Incidence of Acute Kidney Injury (AKI) after Endovascular Abdominal Aortic Aneurysm Repair (EVAR) and Impact on Outcome

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WHAT THIS PAPER ADDS
This prospective analysis reports rates of acute kidney injury (AKI) after elective endovascular aneurysm repair, using consistent contemporary definitions. It suggests that the rate of AKI is high and is associated with medium-term outcome after this procedure. As a result, pre-operative planning and peri-operative care should ensure that appropriate preventive strategies are applied.

Background: Acute kidney injury (AKI) is an important post-operative complication that may impact on mortality, morbidity, and cost. The incidence after endovascular aneurysm repair (EVAR) remains unknown, as the current literature has not employed consistent definitions. The aim of this study is to assess the incidence of AKI after elective EVAR and examine the impact of AKI on mortality and cardiovascular morbidity using the current universally accepted definitions.

Methods: This was a cohort study using prospectively collected data, including consecutive patients undergoing elective EVAR for an infrarenal abdominal aortic aneurysm (AAA). Those with end stage renal failure were excluded. The primary endpoint was incidence of AKI as per the “Acute Kidney Injury Network” (AKIN), and “Kidney Disease Improving Global Outcomes” (KDIGO) criteria. Secondary endpoints included AKI stage, drop in estimated glomerular filtration rate (eGFR), and mortality and cardiovascular morbidity.

Results: 149 patients were included (16 females, 11%; mean age: 69 ± 8 years; mean AAA diameter: 6.0 ± 1.1 cm), 28 (18.8%) of whom developed AKI (26 patients classified as stage 1 and 2 as stage 2). Within 48 hours, those with AKI dropped their eGFR from 61 ± 20 mL/kg/1.73 m² to 51 ± 20 units (p < .001), and those without from 75 ± 9 to 74 ± 10 units (p < .001). None required dialysis during a 33 ± 11 month follow up. Development of AKI was associated with mortality (HR 0.035, 95% CI: 0.005 to 0.240, p < .001) and cardiovascular morbidity (HR: 0.021, 95% CI: 0.004 to 0.11, p < .001) on adjusted regression analysis.

Conclusions: The incidence of AKI after EVAR is significant and is independently associated with medium-term mortality and morbidity.

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INTRODUCTION
Abdominal aortic aneurysm (AAA) constitutes a serious health problem; current prevalence for men above the age of 65 ranges from 2% to 7%. Endovascular abdominal aortic aneurysm repair (EVAR) is now a first line treatment, as early and medium-term outcomes have proven similar or superior to open repair. However, those undergoing EVAR are at risk of developing acute kidney injury (AKI) for several reasons. Acute kidney injury after any type of surgical or radiological intervention is independently associated with higher morbidity, prolonged length of hospital stay, cost, and short-term mortality (even as high as 20%). Long-term survival is also directly affected by AKI in patients undergoing vascular operations. The incidence of AKI after elective EVAR is practically unknown as a uniform widely accepted definition of AKI has never been used consistently in the current literature. Most investigators have not included post-operative urine output in defining AKI. Instead, serum creatinine (SCr) alone has been used as a marker of immediate post-operative renal dysfunction (defined as a rise of more than 25% or 50%) and then reported as “AKI incidence.” The current definitions of AKI require precise urine output measurements for 48 hours. Several other inconsistent reporting criteria, apart from SCr levels, have also been applied in the EVAR...
literature, such as creatinine clearance or estimated glomerular filtration rate (eGFR). As a result, the aim of this cohort study was to assess the prevalence of AKI using the currently accepted criteria (“Acute Kidney Injury Network” and “Kidney Disease Improving Global Outcomes” after elective EVAR and assess its impact on medium-term mortality and cardiovascular morbidity.

**METHODS**

**Study design and population**

This is a cohort study including patients undergoing elective EVAR of an infrarenal abdominal aortic aneurysm (AAA) between September 2010 and September 2013 in a tertiary referral centre for aneurysm disease. Patients were eligible for repair if they had an AAA diameter >5.5 cm or an AAA <5.5 cm with a rapidly increasing sac (>1 cm per year). Endovascular repair was offered as a first line procedure. Data for patients undergoing EVAR during the aforementioned period were entered prospectively in an electronic database, aiming to assess predictors of outcome after elective EVAR. The original cohort consisted of 249 patients. Patients were included in the present analysis if they had serum creatinine (SCr) and hourly urine output measurements for at least 48 hours and all relevant information available, and were able to provide written informed consent; urine output measurements were not available for 72 patients because of missing documentation and an SCr was not available for 41 patients at 48 hours, hence the final cohort consisted of 149 patients. Patients with symptomatic, leaking, ruptured, infected, or inflammatory aneurysms and patients with end-stage renal disease (ESRD) receiving renal replacement therapy (at baseline) were excluded.

