Table 1. Management of Posaconazole-Induced Pseudohyperaldosteronism - p2

8	65y F; Prophylaxis (HSCT)	Dose Reduction (300mg→200mg)	5 weeks	K	4.1	3.3	11-deoxycortisol	1120	682
				HCO_3	27	22	Aldosterone	0	3.4
				SBP	173	135	Renin Activity	0.1	2
				DBP	79	74	Posaconazole	6.4	4.4
9	72y F; Treatment (Empiric)	Dose Reduction (300mg→200mg)	5 weeks	K	3.1	3.8	11-deoxycortisol	803	42.6
				HCO_3	29	25	Aldosterone	0	0
				SBP	149	148	Renin Activity	0.1	0
				DBP	79	86	Posaconazole	5.8	2.7
10	37y F; Treatment (Mucormycosis)	Dose Reduction (300mg→100mg)	6 weeks	K	3.7	3.8	11-deoxycortisol	348	14.3
				HCO_3	22	26	Aldosterone	0	0
				SBP	137	124	Renin Activity	1.2	0.7
				DBP	102	85	Posaconazole	3.5	2.2
11	43y M; Treatment (Coccidioidomycosis)	Dose Reduction* (300mg→200mg)	33 weeks	K	3.8	4	11-deoxycortisol	117	39.3
				HCO_3	27	28	Aldosterone	0	0
				SBP	147	150	Renin Activity	0.6	1.1
				DBP	90	82	Posaconazole	2.2	1.3
12	72y M; Treatment (Coccidioidomycosis)	Dose Reduction (200mg→100mg)	9 weeks	K	5.1	4.8	11-deoxycortisol	188	72.7
				HCO_3	26	22	Aldosterone	0	0
				SBP	130	124	Renin Activity	0.7	1.3
				DBP	71	76	Posaconazole	1.9	0.7
13	53y F; Treatment (Coccidioidomycosis)	Dose Reduction (300mg→100mg)	32 weeks	K	4	4.1	11-deoxycortisol	101	60.2
				HCO_3	23	25	Aldosterone	0	3.9
				SBP	129	120	Renin Activity	0.2	1
				DBP	84	76	Posaconazole	4.4	0.4
14	73y M; Treatment (Mucormycosis)	Dose Reduction (300mg→100mg)	70 weeks	K	3.3	4.7	11-deoxycortisol	293	23
				HCO_3	21	20	Aldosterone	0	2
				SBP	154	145	Renin Activity	0	2.3
				DBP	69	76	Posaconazole	5	0.7
15	41y F; Treatment (Coccidioidomycosis)	Dose Reduction (600mg→300mg)	6 weeks	K	3.7	4.1	11-deoxycortisol	233	79.2
				HCO_3	24	22	Aldosterone	-	-
				SBP	134	100	Renin Activity	-	-
				DBP	95	94	Posaconazole	2.4	1

Table 1. Management of Posaconazole-Induced Pseudohyperaldosteronism - p3

16	45y M; Treatment (Coccidioidomycosis)	Dose Reduction (800mg→400mg)	41 weeks	K	4.1	3.2	11-deoxycortisol	937	21.3
				HCO_3	21	24	Aldosterone	0	0
				SBP	116	164	Renin Activity	0.9	0
				DBP	82	100	Posaconazole	5.2	1.5
17	58y F; Prophylaxis (AML)	Continue Regimen (300mg)	N/A	K	3.7	3.4	11-deoxycortisol	207	-
				HCO_3	21	25	Aldosterone	0	-
				SBP	130	137	Renin Activity	0	-
				DBP	59	55	Posaconazole	1.9	-
18	63y M; Treatment (Mucormycosis)	Continue Regimen (300mg)	N/A	K	4	3.6	11-deoxycortisol	167	-
				HCO_3	30	25	Aldosterone	0	-
				SBP	160	130	Renin Activity	2.9	-
				DBP	66	72	Posaconazole	2	-
19	60y F; Treatment (Coccidioidomycosis)	Continue Regimen (200mg)	N/A	K	3.8	3.9	11-deoxycortisol	78.3	-
				HCO_3	25	22	Aldosterone	0	-
				SBP	126	142	Renin Activity	1.7	-
				DBP	66	88	Posaconazole	2.8	-
20	51y F; Treatment (Mucormycosis)	Dose Reduction (300mg→150mg) Add Spironolactone 25mg daily	24 weeks	K	4	4.5	11-deoxycortisol	214	44.3
				HCO_3	25	23	Aldosterone	3.4	-
				SBP	174	149	Renin Activity	0.1	-
				DBP	79	81	Posaconazole	3.1	1.3

*Patient deceased approximately 5 months after post-intervention values
**Patient initially changed to itraconazole for 2 months before changing back to a reduced dose of posaconazole

KEY:
PRE = Pre-Intervention: Values while on posaconazole prior to intervention
POST = Post-Intervention: Values collected post-intervention

Conclusion: We report our experience with PIPH management, for which there is currently no universally effective strategy. We suggest a stepwise approach for PIPH management, starting with posaconazole dose reduction and repeat assessment of clinical and laboratory parameters. If resolution of PIPH is not achieved, an alternative triazole antifungal or the addition of an aldosterone antagonist are additional potential interventions. Even with this approach, it is possible for PIPH to persist after therapeutic modification. Thus, early diagnosis and continuous, close monitoring of patients is warranted.

