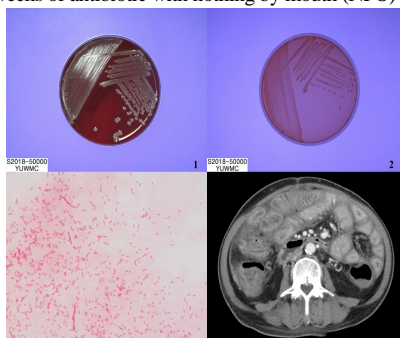


CAPD peritonitis with severe bowel edema caused by Morganella morganii

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Morganella morganii is a rare cause of CAPD peritonitis and may be associated with bowel disease. This paper presents a rare case of CAPD peritonitis caused by *Morganella morganii*. A 65-year-old man on continuous ambulatory peritoneal dialysis (CAPD) developed abdominal pain and turbid peritoneal fluid. He presented a history of ongoing constipation for 5 days. This patient had been on CAPD for 2 years with one episode of peritonitis (caused by *Enterobacter agglomerans* 22 months ago). He had past history of liver cirrhosis, B viral hepatitis, type 2 Diabetes, and coronary artery disease. On presentation, he was afebrile, with a blood pressure of 80/52mmHg. Abdominal examination showed diffuse generalized tenderness, associated with decreased bowel sound and diffuse abdominal wall edema. No expressible infection sign was observed the catheter exit site. The white blood cell count was 11,090 cells/ μ L, C-reactive protein level was 8.98 mg/dL, and the white blood cell (WBC) count of the peritoneal fluid was 5,950 cells/ml (with 76% polymorphonuclear neutrophils (PMN)), suggesting acute CAPD peritonitis. Empiric antibiotic therapy for PD-associated peritonitis was initiated with intra-peritoneal (IP) ceftazidime and amikacin did not improved the patient's symptoms. Initial cultures from peritoneal fluid grew *Morganella morganii* sensitive to piperacillin/tazobactam, ceftazidime, amikacin, ceftriaxone and ciprofloxacin. Peritoneal fluid leukocyte count decreased to 356 on hospital day 2. However, on 3rd hospital day peritoneal WBC count reincreased 1270 cells/ml (78% PMN). We changed ceftazidime and amikacin following culture results. On 11 hospital day WBC count decreased to 7. However, patient's abdominal pain consisted and computer tomography showed severe bowel edema. On 10 hospital day there was no growth in peritoneal fluid culture and the leukocyte count in peritoneal fluid had declined to 7 cells/ μ L. After 3 weeks of antibiotic with nothing by mouth (NPO) patients was discharged with improved general condition.



Author / Year (Reference)	Gender / Age	Comorbidities	Duration of PD (Months)	Co-infection	Initial Treatment	Final Treatment	Treatment Duration (Days)	Complication
H.Atalay / 2010	F / 55	Hypertension	22	Providencia Rettgeri	Cefepazone, Vancomycin	Imipenem, Amikacin HD Convert	14	Uncertain
M.T.Tsai / 2013	F / 62	Hypertension	6	No	Cefmetazole	Ceftazidime	10	Uncertain
M.Winpestl / 2013	M / 53	IgA Nephropathy	10	No	Vancomycin, Ceftazidime	Ceftazidime Intermittent HD	13	Diverticulosis
S.Sipahi / 2014	F / 65	Diabetes Mellitus	6	No	Cefazolin, Ceftazidime	Ceftazidime	14	Uncertain
Y.Kimura / 2015	M / 79	Nephrosclerosis	12	No	Cefazolin, Ceftazidime	Cefazolin, Ceftazidime HD Convert	16	Diverticulosis
V.Keskar / 2017	F / 57	Diabetes Mellitus, Alcoholic LC	Unknown	Coagulase Negative Staphylococcus	Vancomycin, Ceftazidime	Tobramycin, Oral Ciprofloxacin	21	Diverticulosis
This Case / 2018	M / 65	Diabetes Mellitus, B Viral LC	24	No	Cefazolin, Amikacin	Ceftazidime, Amikacin	15	Bowel Edema