**Study protocol**

Demographics, comorbidities and anatomical data were recorded and stored electronically at baseline. All participants underwent a computed tomographic angiography (CTA) with 3 dimensional reconstruction before EVAR to assess aortic anatomy. Blood samples at baseline were obtained prior to any imaging requiring the administration of contrast. Further blood samples were taken at 24 and 48 hours after the repair for every patient. A standard follow up protocol, including laboratory checks at 30 days, 6 months, and 12 months after the operation, and annually thereafter, was employed. Imaging during follow up included plain abdominal radiography and a CTA at 6 months, 12 months, and annually thereafter, which was the standard follow up protocol for EVAR at the time. Since July 2013 patients have undergone follow up with ultrasound imaging at the same intervals.

**Endovascular repair procedures**

The Anaconda (Vascutek, UK) endograft was deployed in all cases, adhering to the manufacturer’s instructions for use. Indications and specifications have been described elsewhere. The specific device does not employ suprarenal fixation (such as a bare stent or any other modality that is deployed above the orifice of the renal arteries). All EVARs were performed in an operating theatre under general anaesthesia. Iopromide was used as contrast-medium (Ultravist 300, Bayer, Berlin, Germany). Prior to EVAR, the administration of any contrast for at least 2 weeks, and non-steroidal anti-inflammatory drugs (NSAIDs) for at least 1 week, were avoided in all cases. Metformin was discontinued for 2 days, where applicable. For patients with a pre-operative eGFR >60 mL/min/1.73 m², intravenous fluids (0.9% saline, 2 mL/kg/hour) were started on the day of the operation. Patients with an eGFR <60 units were admitted 1 day before and received intravenous fluids (0.9% saline, 1.5 L/24 hours) for 24 hours, until nil by mouth, when they were commenced on 0.9% saline at 2 mL/kg/hour. Urinary catheterization and hourly urine output measurements were routinely employed and patients remained catheterized until ambulatory. Intra-operative fluid management was guided by mean arterial pressure, recorded via a peripheral arterial line. The aim of fluid therapy (consisting only of crystalloid solutions) was to keep the mean arterial pressure within 80% of the baseline (before induction) for 90% of the operating time. Urine output measurements continued until at least 48 hours after EVAR, or until discharge. In accordance with the authors’ standard protocol for elective EVAR, aspirin and clopidogrel were administered on the day of the procedure. Aspirin was discontinued on the 30th day, whereas clopidogrel was continued as a life-long treatment. Patients were asked to mobilise and eat and drink, as tolerated, as soon as possible after the repair and were usually discharged on day 2. In case the patient developed AKI over the initial 48 hours after EVAR, they were subsequently reviewed by a nephrologist and further treatment was decided based on that consultation. A blood transfusion (packed red blood cells) was given if a patient’s haemoglobin (Hb) was less than 8 g/dL or if the patient had a history of cardiac disease and was symptomatic with a Hb of less than 10 g/dL.

**Definitions and study endpoints**

In order to define AKI incidence and classify the different stages of AKI, SCr and Urine Output (UO) measurements were taken into account, within the space of 48 hours after the completion of the procedure. The following criteria were applied: Acute Kidney Injury Network (AKIN), and Kidney Disease Improving Global Outcomes (KDIGO).

