REVIEW ARTICLE

Succinylcholine-Induced Hyperkalemia in Patients with Renal Failure: An Old Question Revisited

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The administration of succinylcholine (SCh) in humans results in a mild and transient hyperkalemia. In normal individuals, the increase in serum potassium (K^+) is approximately 0.5–1.0 mEq/L, occurs within 3–5 minutes after the IV administration of SCh, and lasts <10–15 minutes (1). This increase is probably caused by K^+ release from cells as a result of depolarization at the neuromuscular junction. However, in certain conditions, such as trauma, burns, infection, and certain neuromuscular disorders (including spinal cord injury, upper motor lesions, and structural brain damage, peripheral nerve injury, peripheral neuropathy, Parkinson's disease, tetanus, and muscular dystrophy), there is an exaggerated increase in the serum K^+ level that may manifest clinically in cardiac dysrhythmias and even cardiac arrest (1). In these conditions, it is believed that there is a proliferation of postsynaptic acetylcholine receptors beyond the neuromuscular junction (extrajunctional receptors) with the result that K^+ flux is not restricted to the neuromuscular junction. This proliferation leads to an exaggerated increase in serum K^+ levels on depolarization induced by SCh administration.

Because K^+ homeostasis is disturbed in patients with renal failure, the use of SCh in such patients has raised concerns of an exaggerated hyperkalemic response, with its resultant adverse cardiac effects. However, after several case reports (2–4), case series (5–6), and controlled studies (7–10), the consensus has been that use of SCh is safe in patients with renal failure, provided that there is no associated neuropathy or preoperative hyperkalemia, and that repeated SCh doses are avoided (5–10).

Recently, in our institution, several patients with renal failure who were given SCh during surgery developed hyperkalemia in the postoperative period. These patients had normal preoperative serum K^+ levels, but intraoperative levels were not measured. Although the cause of hyperkalemia in these patients was unclear, questions regarding the safety of SCh in patients with renal failure were raised. We thus decided to critically review the literature on the safety of SCh in renal failure patients undergoing surgery. In this report, we summarize the findings of this review and offer our conclusions.

Studies of SCh and Renal Failure

Roth and Wuthrich (2) first raised concerns about the risk of hyperkalemia and cardiac dysrhythmias after the use of SCh in patients with renal failure. They reported two patients with renal failure (a 74-yr-old woman and a 2-yr-old boy) who experienced cardiac arrest within minutes after the administration of SCh while undergoing emergency surgery. Both patients had elevated preoperative serum K^+ levels (6.2 mEq/L and 6.5 mEq/L). However, post SCh administration K^+ levels were not measured in the first case, so a hyperkalemic cause for the cardiac arrest could not be established. The K^+ levels in the second (pediatric) case increased to 8.9 mEq/L. In patients with renal failure, Roth and Wuthrich (2) suggested that SCh should be absolutely contraindicated if preoperative serum K^+ levels are elevated, and relatively contraindicated if they are normal.

In a second case report, Powell (3) described a 44-yr-old woman with renal failure and a normal preoperative serum K^+ level (4.1 mEq/L) who was given three doses of SCh within 25 minutes of anesthetic induction while undergoing elective bilateral nephrectomy. Within 6 minutes after the last SCh dose, the patient developed electrocardiographic (EKG) changes consistent with hyperkalemia and several short runs of ventricular tachycardia and had a serum K^+ level of 6.9 mEq/L (3). Powell's (3) recommendations were even more stringent than Roth and Wuthrich's (2), suggesting that SCh should be absolutely contraindicated in all patients with renal failure, even when the preoperative K^+ level is normal.

In a third case report, Walton and Farman (4) reported on a patient with renal failure and uremic polyneuropathy who received two doses (50 mg and

Accepted for publication March 22, 2000.

