Postoperative goal-directed therapy and development of acute kidney injury following major elective noncardiac surgery: post-hoc analysis of POM-O randomised controlled trial.

Amour Patel, John R. Prowle, Gareth L. Ackland; POM-O Study Investigators.

"Take-home message"

Acute kidney injury following major surgery is associated with early intraoperative cardiometabolic derangement, characterized by need for pressor support (norepinephrine) and higher lactate. Postoperative goal-directed hemodynamic therapy aimed at optimising oxygen delivery fails to prevent AKI.

Tweet: Intraoperative need for norepinephrine associated with development of postop AKI; not reversed by postop optimization of oxygen delivery.

Abstract

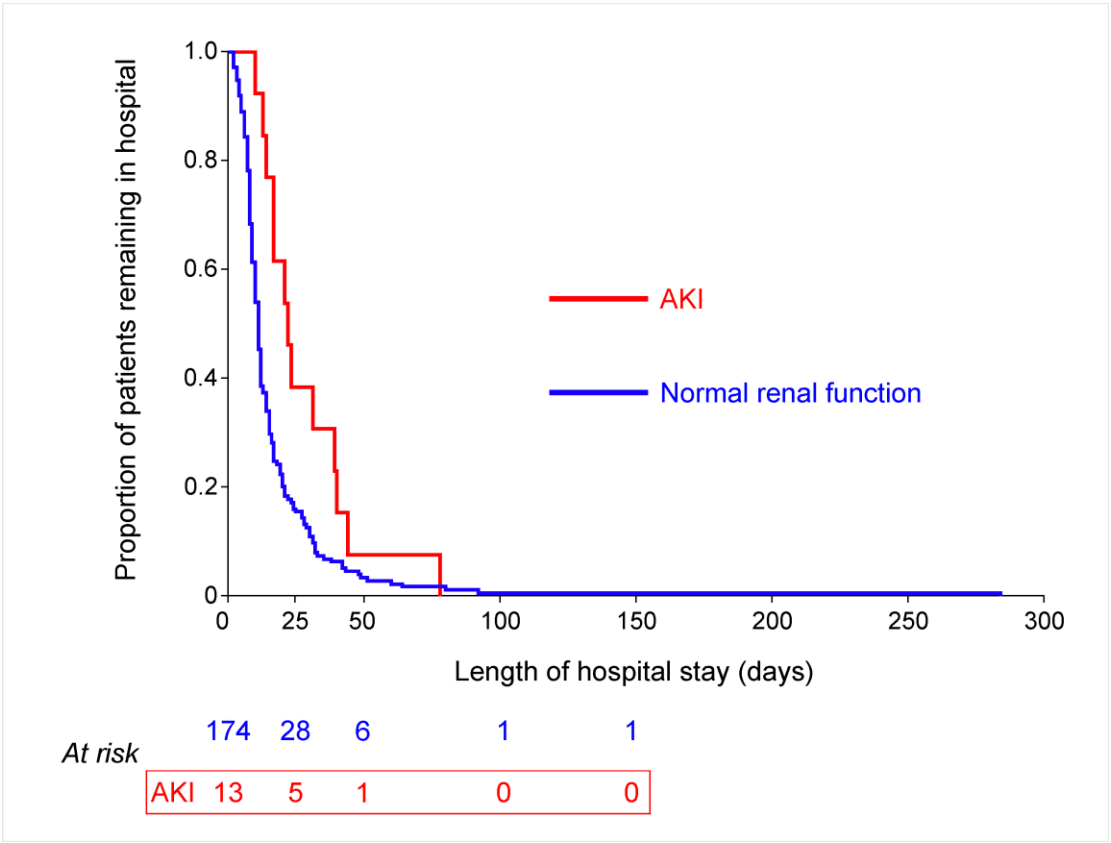
Purpose: Maintenance of adequate cardiac output is associated with protection against perioperative acute kidney injury (AKI), but clinical evidence for a specific hemodynamic strategy is lacking. In this multi-centre blinded, randomized controlled trial, we studied whether postoperative maintenance of oxygen delivery reduced AKI following major noncardiac surgery.