Serum phosphorous is the independent risk factor for vascular access dysfunction

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Background/Aims: It is known that maintenance of function of arteriovenous fistula (AVF) is very important in the management of hemodialysis (HD) patients. Therefore, identifying a risk factor for decreased vascular access flow has a clinical relevance in real world practice. Although hyperphosphatemia plays a crucial role in the pathogenesis of vascular calcification, there is lack of studies evaluating the effect of hyperphosphatemia on AVF. This study investigated the impact of serum phosphorous (P) on vascular access flow in HD patients. **Methods:** Sixty-two maintenance HD patients who visited dialysis unit of Bundang CHA Medical Center from November 2016 to December 2017 were included in this study. Serum P levels were determined every month, and time-averaged serum P was calculated. All patients had left arm AVF (side to side anastomosis) and vascular access flow was assessed by Transonic HD 03. Decreased vascular access flow was defined as less than 600 mL/min. **Results:** The mean age was 57.9 ± 12.1 years, 32 patients (51.6%) were men. The mean serum P levels were 5.1 ± 1.1 mg/dL and the vascular access flow was $1,071.4 \pm 504.2$ mL/min. Decreased vascular access flow was observed in 14 of 62 patients (22.6%). In univariate analysis, higher serum P was significantly associated with decreased vascular access flow (odds ratio [OR]=2.089, 95% confidence interval [CI]=1.159-3.766, P=0.014). But there was no significant association of dialysis blood flow rate, ejection fraction on echocardiography and serum calcium (Ca) levels with vascular access flow. Multivariable analysis indicated that higher serum P was independently associated with greater risk of decreased vascular access flow (OR=4.012, 95% CI=1.651-9.711, P=0.002). Old age, reduced EF, low dialysis blood flow rate and higher serum Ca was not associated with vascular access flow. **Conclusions:** This study demonstrated that higher serum P was the independent risk factor for decreased vascular access flow in maintenance HD patients. Serial monitoring of serum P may be helpful to stratify the risk of vascular access dysfunction in these patients.

Table 1. Baseline characteristics of participants according to vascular access dysfunction

	All (n=62)	Patients with vascular access dysfunction (n=14)	Patients without vascular access dysfunction (n=48)	P
Vascular access flow, mL/min	1071.4 \pm 504.2	423.9 \pm 138.1	1380.2 \pm 404.2	0.00
Age, years	57.9 \pm 12.1	59.9 \pm 14.7	57.3 \pm 11.3	0.484
Men, n (%)	32 (51.6%)	7 (50.0%)	25 (52.1%)	1.00
Diabetes mellitus, n (%)	32 (51.6%)	9 (64.3%)	23 (47.9%)	0.367
CVD, n (%)	24 (38.7%)	5 (35.7%)	19 (39.6%)	1.00
SBP, mmHg	132.9 \pm 16.5	135.4 \pm 11.5	132.2 \pm 17.7	0.532
DBP, mmHg	74.9 \pm 10.8	71.8 \pm 10.1	75.8 \pm 10.9	0.220
Pulse rate, bpm	81.2 \pm 11.2	79.1 \pm 13.4	81.9 \pm 10.6	0.415
Hemoglobin, g/dL	10.0 \pm 1.1	9.8 \pm 1.2	10.1 \pm 1.0	0.343
Platelet, $\times 10^3/\mu$ L	227.4 \pm 87.3	249.9 \pm 102.0	220.9 \pm 82.6	0.279
BUN, mg/dL	43.2 \pm 15.4	40.1 \pm 16.1	44.2 \pm 15.3	0.392
Creatinine, mg/dL	7.2 \pm 1.2	7.3 \pm 1.0	7.1 \pm 1.1	0.516
Albumin, g/dL	3.5 \pm 0.4	3.5 \pm 0.4	3.5 \pm 0.4	0.954
Total cholesterol, mg/dL	175.6 \pm 45.4	166.0 \pm 37.7	178.4 \pm 47.4	0.372
Calcium, mg/dL	8.6 \pm 0.6	8.6 \pm 0.5	8.6 \pm 0.6	0.98
Phosphorus, mg/dL	5.1 \pm 1.1	5.7 \pm 1.2	4.8 \pm 1.0	0.008
Blood flow rate, mL/min	284.7 \pm 28.5	278.5 \pm 35.7	286.4 \pm 26.2	0.367
URR (%)	72.3 \pm 4.3	73.1 \pm 3.8	71.8 \pm 4.4	0.313
Ejection fraction, %	61.2 \pm 11.9	58.6 \pm 15.2	62 \pm 10.8	0.357

Note: Data are expressed as the mean \pm standard deviation, median (interquartile range), or number of patients (percent).

Abbreviation: BUN, blood urea nitrogen; CVD, cardiovascular disease; DBP, diastolic blood pressure; SBP, systolic blood pressure; URR, urea reduction ratio

Table 2. Univariate and multivariable binary logistic regression analyses for vascular access dysfunction

	Univariate		Multivariable	
	OR (95% CI)	P	OR (95% CI)	P
Age (per 1 years)	1.018 (0.988-1.071)	0.478	1.072 (0.986-1.166)	0.102
Women (versus men)	1.087 (0.330-3.567)	0.889	0.408 (0.116-1.380)	0.156
Diabetes (versus non-diabetes)	1.957 (0.571-6.702)	0.285	1.360 (0.362-5.238)	0.551
Calcium (per 1mg/dL increase)	0.988 (0.378-2.584)	0.98	1.847 (0.403-8.476)	0.430
Phosphorus (per 1mg/dL increase)	2.089 (1.159-3.766)	0.014	4.012 (1.651-9.711)	0.002
BFR (per 1 mL/min increase)	0.990 (0.969-1.012)	0.501	0.979 (0.948-1.012)	0.212
EF (per 1% increase)	0.977 (0.911-1.025)	0.354	0.949 (0.893-1.009)	0.096

Abbreviation: BFR, blood flow rate; CI, confidence interval; EF, ejection fraction