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0003-2999/00

25 mg) of SCh while undergoing bronchoscopy. The serum K^+ level, which was 4.5 mEq/L at baseline, did not increase after the first dose, but did so markedly to 7.3 mEq/L within two minutes after the second dose. However, the patient did not exhibit any cardiac disturbances. The authors suggested that the combination of polyneuropathy and repeated doses of SCh may have triggered the hyperkalemia.

Subsequently, four controlled studies (7–10) and two case series (5,6) that specifically measured serial serum K^+ levels after SCh administration in patients with renal failure undergoing surgery did not show an increase in serum K^+ more than that expected in individuals with normal renal function.

In the first controlled study, Koide and Waud (7) measured serum K^+ levels serially in 34 patients with chronic renal failure and in 22 with normal renal function. The mean levels of preoperative serum K^+ in patients with and without renal failure who were pretreated with d-tubocurare were 3.8 ± 0.6 mEq/L and 4.1 ± 0.3 mEq/L, respectively. The levels were 4.4 ± 0.8 mEq/L and 4.2 ± 0.5 mEq/L, respectively, in those who were not pretreated with d-tubocurare. After a single of dose of SCh (1 mg/kg body weight), the mean maximal increase in K^+ levels observed in both groups of patients was 0.5 mEq/L, with more variation in patients with renal failure. The authors did not observe any cardiac arrhythmias other than sinus tachycardia. They concluded that a single dose of SCh is safe in patients with renal failure who have preoperative serum K^+ levels below 5.5 mEq/L. However, they cautioned its use in patients with high levels of preoperative serum K^+, in whom even a small increase in the serum K^+ may increase the risk of cardiac arrhythmias.

In the second controlled study, Miller et al. (8) compared serial serum K^+ concentrations after a single dose of SCh (1 mg/kg) in 10 patients with renal failure who were undergoing renal transplantation with 10 patients without renal failure undergoing intraabdominal surgery. Mean preoperative serum K^+ levels in patients with and without renal failure were 5.0 ± 0.71 mEq/L (range 4.3 to 6.6 mEq/L; median 4.7 mEq/L) and 3.7 ± 0.42 mEq/L (range 2.8 to 4.1 mEq/L; median 3.85 mEq/L), respectively. They observed that the mean maximal increase in serum K^+ in patients with renal failure was 0.24 ± 0.45 mEq/L (range −0.4 to + 0.6 mEq/L) which was not significantly different from the increase observed in patients with no renal disease, 0.18 ± 0.50 mEq/L (range −0.9 to + 0.7). The largest increase was 0.7 mEq/L, which was observed in a patient with no renal disease. The authors concluded that SCh at this dose was not contraindicated in patients with renal failure in the absence of uremic neuropathy.

In the third controlled study, Day (9) studied serial serum K^+ levels in 21 patients with chronic renal failure and 54 patients with normal renal function who received either SCh 100 mg IV or suxethonium (a depolarizing muscle relaxant similar in structure to SCh but with a shorter duration of action) 150 mg IV. After SCh administration, there was a similar increase from baseline (4.14 ± 0.47 control and 4.18 ± 0.61 mEq/L renal failure patients) in the mean serum K^+ levels in both groups. However, statistically significant increases in the serum K^+ levels from baseline were observed in the control group at 10 (4.37 ± 0.66 mEq/L) and 25 minutes (4.58 ± 0.72 mEq/L) and in the renal failure group at 2 (4.55 ± 0.49 mEq/L) and 10 minutes (4.60 ± 0.63 mEq/L) after SCh administration. No such increases were seen in patients with renal failure after suxethonium administration. One 24 yr-old patient with acute renal failure who received SCh developed cardiac arrest and was successfully resuscitated. His serum K^+ level at the time of cardiac arrest was 4.3 mEq/L (baseline 4.5 mEq/L). Day postulated that this patient's severe metabolic disturbances, as evidenced by the preoperative plasma urea level (30 mmol/L), may have contributed to the arrest. No cardiac arrhythmias were observed in any of the other renal failure patients.

