Absence of adverse outcomes in hyperkalemic patients undergoing vascular access surgery

[Absence de complications chez des patients hyperkaliémiques devant subir une intervention chirurgicale d'accès vasculaire]

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Purpose: The decision to cancel vascular access surgery because of hyperkalemia requires knowledge of the risks *vs* benefits. This study sought to identify and characterize cases where surgery had been performed in patients with uncorrected hyperkalemia.

Methods: One thousand four hundred and seventy-two consecutive cases of vascular access surgery at an academic medical centre between 1995 and 2000 by a single surgeon were analyzed retrospectively.

Results: Eight cases had clear documentation that the case proceeded with hyperkalemia. Anesthesia techniques were one general anesthetic, one regional block, five monitored anesthesia care (MAC), and one local infiltration only. Mean potassium was 6.9 $mmol \cdot L^{-1}$ (range 6.1–8.0). In this series of selected asymptomatic hyperkalemic patients undergoing low risk surgery, no adverse results occurred.

Conclusion: While this review of eight cases (only one receiving general anesthesia) cannot be used to prove the safety of proceeding to surgery with uncorrected hyperkalemia, it does suggest that asymptomatic hyperkalemia may not be an absolute contraindication to vascular access surgery.

Objectif : *La décision d'annuler une opération d'accès vasculaire à cause de l'hyperkaliémie exige de connaître les risques et les avantages. La présente étude visait à identifier et à caractériser les cas où l'opération a été réalisée chez des patients présentant une hyperkaliémie non corrigée.*

Méthode : *Nous avons fait l'analyse rétrospective des opérations d'accès vasculaires réalisées chez 1 472 patients consécutifs au centre médical universitaire entre 1995 et 2000 par le même chirurgien.*

Résultats : *Dans huit cas, les données indiquaient clairement que l'opération avait eu lieu malgré l'hyperkaliémie. Les techniques anesthésiques étaient une anesthésie générale, une anesthésie régionale, cinq cas de surveillance anesthésique (SA) et une infiltration locale seulement. La moyenne du potassium était de 6,9 $mmol \cdot L^{-1}$ (intervalle de 6,1–8,0). Dans cette série de patients hyperkaliémiques asymptomatiques choisis, devant subir une intervention à faible risque, aucune complication n'est survenue.*

Conclusion : *Même si cette revue de huit cas, dont un seul cas d'anesthésie générale, ne prouve pas la sécurité d'une intervention chirurgicale en présence d'hyperkaliémie non corrigée, elle suggère que l'hyperkaliémie asymptomatique ne serait pas une contre-indication absolue à une opération à accès vasculaire.*

SIGNIFICANT preoperative hyperkalemia is considered a contraindication to anesthesia.[1–3] Retrospective studies have detected an association between hyperkalemia and increased mortality in hospitalized patients,[4–6] and surgical patients,[7–9] but reports of perioperative morbidity directly attributable to hyperkalemia are actually quite rare.[4,5,8–10]

Patients with renal failure scheduled for vascular access surgery present a dilemma. Long-term potassium (K) homeostasis is dependent on vascular access for dialysis, yet the surgery to construct this access may be delayed if the patient is hyperkalemic. The National Kidney Foundation recommends that a vascular access fistula or graft be placed well before the need for dialysis.[11] Because these accesses are superior to catheters,[11] delaying this type of surgery in renal failure patients because of hyperkalemia may be counterproductive. The decision to delay because of hyperkalemia requires some knowledge of the hazards associated with proceeding to surgery in the presence of hyperkalemia.

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While anecdotal experience suggests that surgery may be performed safely in some of these patients, there are no publications which address this. The purpose of this study was to retrospectively identify and characterize cases where surgery was performed in patients with uncorrected hyperkalemia. Eight such cases were identified where the K was greater than 6.0 mmol·L^{-1}. There were no adverse outcomes.

Methods

Following approval by the hospital Institutional Review Board, a list of all vascular access procedures for hemodialysis (1995–2000) by a single surgeon (who maintained a complete and detailed database on his surgical caseload) were matched with the preoperative K results recorded in the hospital laboratory database. The hospital charts of those cases where the preoperative K was greater than 6.0 mmol·L^{-1} were carefully reviewed for the following: confirmation that the case proceeded to surgery with hyperkalemia, clinical or electrocardiographic signs of hyperkalemia, management of hyperkalemia, and adverse outcomes. Adverse outcomes sought included intraoperative or postoperative (in-hospital) arrhythmias, chart notation of lethargy, weakness, myocardial infarction (MI), or death within one week. MI was defined as the documentation of symptoms compatible with MI, accompanied by biochemical or electrocardiogram (ECG) evidence of MI.

