A Comparison of Blood Measures while NFL players are Experiencing EAMC and after IV Treatment when EAMC are Alleviated

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Context: Electrolyte imbalances and specifically sodium (Na⁺) losses have been implicated in exercise associated muscle cramps (EAMC). Objective: To measure blood electrolytes, hemoglobin, hematocrit and lactate while players have EAMC and make comparisons to those same measures immediately after IV treatment when EAMC are alleviated, and to compare sweat rate (SwtR), sweat Na⁺ concentration [SwtNa⁺] and sweat Na⁺ losses in these players to matched controls with no EAMC (No-EAMC). Design: Observational cohort study. Setting: Pre-season training camp of one NFL team. Patients or Other Participants: Seven NFL players who sustained EAMC with the following physical characteristics: age=25±3yr, mass=114±24 kg, height=188±6cm, BSA=2.39±0.27m² and BSA/mass =213±20cm²/kg agreed to participate and were matched with 7 No-EAMC volunteers with age=27±3yr, mass=111±30 kg, height=187±10cm, BSA=2.34±0.34m² and BSA/mass=216±25 cm²/kg. Interventions: Seven cc of blood was drawn from players during EAMC and immediately analyzed. A second blood sample was taken after IV treatment with 1 liter of 9% saline and I liter of ½ saline with 5% dextrose. Blood sodium, potassium and chloride were analyzed by ion-selective electrode, hematocrit by microhematocrit technique, and hemoglobin and lactate by hemoglobin and lactate meters respectively. All 14 players (7 EAMC and 7 No-EAMC) participated in sweat testing during the second week of camp. Sweat samples were collected from sweat patches applied to the upper forearm after the skin was washed with deionized water and dried with sterile gauze. The neat sweat samples were frozen and later analyzed by flame photometry for [SwtNa⁺]. Sweat rate was calculated as change in mass plus fluids consumed minus urine produced/practice time. Paired t-tests were used for comparisons between pre and post IV blood measures and between EAMC and No-EAMC sweat measures. Main Outcome Measures: Blood sodium, potassium, chloride, hemoglobin, hematocrit and lactate, and SwtR, [SwtNa⁺] and Na⁺ losses. Results: Compared with pre IV there were significantly lower post IV blood measures of sodium (141.8±2.8 versus 138.6± 2.9mmol/l, P = .02), hemoglobin (17.3±1 versus 14.7±1 mg/dl, P = .004), and hematocrit (51±3% versus 44±2% P < .001). Conversely, chloride increased significantly post IV (99±2 versus 104±2 mol/l, P<.001) and in 6 of the 7 players was clinically low, < 100 mmol/I during EAMC. There were no differences in lactate or potassium but pre IV potassium ranged from 3.7 – 7.6 mmol/l. There were no differences between EAMC and No-EAMC in physical characteristics, SwtR (1.7±.2 and 1.8±.6 l/h), [SwtNa⁺] (52±19 and 49±23 mmol/l) or Na⁺ losses (2109±1203 and 1983±997 mg/h). Conclusions: Low blood chloride may be important in the etiology of EAMC, potentially caused by a pathologic anion gap from excessive alkalosis, or by reducing hyperpolarization of the alpha motor promoting hyperexcitability. Hyperkalemia in a concern and EAMC appears unrelated to sweat sodium concentration or sodium losses. Word Count: 450