In the fourth controlled study, Radnay et al. (10) evaluated serial serum K^+ levels in 20 patients with chronic renal failure and 20 with normal renal function who received SCh (0.2 mg/kg) after the induction with neuroleptanesthesia (droperidol, fentanyl, nitrous oxide and oxygen) and pretreatment with hexafluorenium (0.3 mg/kg). Mean baseline serum K^+ levels (3.90 ± 0.10 mEq/L in patients with normal renal function and 4.70 ± 0.12 mEq/L in those with chronic renal failure) decreased substantially after the induction with neuroleptanesthesia (3.59 ± 0.13 mEq/L, 4.17 ± 0.11 mEq/L, respectively) and even further after hexafluorenium administration (3.39 ± 0.10 mEq/L and 3.92 ± 0.11 mEq/L, respectively). Compared with the levels after hexafluorenium administration, the mean levels increased by 0.16 mEq/L in patients with normal renal function and 0.10 mEq/L in those with chronic renal failure after SCh administration. In neither group did the serum K^+ levels exceed baseline levels. Based on these findings, the authors recommended a combination of neuroleptanesthesia and pretreatment with hexafluorenium preceding SCh administration as a suitable and "relatively risk-free" technique for muscle relaxation in patients with chronic renal failure. This technique, however, is not a feasible option; hexafluorenium, a nondepolarizing neuromuscular blocking drug with selective inhibition of plasma cholinesterase that was used mainly to prolong muscle relaxation and to minimize muscle fasciculations induced by SCh, has not been commercially available in this country for nearly two decades (11,12).

In the first case series, Walton and Farman (5) measured serial serum K^+ levels after a dose (1 mg/kg) of SCh in a random sample of 12 candidates for renal transplantation. Initial serum levels of K^+ ranged from 2.7 to 7.3 mEq/L. In 11 of the patients, the variability in K^+ level induced by SCh administration was <0.7 mEq/L; only 7 patients exhibited an elevation in their serum K^+ levels. In the 12th patient, the K^+ level increased by 1.2 mEq/L (from 3.3 mEq/L at baseline to 4.5 mEq/L, two minutes after the administration of SCh) but decreased to 3.5 mEq/L at 6 min. Interestingly, the two patients with high baseline K^+ levels (6.5 mEq/L and 7.3 mEq/L) showed a maximal increase of 0.1 mEq/L and 0.7 mEq/L, respectively. The authors did not report any cardiac disturbances.

In the second case series, Powell and Miller (6) studied the effects of repeated doses of SCh on the serum K^+ levels of 11 patients undergoing renal transplantation. An initial dose of 1 mg/kg was followed by two more doses at 5-minute intervals. Serial serum K^+ levels were measured at baseline, 2, 5, 7, and 10 minutes after each dose of SCh. The mean baseline serum K^+ level was 4.7 ± 0.93 mEq/L. The largest increase in serum K^+ with repeated SCh doses was 0.6 mEq/L. The mean net change was + 0.34 mEq/L with the mean increase and decrease being +0.5 mEq/L and −0.4 mEq/L, respectively. The largest serum K^+ change was negative (-0.9 meq/L). The maximum increase in serum K^+ in the patient with the highest baseline level (6.4 mEq/L) was 0.4 mEq/L and was seen 5 minutes after the second dose of SCh. In 15 of 22 (68%) of the repeated injections, the authors observed sinus bradycardia. The authors concluded that SCh administered repeatedly at this dose was not contraindicated in patients with renal failure.