Both the paper and the electronic medical records were reviewed. The paper chart included nursing notes, progress notes, operative reports, consultation reports, hemodialysis notes, anesthesia record, ECG tracings, and laboratory reports. The electronic record included laboratory results, cardiology interpretation of ECGs, discharge summaries, surgical clinic notes and consultant clinic notes. All records for the period two weeks before to two weeks after surgery were examined.

At this institution, continuous 3 and 5 lead ECG monitoring occurs while the patient is in the operating room and postanesthesia care unit, but no records are retained unless there is an event of clinical interest. Similarly, postoperative care only includes ECG analysis if symptoms or signs indicate it. The absence of records of arrhythmia implies there were no arrhythmias that required management.

Results

Of the 1472 cases recorded in the surgeon's database, 1350 (92%) had a preoperative K concentration recorded in the hospital laboratory database on the day of surgery. Forty-five (3.3%) of these were greater than 6.0 mmol·L^{-1}. There was inadequate information in the medical record of 13 patients to confirm that the case proceeded to surgery with hyperkalemia uncorrected. The record showed no adverse outcomes in any of these excluded cases. Seventeen patients were treated with Kayexalate (sodium polystyrene sulfonate) or dialysis, following which surgery proceeded with normalized K. Seven patients were found to have normal K when repeated. Patients with hemolyzed specimens were excluded. Eight proceeded to surgery with K > 6.0 mmol·L^{-1} (Figure).

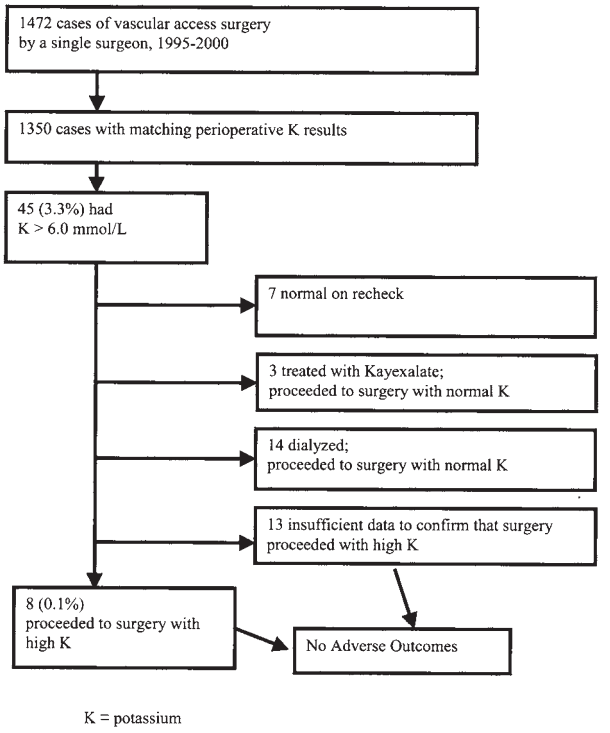

FIGURE Flow chart of record selection. K = potassium.

All of the recorded K measurements were taken the day of surgery. The times of sample collection and the time of start of anesthesia are shown in Table I. In five of the eight cases, a repeat potassium level was recorded.

These eight patients (four male, four female) had a mean age of 53 yr (range 35–75). Five patients were diabetics; two had Type 1 and three had Type 2 diabetes. The mean creatinine was 1035 μmol·L^{-1} (range 575–1644) and the mean K was 7.0 mmol·L^{-1} (range 6.1–8.0). There was no clinical or electrocardiographic evidence of hyperkalemia in any of these patients. Anesthesia technique included one general anesthetic

