

# Acinetobacter peritoneal dialysis peritonitis: a changing landscape over time

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## OBJECTIVES

*Acinetobacter* species are assuming an increasingly important role in modern medicine, with their persistent presence in health-care settings and antibiotic resistance. However, clinical reports addressing this issue in patients with peritoneal dialysis (PD) peritonitis are rare.

## METHODS

All PD peritonitis episodes caused by *Acinetobacter* that occurred between 1985 and 2012 at a single centre were retrospectively reviewed. Clinical features, microbiological data, and outcomes were analysed, with stratifications based upon temporal periods (before and after 2000).

Table 4. Comparison between the current (after-2000) and the historic cohort

Characteristics	Historic 1 (n=23, 1989) <sup>a</sup>	Historic 2 (n=28, 1994) <sup>b</sup>	Current (after 2000) (n=14)			
Age (years)	41 (9-66)	NA	55 (23-84)			
Gender (male %)	16 (70)	NA	7 (50)			
<b>Comorbidities</b>						
DM	5 (22)	NA	6 (43)			
Autoimmune disorders	2 (9)	NA	1 (7)			
Time to last peritonitis (months)	4 (0.5-14.5)	5 (1-29)	13.6 (3-27)			
<b>Pathogen</b>						
<i>Acinetobacter baumannii</i>	8 (35)	27 (96)	9 (64)			
<i>Acinetobacter iwoffii</i>	3 (13)	1 (4)	3 (21)			
<i>Acinetobacter ursingii</i>	0 (0)	0 (0)	1 (7)			
Unspecified <i>Acinetobacter</i>	12 (52)	0 (0)	1 (7)			
<b>Presumed peritonitis origin</b>						
Break in exchange sterility	4 (17)		5 (36)			
Gastrointestinal bacterial translocation	3 (13)	NA	4 (29)			
Exit site infection or tunnel infection	0 (0)		0 (0)			
Un-identified	16 (70)		5 (36)			
<b>Antibiotic susceptibilities</b>						
Aminoglycoside	S	20 (87)	NA	S	14 (100)	
	I	3 (13)				
Ceftazidime	S	23 (100)	S	19 (69)	S	13 (93)
			R	9 (31)	R	1 (7)
Cefepime	NA		NA		S	14 (100)
Fluoroquinolone	S	23 (100)	S	22 (79)	S	14 (100)

