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Title: THE TRUTH ABOUT URINARY POTASSIUM AND INTERSTITIAL CYSTITIS: NEW INSIGHTS ON THE POTASSIUM SENSITIVITY TEST

Aims:

In 1994 Parsons described the potassium chloride (KCl) sensitivity test and demonstrated that most IC patients, but not controls, are sensitive to intravesical instillation of potassium. The Parsons' test is performed with a pharmacologic dose of KCl, more than 5 times higher than normal urinary levels. We performed a series of three experiments to assess the significance physiologic concentrations of urinary potassium. Our hypothesis was that physiologic levels of potassium would not be clinically significant.

Methods:

Experiment 1 was designed to determine whether KCl sensitivity is an absolute characteristic of an individual patient or a dose-dependent phenomenon. Twenty symptomatic IC patients and 20 controls underwent sequential bladder instillation with 0, 10, 20, 30 and 40mEq KCl, and 0, 20, 40 and 60 mEq of KCl, respectively. Pain and urgency were rated on a scale of 1-10 for each solution. Experiment 2 determined the mean concentration of urinary potassium for 20 symptomatic IC patients, 10 negative controls, and 10 positive controls (overactive bladder patients). Spot urine specimens were collected on a convenience basis.

The third study investigated the physiologic significance of potassium through urodynamic testing. Twenty-three female patients (aged 24-71) underwent single channel filling/voiding cystometry with two solutions: normal saline, and normal saline with 115 mEq/l KCl (the mean urinary potassium concentration plus two standard deviations). Subjects and investigators were blinded to the order of the solution. Bladder capacity for two fills with each solution and the patients' qualitative estimation of the "worst" solution were recorded.

Results:

Experiment 1 showed that potassium sensitivity is a dose-dependent response for both IC patients and controls. IC patients were much more sensitive than controls but, at very high KCl concentrations, almost all controls are also sensitive. Experiment 2 produced a mean urinary potassium level of 53.4 (SD = 30.9) mEq/l for the IC patients, similar to the control groups.

Experiment 3 demonstrated a mean bladder capacity with normal saline of 234cc (SD=78) and 208cc (SD=87), respectively. The difference between the two solutions was statistically significant (p=.05). In addition, patients correctly identified the KCl solution 74% of the time, with only 11% choosing saline, and 16% reporting no difference between the solutions. Patients with objectively more severe disease (bladder capacity < 600cc and/or ulcers or cracking during cystoscopy under anesthesia) showed very marked sensitivity to potassium.

Conclusions:

We confirmed the original report of sensitivity to KCl by Parsons but demonstrated that sensitivity is a dose-dependent phenomenon, not an all-or-nothing quality. Furthermore, we have shown that physiologic concentrations of potassium reduce cystometric bladder capacity and that IC patients are able to detect physiologic levels of urinary potassium as an unpleasant sensation. These results argue for continued research into the role of potassium and its interaction with the bladder epithelium. (Funding ICA Pilot Projects Grant)