TABLE I Details of hyperkalemic patients

Case	*Age yrs*	*Gdr*	*Procedure*	*Anesthetic*	*Periop K mmol·L⁻¹*	*Time periop K*	*Time start of anesth*	*DM Type*	*Time of ECG*
1	75	f	revision UE graft, drainage of seroma	MAC 6.1	6.1 3:17	11:00	13:40	1	11:40
2	45	m	basilic vein transposition	6.2 regional block	8:15	9:30	2	09:41	
3	60	f	UE Gortex fistula	MAC	6.3 6.6	14:30 14:54	15:55	2	14:14
4	37	m	UE thrombectomy	MAC	6.9 7.0 6.7	21:20 21:27 21:40	21:40	none	21:25
5	37	m	IJ cath insertion	local	8.0	10:30	10:40	none	nr
6	54	f	saphenous vein to femoral artery graft	GA, sux induction	7.0 7.3	12:05 13:42	12:46¶	2	intraop
7	70	f	wrist fistula	MAC	7.3	12:55	14:04	none	nr
8	44	m	IJ cath insertion	MAC	7.5 7.1	13:02 15:00	12:52¶	1	15:00

Gdr = gender; m = male; f = female; Periop K = perioperative K; DM = diabetes mellitus; IJ cath = internal jugular catheter; K = potassium in mmol·L^{-1}; ECG = electrocardiogram; MAC = monitored anesthesia care; sux = succinylcholine; UE = upper extremity; GA = general anesthesia; nr = not recorded; intraop = intraoperative-exact time not specified; ¶ abnormal K alert came after start of case.

TABLE II Laboratory values of hyperkalemic patients

Case	*Creat mmol·L⁻¹*	*CO_2 mmol·L⁻¹*	*Glu mmol·L⁻¹*	*Ca mmol·L⁻¹*	*Na mmol·L⁻¹*	*Plt x 10^9·L⁻¹*	*WBC x 10^9·L⁻¹*
1	654	27	28.9	2.43	133	136	8.1
2	1149	18	5.8	1.75	136	198	5.4
3	813	27	10.5	2.30	135	212	5.3
4	1644	20	4.1	2.05	132	199	5.1
5	1520	22	4.2	2.55	137	319	7.7
6	999	22	7.1	2.13	141	211	9.1
7	575	19	4.1	N-A	133	213	8.1
8	928	19	20.0	1.80	141	nr	nr

Creat = creatinine; CO_2 = bicarbonate; Glu = glucose; Ca = calcium; Na = sodium; Plt = platelet count; WBC = white blood cell count; nr = not recorded.

(with succinylcholine for tracheal intubation), five monitored anesthesia care with local anesthetic infiltration of the surgical site, one local anesthetic, and one regional anesthetic (Table I). Two of the procedures were internal jugular catheter insertions only.

Of the conditions known to affect potassium or to exacerbate hyperkalemia (Table II), only hyperglycemia was present (two cases). This was treated with *iv* insulin in both cases. ECG tracings performed within two hours of surgery were available in the record of six of the eight patients and there were no changes suggestive of hyperkalemia. While the perioperative ECG in the other two patients was interpreted as normal, the records did not confirm that it was done within hours of surgery.

Examination of the record for evidence of lethargy, weakness, or symptoms of MI revealed documentation of lethargy in only one case. This was attributed to more global causes. K was normal at the time of the observation.

Discussion

In this retrospective investigation, we report a series of eight patients with documented hyperkalemia preceding vascular access surgery with no adverse events perioperatively. Hyperkalemia is the most frequent perioperative complication in renal failure patients.[10] It is often asymptomatic, but it may also lead to weakness, paresthesias, and fatal cardiac irritability.

The reported incidence of hyperkalemia in hospitalized patients ranges from 1.1% to 1.4%.[5,10] Mortality attributable to hyperkalemia in hospitalized patients is 0% to 1.7%,[4,6,9] with the most recent studies reporting the lower values. The level at which

hyperkalemia should be treated is controversial. Newmark and Dluhy[12] advocate immediate treatment when K > 6.5 mmol·L^{-1}. Levinsky[13] defined K < 6.5 mmol·L^{-1} as mild and not requiring emergency treatment. Major anesthesiology textbooks describe thresholds for postponing surgery varying from 5.5 mmol·L^{-1} [1] to 5.9 mmol·L^{-1}.[2] Clinical signs and symptoms do not correlate predictably with K serum concentrations, and there is little evidence on which to select such a threshold.

