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Hypertensive pulmonary edema related to desmopressin acetate

Edema pulmonar hipertensivo relacionado con el acetato de desmopresina

Dear Editor,

Desmopressin acetate, known as DDVAP (1-deamino-8-D-arginine vasopressin), increases factors VIII and vWF, shortens activated partial thrombin time and bleeding time. Desmopressin is the best treatment in emergency situation, increases the release of the von Willebrand factor from the endothelium in uremic bleeding.[1] Potential side effects of desmopressin include headache, nausea, malaise, hypotension, facial flushing, tachycardia, dizziness and hyponatremia.[2] However hypertension weren't defined before, we present two cases of hypertensive pulmonary edema after the infusion of desmopressin prior to ultrasound (USG) guided percutaneous kidney biopsy.

A 49 year-old female with end-stage renal failure secondary to unknown etiology received a living-donor transplant 3 years ago, was scheduled to have a transplant biopsy due to deteriorating graft function. Blood pressure was 160/90 mmHg, pulse was 72 beats/min, and respiratory rate was 14 breaths/min.

She was given an infusion of 15 μg of intravenous DDAVP over 20 min in preparation for biopsy. Biopsy performed without complications. After 40 min from the infusion; the patient suffers from dyspnea. Her blood pressure elevated to 220/140 mmHg. On examination there was crepitation on the both lungs on every area. SPO2 was 70 on the pulse oximeter.

Respiratory rate was elevated to 40. Immediately she was given furosemide and nitroglycerin infusion with oxygen. Her SPO2 levels elevated after the treatment. She was monitoring all day and her blood pressure fall to 140/80 after 8 h.

A 42 year-old male with crescentic IgA nephropathy, who was diagnosed 7 months ago, was treated with pulse steroid and cyclophosphamide for induction. On examination, the patient was relieve, body temperature was 36.6 C, blood pressure was 150/90 mmHg, pulse was 72 beats/min, and respiratory rate was 14 breaths/min.

He was given an infusion of 15 μg of intravenous DDAVP over 20 min in preparation for biopsy. Biopsy performed without complications. After 2 h from the infusion; the patient suffers from dyspnea. His blood pressure elevated to 230/120 mmHg. He was treated with oxygen, furosemide and nitroglycerin. His blood pressure was 150/90 mmHg after 8 h of intense therapy. His oxygen saturation had risen to 98 from 75 on room air.

Both of cases didn't have any history of coronary heart disease or heart failure but they have hypervolemia due to renal failure.

Hergesell et al. reported that risks in order to minimize, to adopt adequate biopsy technique is not only important, but also high-risk patients, especially uncontrolled blood pressure, with a clotting disorder, or has emphasized that it is necessary to exclude those unwilling to cooperate.[3] Mannucci et al. reported that prohibited for use in who have arterial disease. DDAVP related myocardial infarction and cerebrovascular disease has been reported to case presentations, can't be used in patients with known cardiovascular disease.[4]

DDAVP was an analog of vasopressin, may provoke symptomatic hyponatremia because of water retention. According to the U.S. Food and Drug Administration Agency, the half-life of DDAVP in patients with severe renal impairment can be extend by 9 h. Therefore, patients should be advised to restrict fluid intake from 1 h before to 9 h after administration of DDAVP. Blood sodium control is proposed following use in hyponatremic patient.[5,6]

There are no publications that specify the use of DDAVP in hypervolemic patient. In our cases DDAVP effected like vasopressin and increased water retention in the hypervolemic patients. In patients without structural heart disease DDAVP may have a role that could lead to hypertensive pulmonary edema. In our patients; increased blood pressure responded diuretic and vasodilator treatment.

Hypertension is not a limiting factor to evaluate the use of DDAVP because of falsely elevated blood pressure due to anxiety. We recommend careful use of DDAVP in hypertensive patients due to a hypervolemia without structural heart disease, and we think this situation could lead role in the hypertensive pulmonary edema.

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Serratia marcescens, *Morganella morganii*, *Klebsiella oxytoca* related peritonitis attacks in a patient on automated peritoneal dialysis: A case report

Serratia marcescens, *Morganella morganii*, *Klebsiella oxytoca* relacionados con ataques de peritonitis en un paciente en diálisis peritoneal automatizada: Un caso

Dear Editor,

Bacterial peritonitis is a common complication of peritoneal dialysis.[1] We report here a case presented with peritonitis attacks caused by rarely reported unusual pathogens, probably related with poor home environment and hygienic conditions.

A 57-year-old female patient had a history of end-stage renal disease secondary to hypertensive nephrosclerosis and undergone dialysis for 4 years. She was sharing a small house in poor hygienic conditions with eleven other family members with low socioeconomic status. Five months after the initiation of automated peritoneal dialysis (APD), the patient presented with abdominal pain and nausea to our PD clinic. She was febrile (38 °C), had involuntary abdominal guarding and rebound tenderness on physical examination. Dialysate white blood cell count was 1100/mm^3 (79% neutrophils). Empiric antibiotherapy was initiated with intraperitoneal cefazolin (1 g/day) and oral ciprofloxacin (250 mg every 12 h).

A pure growth of *Serratia marcescens* was obtained in both different culture media. The organism was resistant to cefazolin, ceftriaxone, piperacillin/tazobactam, but sensitive to cefepime. Cefazolin was stopped; cefepime could not be used due to a drug shortage; instead, intraperitoneal gentamicin (0.6 mg/kg/day). Oral ciprofloxacin was also continued based upon the susceptibility results. Following the treatment modification, high-sensitivity CRP level decreased from 240 mg/L to 9 mg/L. Peritoneal effluent became clear and drainage fluid leukocyte count was 100/mm^3 (10% neutrophils) on the third week of admission.

The patient was readmitted to the hospital with similar complaints 7 months after the first peritonitis attack. Peritoneal fluid leukocyte count was found to be 17 000/mm^3 and empiric antibiotherapy was initiated with intraperitoneal cefazolin (1 g/day) and gentamicin (0.6 mg/kg/day). Dialysate cultures showed the growth of *Morganella morganii*, resistant to cefazolin, cefuroxime but sensitive to cefepime, gentamicin. Cefazolin was stopped and gentamicin was continued