Effects of Repeated Doses of SCh on Serum K^+ Levels

In the case reports by Powell (3) and by Walton and Farman (4), an increase in serum K^+ levels was seen only after repeated doses of SCh, suggesting that the cumulative effects of multiple doses may increase the risk of hyperkalemia. In addition, a patient who received a second dose of SCh in the study by Koide and Waud (7) had an additional increase in serum K^+ of 1.0 mEq/L, in comparison to a 0.2 mEq/L increase after the first dose. Powell and Miller (6) specifically studied the effects of repeated doses of SCh on the serum K^+ levels in patients with renal failure and did not find an excessive increase in serum K^+ after repeated administration of SCh. Consistent with other reports (13,14), they found that 15 of 22 (68%) repeated injections in their study resulted in sinus bradycardia. List (14) reported bradyarrhythmias in 40% of 96 patients with heart disease after a second dose of SCh. Two of these patients developed asystole that responded to external cardiac massage. The mechanism for the bradycardia is postulated to be mediated via the effect of the parasympathetic nervous system on the heart or via a direct action on the myocardium (13) and not hyperkalemia *per se*. Pretreatment with glycopyrrolate or atropine protects against bradyarrhythmias induced by repeated administration of SCh (13,15–17).

Prevention of SCh-Induced Hyperkalemia

To minimize the hyperkalemia associated with SCh administration, investigators have tried pretreatment with various drugs, including nondepolarizing neuromuscular relaxants (7,10,18–23), flunitrazepam (24), diazepam (25,26), and magnesium sulfate (19) with mixed results. Only Koide and Waud (7) and Radnay et al. (10) specifically evaluated the role of pretreatment in patients with chronic renal failure. Koide and Waud (7) found that pretreatment with d-tubocurarine did not prevent an increase in serum K^+ after SCh administration. In contrast, after the induction with neurolept anesthesia (droperidol, fentanyl, nitrous oxide and oxygen) and pretreatment with hexafluorenium, Radnay et al. (10) reported a SCh-induced increase in mean serum K^+ levels of 0.16 mEq/L in patients with normal renal function and 0.10 mEq/L in those with chronic renal failure. In neither group did the serum K^+ levels after SCh administration exceed baseline (i.e., preinduction) levels. As discussed earlier, this technique is not feasible in clinical practice because of the commercial unavailability of hexafluorenium.

Effects of SCh on Serum K^+ in Chronic Versus Acute Renal Failure

In chronic renal failure, adaptive changes in the kidneys and the gut prevent significant hyperkalemia. Depending on whether K^+ intake is normal or increased, the kidneys can maintain K^+ equilibrium until a glomerular filtration rate of 5–10 mL/min or 10–40 mL/min, respectively. In chronic renal failure, fecal excretion of K^+ is considerably enhanced. However, because the adaptive mechanisms in chronic renal failure are not present in patients with acute renal failure, they are at high risk of developing hyperkalemia (greater than 50% of cases) (27). The hyperkalemia is especially severe in the presence of oliguria and tissue destruction, with or without sepsis.

K^+ is distributed unevenly, with 98% in the intracellular and 2% in the extracellular fluid compartments. As a result, small changes in the extracellular K^+ level cause significant hyperkalemia or hypokalemia, depending on the direction of the K^+ shift. Since

the membrane potential of cells is dependent upon the ratio of the intracellular to the extracellular K^+ concentration [I_K^+/E_K^+], any acute increase in extracellular K^+ will cause depolarization of the membrane, and resultant cardiac dysrhythmias. Thus, the actual level of serum K^+ that causes toxicity may vary, with the rate of rise of K^+ more important than the absolute level. Patients with chronic renal failure with chronic K^+ elevations also have a higher intracellular K^+, so that the ratio [I_K^+/E_K^+] is normal (27,28). Thus, the myocardium appears to have some tolerance to hyperkalemia in uremia, as evidenced by reports of cases of severe hyperkalemia without EKG changes (29). Because of the widespread availability of hemodialysis, a significantly elevated serum K^+ level and its associated morbidity are infrequently encountered. Nevertheless, SCh-induced hyperkalemia in renal failure could result in adverse cardiac outcomes. The demonstrated absence of significant hyperkalemia and resultant adverse cardiac outcomes associated with SCh use in patients with chronic renal failure suggests otherwise.