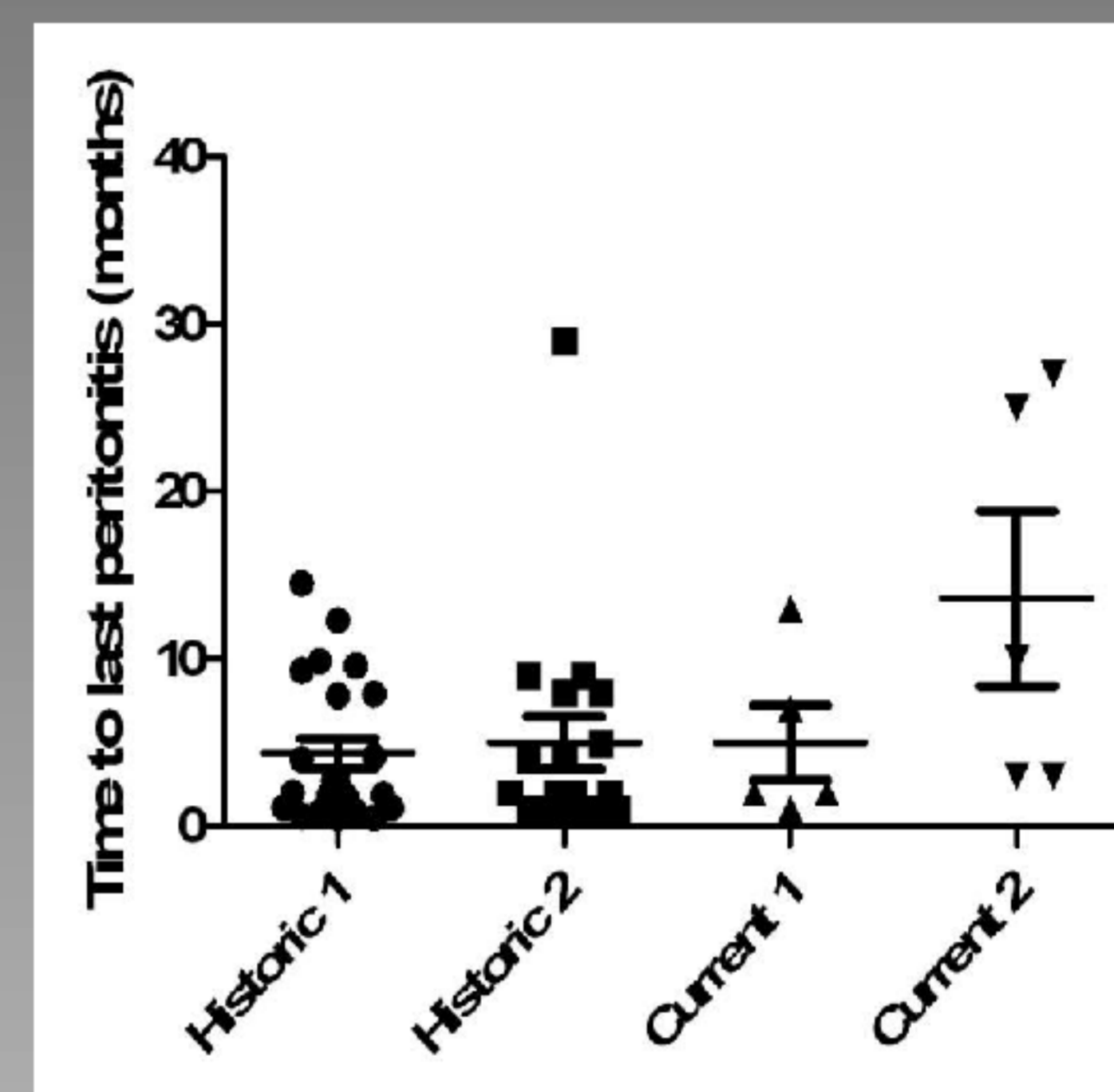


Figure 1. Diagram plotting the distribution of time between the last and current peritonitis episodes in different reports. Our contemporary cohort demonstrated a significantly longer latency compared with the others ( $P < 0.01$  between the after-2000 cohort and the historic 1 and 2 cohorts). Historic 1 cohort, data derived from *Am J Kidney Dis* 1989;14(2):101-4. Historic 2 cohort, data derived from *Perit Dial Int* 1994;14(2):174-7.

## RESULTS

*Acinetobacter* species were responsible for 26 PD peritonitis episodes (3.5% of all episodes) in 25 patients. *A. baumannii* was the most common pathogen (54%), followed by *A. iwoffii* (35%), with the former being predominant after 2000. Significantly more episodes resulted from breaks in exchange sterility after 2000, while those from exit site infections decreased ( $P = 0.01$ ). The interval between the last and current peritonitis episodes lengthened significantly after 2000 (5 vs. 13.6 months;  $P = 0.05$ ). All the isolates were susceptible to cefepime, fluoroquinolone, and aminoglycosides, with a low ceftazidime resistance rate (16%). Nearly half of the patients (46%) required hospitalisation for their *Acinetobacter* PD-associated peritonitis, and 27% required an antibiotic switch. The overall outcome was fair, with no mortality and a 12% technique failure rate, without obvious interval differences.

## CONCLUSIONS

The temporal change in the microbiology and origin of *Acinetobacter* PD-associated peritonitis in our cohort suggested an important evolutionary trend. Appropriate measures, including technique re-education and sterility maintenance, should be taken to decrease the *Acinetobacter* peritonitis incidence in PD patients.

**Keywords:** *Acinetobacter*; end-stage renal disease; gram-negative bacteria; peritoneal dialysis; peritonitis

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