

Potassium Homeostasis in Patients with Acute Organophosphorus Poisoning

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Introduction : Although only 2% of total body potassium (70-100meq) remains in the extracellular compartment, it plays a critical role in maintaining cell membrane resting potential (RMP). Relatively small changes in extracellular potassium concentration can significantly alter RMP and functional activity of electrically excitable cells.

Hypokalaemia was noted on admission in 5/60 patients with organophosphate poisoning¹. This phenomenon may compound the weakness due to inhibition of acetylcholine esterase by organophosphorus compounds (OP). K is tightly balanced in that urinary K excretion (1 - 1.5mmol/Kg/day) is directly proportional to the total body potassium and is a good marker of total body K. **Methods :** We prospectively measured serum K and urinary potassium excretion on day 1 to 3 in consenting patients following ingestion of OP. **Results :** (table1) There were 35 patients (median age 45 years- IQR 24-45, 18 males) in acute cholinergic phase. 41% ingested chlorpyrifos. Median serum K remained in the lower range of normal. K excretion was lower than average 1mmol/Kg/day on day 1. There was a significant reduction in median K excretion on day 2 (p=0.004) which did not differ between OP type and gender. **Discussion :** Potassium homeostasis appears to be altered in acute organophosphate poisoning with evidence suggesting renal conservation of potassium. Potential causes include intracellular shift, poor intake, gastrointestinal loss and changes in aldosterone and cortisol levels. It is possible that alterations in K may alter neuromuscular junction function in OP poisoned patients or alter cardiac conduction and contribute to the overall mortality and morbidity.

Conclusion : Patients with OP poisoning seem to conserve excretion of K to maintain normal serum K even on day 1 which reached a maximum on day 2. Further studied should be done to investigate the causes of this phenomenon and the effect of routine K replacement to K excretion and RMP. **References :** 1. Mahdi Balali-Mood et al., Use of high doses of sodium bicarbonate in acute organophosphorous pesticide poisoning is advancing. Clinical Toxicology 45, no. 1 (2007): 92,

	Day1	Day2	Day3
Serum K (median and IQR (mmol/L))	3.6(3.2-3.8)	3.8(3.4-4)	3.6(3.2-4)
Urinary K excretion (median and IQR (mmol/Kg/day))	0.8(0.3-1)	0.2(0.1-0.6)	0.45(0.28-0.7)

Table 1