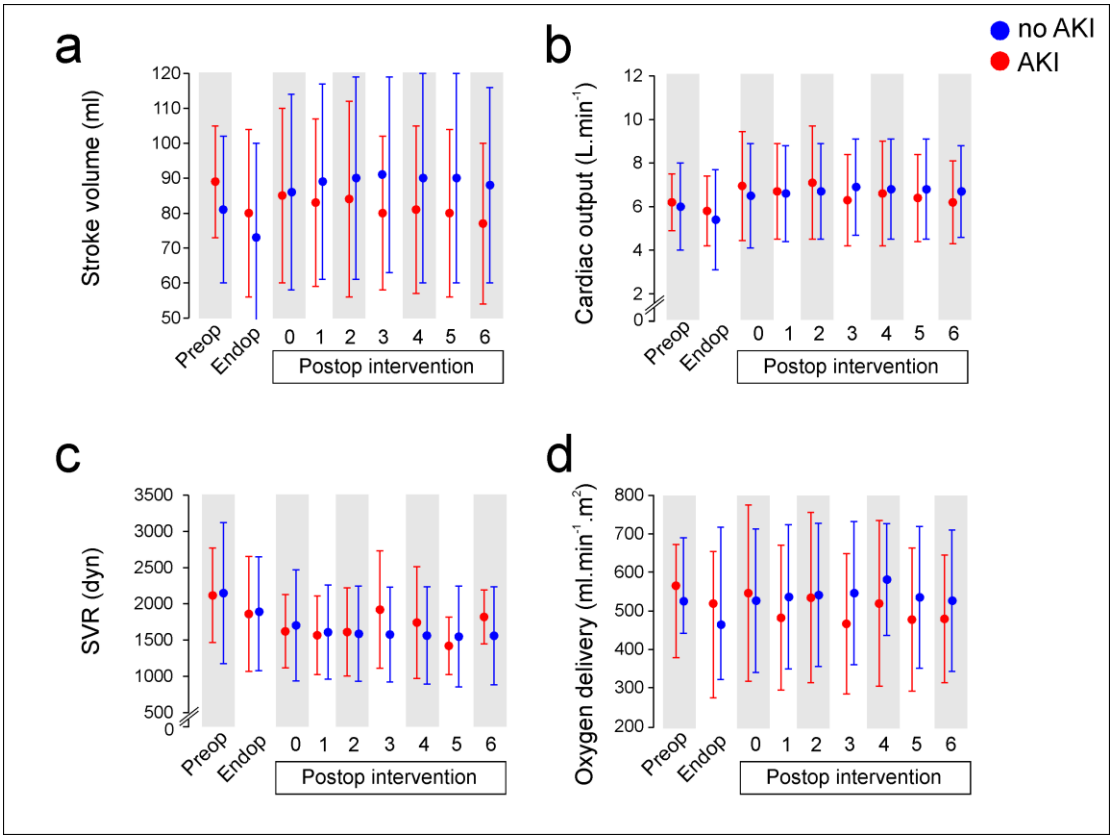
Methods: The primary end point for this post-hoc analysis was the development of AKI within 48h postoperatively, as defined by Acute Kidney Injury Network criteria. One hundred and eighty-seven patients who underwent major surgery were randomly assigned immediately postoperatively to receive either fluid and/or dobutamine therapy to maintain/restore their preoperative oxygen delivery, or protocolized therapy (where oxygen delivery was measured only).

Results: AKI occurred within 48h in 13/187 patients (7.0%). AKI was not related to cardiac output (mean difference: 0.4L.min^{-1} (95%CI:-1.7 to 0.9); p=0.77), hemodynamic intervention (relative risk: 1.21 (95%CI: 0.42-3.45);p=0.77), operation type or chronic kidney disease. Prior to the postoperative intervention, AKI patients were more likely to be administered vasopressors (relative risk: 2.4 (95%CI: 1.2-4.7); p=0.02), despite similar volumes of intraoperative fluid being administered. AKI was associated with persistently higher lactate during the intervention period (mean difference: 1.15mmol.L^{-1} (95%CI: 0.48-1.81); p=0.01). Prolonged hospital stay was associated with early AKI (hazard ratio: 1.91 (95%CI: 1.23-2.94); p=0.02).

Conclusion: Early tissue dysoxia is associated with AKI following noncardiac surgery. Our data confirm that hemodynamic manipulation does not prevent AKI once systemic inflammation is established, as suggested by recent trials in sepsis.

Figures


Proportion of patients remaining in hospital
1.0
0.8
0.6
0.4
0.2
0
AKI
Normal renal function
0
25
50
100
150
200
250
300
Length of hospital stay (days)
At risk
174
28
6
1
1
AKI
13
5
1
0
0


a
Stroke volume (ml)
120
110
100
90
80
70
60
50
Preop
Endop
0
1
2
3
4
5
6
Postop intervention
b
Cardiac output (L.min-1)
12
10
8
6
4
2
0
no AKI
AKI
c
SVR (dyn)
3500
3000
2500
2000
1500
1000
500
0
d
Oxygen delivery (ml.min-1.m-2)
800
700
600
500
400
300
200