A study of potassium canrenoate on the raised intracranial pressure

Yoji Node, Kozo Yajima, and Shozo Nakazawa

Department of Neurosurgery, Nippon Medical School

Brain edema is still one of the most significant complications in a variety of neurological disorders and after neurosurgical operations. The diagnosis and treatment of this condition have critical importance for recovery of the patients. Therapy of brain edema today consists mainly of dehydration employing hypertonic solutions such as mannitol and glycerol\(^1\) or powerful diuretics and steroids.

Several studies have demonstrated that potassium canrenoate (C-K), an aldosterone antagonist, reduces perifocal brain edema\(^2\)\(^-\)\(^3\). The action of C-K on the central nervous system is probably not specific but rather due to a reactive increase of aldosterone as a result of the competitive blockades of its renal site of action by the antagonist. However little information is available as regards the effects of C-K on the raised intracranial pressure (ICP). We have studied the effects of C-K on the raised ICP and on the metabolism of electrolytes and hormones.

Fourteen patients were subjected to this study. Seven were male and 7 female. The age of the patients ranged from 34 to 56 years (average: 45). Of these patients, 8 were with cerebral aneurysms and 6 with brain tumors. All patients were administered 400 mg of C-K per day intravenously over 2 days before and 3 days following the operation. ICP was monitored during of this period by epidurally placed transducer (Koningsberg, p-15) through a frontal burr hole. And we compared the postoperative mean baseline intracranial pressure (MB-ICP) of the C-K administered 14 patients with that of the C-K non-administered 23 patients. The mean ICP was calculated as the diastolic pressure plus one-third of the pulse pressure\(^4\), and the baseline ICP was taken as the maximum stable level of ICP recording. Levels of electrolytes in plasma and urine as well as aldosterone, activities of renin, antidiuretic hormone, catecholamines (epinephrine, norepinephrine), and cyclic nucleotides (cyclic AMP and cyclic GMP) were also measured in all C-K treated patients.

After administration of C-K there were the increase of sodium, decrease of potassium, and increase of the sodium-potassium ratio with a statistical significance at p<0.01, and urine volume tended to increase. There were no significant changes in the plasma electrolytes after administration of C-K, nor in the value of antidiuretic hormone, catecholamines, and cyclic nucleotides.

Table 1 A shows the postoperative mean baseline ICP (MB-ICP). In the C-K non-
administered 23 patients, postoperative MB-ICP was less than 15 mmHg in all 11 patients (48%) and greater than 15 mmHg in 12 patients (52%). On the other hand, in the C-K administered 14 patients, MB-ICP was less than 15 mmHg in 9 patients (64%) and greater than 15 mmHg in 5 patients (36%). Fig. 1 shows the changes of aldosterone in plasma in the C-K administered 14 patients. The changes of aldosterone in plasma were divided into four groups as shown in Table 1B. A: increasing group. B: at first decreasing and thereafter increasing group. C: decreasing group. D: at first increasing and thereafter decreasing group. In group C and D, 50% of the patients showed less than 15 mmHg in MB-ICP. In contrast, in group A, 75% of patients and in group B all the patients exhibited less than 15 mmHg in MB-ICP. These results suggest that, in group A and B, the increase of the postoperative MB-ICP have been more effectively presented than in group C and D.

We conclude therefore that potassium canrenoate could present the increase of the postoperative mean baseline intracranial pressure, and that the changes of aldosterone in plasma might be one of the potent factors for the effect of C-K on the raised ICP.

References


(Received for publication, February 2, 1984)