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753. Performance Characteristics of Diagnostic Assays in Blastomycosis

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Session: P-30. Eukaryotic Diagnostics

Background: Blastomycosis has historically been a difficult diagnosis to establish, often initially misdiagnosed as bacterial pneumonia. Serologic assays and polymerase chain reaction (PCR) tests are available, but their performance is not well defined. The objective of this study was to characterize their performance.

Methods: Subjects were identified via chart review of patients diagnosed with blastomycosis from 2005 to 2020. A definitive diagnosis was based on fungal culture, histopathology, or cytology. Performance characteristics of the *Blastomyces* antibody enzyme linked immunosorbent assay (ELISA), immunodiffusion (ID), complement fixation (CF), urine and serum antigen ELISAs, and PCR were evaluated in patients with confirmed blastomycosis. Data on patient demographics, location of disease, and mortality was also collected.

Results: We identified 193 patients with blastomycosis. The mean age was 51.8 years (range, 11-84) and 73.6% of patients were male. 42.5% resided in Minnesota, 18.1% in Wisconsin, and 12.9% in Iowa. Diagnosis was based on culture in 142 (73.2%) or histopathology/cytology in 67 (34.7%) patients. Granulomatous inflammation was present in 73.1% (38/52) while 21.2% (41/193) had evidence of extrapulmonary dissemination.

The antibody, ID, and CF assays were positive in 43.5% (37/85), 35.1% (33/94) and 20.5% (8/39) of patients, respectively. Sensitivity of *Blastomyces* PCR was 40% (4/10) in sputum and 75% (21/28) in bronchoalveolar lavage (BAL) fluid. *Blastomyces* urine and serum antigen tests were positive in 68% (34/50) and 50% (9/18) of cases, respectively, while the urine antigen was positive in 63.6% (7/11) of disseminated cases. Patients had a positive *Histoplasma* urine antigen test in 54.1% (20/37) and *Aspergillus* galactomannan in BAL in 34.8% (8/23) of cases. Serum beta-D-glucan test was positive in 16.7% (2/12). 90-day mortality was 21/193 (10.9%) and median time from diagnosis to death was 18 days.

Conclusion: In this cohort, *Blastomyces* urine antigen was the most sensitive noninvasive test, with similar performance in pulmonary and disseminated disease. However, its utility is limited by poor specificity due to cross-reactivity. *Blastomyces* PCR from BAL fluid demonstrated the highest sensitivity. *Blastomyces* antibody, ID, and CF had poor sensitivity.

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754. Performance Characterization of a Real Time PCR Assay for *Pneumocystis jirovecii* in Bronchoalveolar Lavage Fluid and Sputum

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Session: P-30. Eukaryotic Diagnostics

Background: *Pneumocystis jirovecii* pneumonia (PJP) affects immunocompromised patients and contributes significantly to mortality. Outcomes depend on early treatment, making timely and accurate diagnosis critical. Typically, PJP diagnosis is through identification of trophozoite or cyst forms in bronchoalveolar lavage (BAL) fluid or sputum, a labor-intensive and insensitive process. Options for more accessible and sensitive molecular detection are limited. It is known that patients may be colonized, which can cast doubt on the clinical significance of low levels of DNA amplification in qualitative result reporting. In this study, we describe a real time (rt) PCR assay utilizing analyte specific reagent primers targeting the mtLSU gene of *P. jirovecii* and correlate amplification with morphological PJP identification.

Methods: IUHPL Clinical Microbiology assessed sputum or BAL fluid from 109 patients with clinical concern for PJP microscopically via fungal stains (GMS, calcofluor white). Comparative rtPCR was conducted as follows. First, 2µL of residual specimen or control were mixed with an 8µL combination of rtPCR mastermix, control DNA, and primer pairs (Simplexa). No nucleic acid extraction was performed. Real time PCR was executed and analyzed on the LIAISON MDX (DiaSorin) platform. Qualitative amplification results and cycle threshold (CT) values were correlated with microscopic methods to establish performance. Chart review was performed to assess the clinical impact of this assay.

Results: *P. jirovecii* was microscopically detected in 26% (29/109) of samples, while 31.1% (34/109) exhibited amplification by rtPCR. Agreement between the two methods was 95.4%; rtPCR demonstrated 100% sensitivity and 93.8% specificity in comparison.

Conclusion: Our results indicate that this assay has exceptional negative predictive value (100%), and therefore may be valuable as a screening test. Considering this data alone, the positive predictive value is lower (85.3%). Further examination of the data, however, revealed that 80% (4/5) of discrepant results demonstrated CT values of >34, while the highest CT for a microscopically positive sample was 31.2. Further clinical correlation may establish a CT cutoff that will reduce false positive and potentially clinically insignificant cases.

Disclosures: **All Authors**: No reported disclosures