CLARITY

Clinical implications of azole-resistant Aspergillosis in hematological malignancy

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Background

Survival of patients with invasive Aspergillosis (IA) has improved in recent years mainly due to the availability of azole antifungal drugs. Those advances are jeopardized by the emergence of azole resistance in *Aspergillus fumigatus*, the main causative pathogen for IA. Resistance mechanisms, with differing degrees of cross-resistance, are mainly characterized by point mutations in the *cyp51* gene encoding the target for azoles.[1]
Despite several studies suggesting a high probability of azole treatment failure in patients with azole-resistant isolates, it remains unclear what the clinical implications are of azole-resistant IA compared to azole-susceptible IA.

Objective

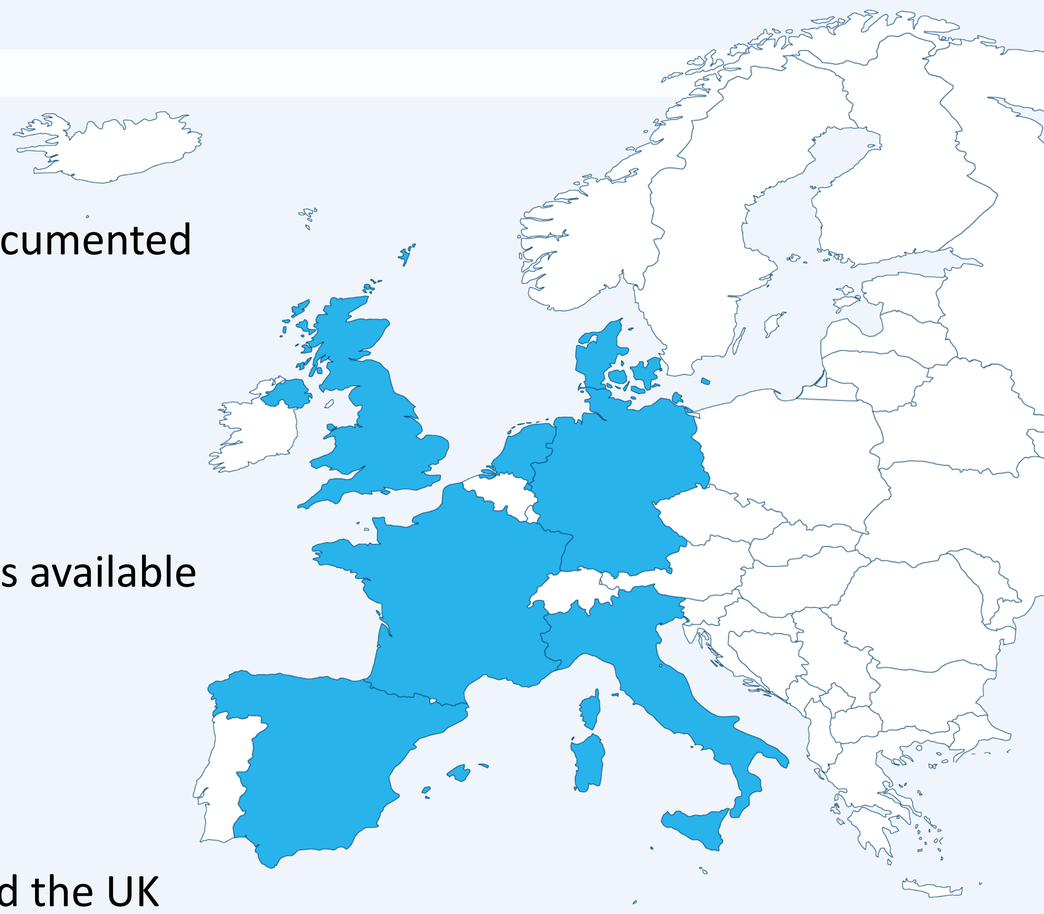
To determine the efficacy of antifungal therapy in patients with documented azole-resistant IA

Inclusion criteria

Adult and pediatric patients from 2016 and onwards
Patients with a hematological malignancy
Aspergillus fumigatus culture from clinical material of the patient is available
Evidence of proven or probable IA[2]

Study cohort

55 azole resistant IA cases
≥130 azole susceptible IA cases
From Denmark, France, Germany, Italy, the Netherlands, Spain, and the UK



Workflow

Sites routinely identify patients with IA

If **culture** is available and
IA is **proven or probable**[2]

Fungal isolate

Screen for azole resistance using **VIPcheck™ screening agar plates**

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Send VIPcheck™ plates/cultures with resistant or susceptible isolates **to Radboud University Medical Center** (Paul Verweij)

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Confirmation of fungus (beta-tubulin)
MIC testing (EUCAST)
Analysis of resistance mechanisms (*cyp51A*)

Clinical data

Documentation of anonymized patient data in a web-based case report form after treatment completion. The questionnaire is accessible through www.clinicalsurveys.net.

↓ Documentation by site (250EUR) or by CLARITY team

→ Comprehensive analysis
Joint publication

[1]Verweij et al. *Clin Infect Dis*. 2016 Feb 1; 62(3): 362–368; [2]De Pauw, Walsh et al. *Clin Infect Dis*. 2008 Jun 15; 46(12): 1813–1821

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