Discussion

Since SCh was first introduced clinically in the early 1950s, it has been used extensively in situations requiring rapid tracheal intubation during the induction of anesthesia in an effort to avoid aspiration of gastric contents. Rapid induction is possible because of its rapid onset of action (one minute) and short duration of action (seven-eight minutes) when administered in doses of 1 mg/kg. However, because of the undesirable side effects of SCh, which include hyperkalemia, postoperative myalgia, and increased intraocular and intragastric pressure, some of the newer nondepolarizing neuromuscular relaxants, particularly rocuronium with an onset of action almost as rapid and without its side effects, have been recommended as alternatives (13,30). Interestingly, in an excellent review, Durant and Katz (13) in 1982 speculated that "is likely that suxamethonium will be of historical interest. This review may well be a requiem for suxamethonium." Nevertheless, the use of SCh continues, primarily because of the higher costs and longer duration of action of the newer anesthetics.

Based on our review of the literature in English language medical journals, we conclude that there is sufficient evidence to support the current consensus that SCh can be safely administered to patients with renal failure. Of the nine studies (2–10) reviewed that specifically addressed the issue of SCh use and associated hyperkalemia in patients with renal failure, six (two case series and four controlled studies) did not find an excess risk of hyperkalemia or its adverse cardiac effects (5–10). Only three case reports (2–4) reported adverse cardiac effects after SCh use. In one (4), the authors attributed the hyperkalemia to a combination of uremic neuropathy and a repeated dose of SCh. The other two case reports (2,3) implicated SCh as the likely cause of the hyperkalemia and its ensuing cardiac consequences. In one report, SCh-induced hyperkalemia was not confirmed by serum K^+ measurements or EKG evidence of hyperkalemia in one patient (2). In the other, there was a delayed appearance of EKG changes with the hyperkalemia persisting postoperatively for at least 24 hours (3). This finding led Miller et al. (8) to conclude that these effects could not be attributed to SCh-induced hyperkalemia. Alternative factors, such as the presence of acidosis, especially after hemorrhage or gut ischemia, have been reported to have a synergistic effect on SCh-induced hyperkalemia (31–34). Acidosis tends to drive K^+ from the intracellular to the extracellular fluid compartment leading to hyperkalemia.

In renal failure patients who have preoperative hyperkalemia, data on the safety of SCh are unclear. In four patients with preoperative hyperkalemia (5,6,8), there were no excessive increases in serum K^+ after SCh administration. However, given the small number of patients in these studies and the known risks of cardiac dysrhythmias associated with hyperkalemia, prudence would dictate that it be avoided under these circumstances. Because even small increases in a hyperkalemic patient may increase the risk of triggering dangerous cardiac dysrhythmias, some authors recommend medical treatment of serum K^+ levels above 6.0 mE/L preoperatively (35).

The only study (6) that addressed the effect of repeated doses of SCh administration in patients with renal failure did not show an excessive increase in serum K^+, but did show that 68% of the repeated injections resulted in sinus bradycardia—a finding consistent with the literature. Nevertheless, repeated doses of SCh in patients with renal failure are probably best avoided. If administration of repeated doses is contemplated, pretreatment with glycopyrrolate or atropine to protect against SCh-induced bradycardia should be strongly considered (13,15,16).

Pretreatment with a nondepolarizing neuromuscular relaxant or diazepam to minimize SCh-induced hyperkalemia may also be considered in the clinical setting. In patients with renal failure who may also have associated conditions that increase the risk of an exaggerated hyperkalemic response, however (e.g., burns, trauma, tissue ischemia, infections, and neuromuscular disorders including neuropathies), SCh should be avoided.

Conclusions

Case reports (31,36–40) of SCh-induced hyperkalemia and cardiac arrests in various disease settings continue to appear in the literature. However, we did not

find additional case reports and studies of SCh-induced hyperkalemia in patients with renal failure. This, and the clinical experience of investigators (5,8,41–43) who have safely used SCh in a large number of patients with renal failure corroborate the above conclusions that SCh, with the exceptions noted above, can be used safely in such patients.

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