, all isolates including *C. rugosa* were susceptible to voriconazole, amphotericin and flucytosine. However, this level of resistance may in fact be relatively low. In a global survey of azole susceptibility, Pfaffer et al. (2010) found that 40% of *C. rugosa* isolates were resistant to fluconazole and 20% also resistant to voriconazole.

*C. rugosa* has been described as the cause of infection in various patient groups, usually in case reports of single cases but also occasionally in outbreak situations. Risk factors typical for systemic *Candida* infections include central venous catheters, surgical interventions and use of broad-spectrum antibiotics (Mincés et al., 2009). However, in the league tables of frequency of isolation, this organism does not rank highly. Taking a look at data from my own institution (a 3000-bed teaching hospital in the UK), out of 335 cases of candidaemia between 2005 and 2009, I can find one case caused by *C. rugosa*. In this context, the high level of infection described by Singh et al. (2011) begs the question – what is happening? This species has not been previously seen at this institution. These authors have previously published a brief report specifically on the *C. rugosa* isolates suggesting that all of the isolates from this series of cases of candidaemia are in fact closely related as determined by random amplified polymorphic DNA (RAPD) analysis (Behera et al., 2010). RAPD is of course notoriously unreliable for strain typing. Furthermore, as the authors themselves admit, they only included two *C. rugosa* isolates from patients who were not in this candidaemia series, making it difficult to adequately assess the strain diversity of this species (though those two isolates were clearly different). However, this finding does suggest a hypothesis of a single source.

The authors are understandably guarded about speculating on a source of the *C. rugosa* but part of the joy for the editorial writer is the freedom to do just that! *C. rugosa* has been described in association with, or as the causal agent of, mastitis in cattle and is one of the more common yeasts isolated from milk from mastitic cows (Crawshaw et al., 2005). *C. rugosa* is also used in the biotechnology industry, with its lipases used for a wide variety of applications including the production of ice cream and single cell protein (Benjamin & Pandey, 1998). Thus, it is tempting to think that the source for this may be some form of contaminated milk or food product. This hypothesis, of course, presents a relatively simple test in terms of looking at intestinal tract colonization – the type strain of this species was originally isolated from human faeces. Indeed, although there is speculation about the causes of the increased incidence of *C. tropicalis* infection in India – in this study 39% of cases and the most common species causing candidaemia – and the role of fungal virulence factors and host defence defects (Kothavade et al., 2010), the source of acquisition is rarely discussed. There is good evidence that *C. tropicalis* invades systemically largely from the gastrointestinal tract (Walsh & Merz, 1986). Again, is the high level of *C. tropicalis* candidaemia seen in India due to high gastrointestinal tract colonization and does this relate to a dietary source of this yeast?

As with all good science, the report of Singh et al. (2011) raises as many questions as it answers. The medical mycology community will have to work hard to keep up with the evolution of the aetiology and epidemiology of fungal infections around the world.

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DOI 10.1099/jmm.0.029199-0 © 2011 SGM Printed in Great Britain
a possible emerging cause of candidaemia in trauma patients. Infection 38, 387–393.