Serum K test results may be factitiously elevated, when the actual serum concentration or the transmembrane potential is normal. This can occur with fist clenching[14,15] prolonged use of a tourniquet,[3] leukocytosis, or thrombocytopenia.[14]

True hyperkalemia is caused by increased K intake or release, decreased K elimination, or inappropriate shifting of K from intracellular to extracellular compartments. It may be that more than one factor must be present before hyperkalemia becomes symptomatic.[16,17]

Hyperkalemia that develops just before surgery is likely due to inadequate extra-renal shifting by insulin and catecholamines.[15] This insulin shifting is reduced in diabetes and fasting.[16,17,18–20]

Catecholamines may rise with perioperative stress.[3] The α-adrenergic effects, which predominate in renal failure,[17,18] raise intravascular K, while ß$_2$ adrenergic stimulation reduces K.[17,18,19] Thus, selective ß$_1$ agents are preferable if ß-blockade is needed.[17,21] No ß-adrenergic blockers were used in these patients. Succinylcholine may induce a detrimental shift of K, causing hyperkalemia,[22] although this is not always the case.[23] There were no adverse effects in the one case of succinylcholine use in this series.

Chronic hyperkalemia may be better tolerated than an acute increase.[17,18,24] However, the clinical applicability of this is unproven. In a frequently quoted study, Surawicz showed, in dogs, a greater tolerance of hyperkalemia when K was infused slowly.[24] However the "slow" infusions were administered over four to 25 min and the "fast" infusions over three to 12 sec. It is unlikely that hyperkalemia develops clinically as quickly as this "slow" infusion, let alone the "fast" one. While sodium-K pump activity is decreased in chronic renal failure, thus decreasing membrane excitability,[25] this activity returns to normal after several weeks of dialysis.[22] It is likely that these reported cases were acute-on-chronic events, but there were insufficient data to verify this. No conclusions on the tolerability of chronic hyperkalemia can be made.

There were no ECG changes noted, and while the ECG is usually a sensitive monitor of the cardiac effects of hyperkalemia,[25,26] changes are not routinely evident even at high K levels. Tarail reported inconsistent ECG changes until K > 7.9 mmol·L^{-1},[27] Szerlip reported two cases of K > 9.0 mmol·L^{-1} with no ECG changes,[28] and Wrenn showed that the sensitivity of the ECG to detect K > 6.5 mmol·L^{-1} was 62% or less.[29]

In two cases of this study, an actual tracing with the time of the ECG was not present in the chart. While regular ECG monitoring is part of routine care at this institution, tracings are generally not included in the medical record unless there was an abnormality. A prospective study would be necessary to reliably confirm normal results.

Although the attending anesthesiologist for each case was able to confirm the conclusions drawn, (these were all memorable cases) the discovery of cases and adverse effects, and confirmation of details depended on retrospective analysis of chart data. Silent MI or asymptomatic arrhythmias might not have been detected. We did not examine cases that were cancelled because of hyperkalemia, and so no comparison of outcomes can be made. All procedures were minimally invasive, and all but one was done without general anesthesia.

If other reports of hyperkalemia in anesthetic practice corroborate the low morbidity shown with this approach, a prospective, observational study would be indicated to clarify predisposing factors for morbidity. This would then allow a randomized study to be safely designed.

While we await further publications in this area, the following distillation of expert opinion is suggested. Discovery of preoperative hyperkalemia should lead to confirmation with a repeat test, with attention to causes of factitious hyperkalemia. Elective surgery should ideally be postponed. However, if the clinical situation favours proceeding, this can probably be done safely if the patient is asymptomatic and the surgery presents little risk of tissue damage, hypoxemia, or acidosis. The 12 lead ECG must be normal and the patient must not be acidotic, hypocalcemic, hyponatremic, or severely fasted. If ß-blockade is needed, a cardioselective agent should be used. Management of hyperkalemia and any hyperglycemia should be initiated, and preoperative anxiety addressed.

In summary, we report eight cases of vascular access surgery proceeding with a K concentration between 6.1 and 8.0 mmol·L^{-1}. None had factors known to increase the morbidity of hyperkalemia. None of these patients displayed clinical or electrocardiographic signs of hyperkalemia, and in no case was there any adverse effect. While this review of eight cases (only one receiving general anesthesia) cannot be used to

prove the safety of proceeding to surgery with uncorrected hyperkalemia, it does suggest that asymptomatic hyperkalemia may not be an absolute contraindication to vascular access surgery.

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