The diagnosis of AKI was defined as an absolute increase in SCr of more than or equal to 0.3 mg/dL (≥26.4 µmol/L), or a percentage increase in SCr of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output to less than 0.5 mL/kg per hour for more than 6 hours — all within the space of 48 hours after the completion of the procedure. This represents the patient meeting the minimum criteria for “stage 1” AKI as per the AKIN and KDIGO definitions, and is in line with the current National Institute of Health and Care Excellence (NICE) definition for diagnosing AKI.
For those who developed AKI using this definition (within the 48 hour window), further serial SCr measurements were obtained for a period of at least 7 days (1 week) after the completion of the procedure or until necessary. Patients were then classified into three different stages of AKI, according to AKIN and KDIGO criteria staging. Stage 2 AKI was defined as 100–199% SCr rise within 7 days or a urine output <0.5 mL/kg/h for >12 hours, and Stage 3 AKI was defined as ≥200% SCr rise within 7 days or rise to >354 μmol/L with an acute rise >44 μmol/L or a urine output <0.3 mL/kg/h for >24 hours or anuria for >12 hours.

The primary study endpoint was incidence of AKI. The secondary study endpoints included: stage of AKI as per AKIN and KDIGO, SCr and eGFR (CKD-EPI formula) levels at 24 and 48 hours compared with baseline, as well as SCr and eGFR levels at 7 days compared with baseline for those who developed AKI. Additionally, a multivariate analysis (adjusted for age, sex, AAA size, and established pre-operative cardiovascular risk-factors) was performed to evaluate the effect of AKI on mortality and cardiovascular events during follow up. To determine the latter, a composite “cardiovascular” secondary endpoint was calculated by adding the following: death, non-fatal myocardial infarction, non-fatal stroke, non-fatal peripheral vascular complication. All complications were defined according to the reporting standards for EVAR by Chaikof et al.24 Hypercholesterolaemia was defined as baseline total cholesterol levels of >5 mmol/L. Hypertension was defined as patient taking antihypertensive medication at recruitment or blood pressure exceeding 140/90 mmHg at baseline.

Statistical analysis

Analyses were performed using the Statistical Package for Social Sciences Version 21.0 (SPSS, Chicago, IL, USA). Continuous parametric data are presented as mean ± standard deviation (SD) and categorical data are presented as absolute values and percentages. Comparisons between the study groups were performed using the independent or paired (where applicable) samples t test for continuous parametric variables and Pearson’s chi-square test for categorical variables. A multivariate analysis was performed using binary logistic regression to assess the effect of important risk factors at baseline on AKI incidence. Additionally, Cox regression was employed to assess the impact of AKI on mortality and cardiovascular morbidity during follow up (using the composite endpoint), together with factors (at baseline) where statistical comparison disclosed a p value <.1. Kaplan-Meier analysis and the log-rank test were used to compare survival between those with and without AKI during follow up. A p value level <.05 was considered statistically significant.

RESULTS

Operative outcomes

149 patients were included (16 females, 11%; mean age: 69 ± 8 years; mean AAA diameter: 6.0 ± 1.1 cm), undergoing elective EVAR for an infra-renal AAA. None of the procedures were converted to open repair and all aneurysms were successfully excluded on the completion angiograms with no evidence of immediate endoleak. None of the main renal arteries were covered by the endografts; a total of eight accessory renal arteries were covered (2 of these patients then developed AKI, p = .65). None of the patients had excessive calcification, thrombus, or severe angulation at the level of the proximal neck25; none of the patients had a neck length of <12 mm. The mean volume of contrast used was 121 ± 15 mL. This did not differ significantly between those who did and did not develop AKI (120 ± 15 mL vs. 121 ± 14 mL, p = .52). Only two patients received a blood transfusion (packed red blood cells) within 7 days of the repair, one of whom then developed AKI. None of the patients developed severe bleeding, hypovolaemic shock, or required inotropic support at any point during the first post-operative week.

Primary endpoint

A total of 28 patients (18.8%) developed AKI. Table 1 summarizes the main characteristics for patients with and without AKI.

Secondary endpoints and events during follow up

Table 2 summarizes SCr and eGFR levels (renal outcomes) at baseline, 24 hours, 48 hours, and 7 days after the completion of the procedure. Overall, of those 28 patients who developed AKI, 25 patients (89%) were classified as stage 1 as per both the AKIN and KDIGO criteria and three (11%) were staged as stage 2 as per both the AKIN and KDIGO criteria; none were clustered as stage 3. None of the patients required renal dialysis after the procedure (throughout follow up). Mean SCr increased from

Table 1. Baseline characteristics for those with and without acute kidney injury (AKI).

<table>
<thead>
<tr>
<th>Variable</th>
<th>AKI</th>
<th>No AKI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>28 (18.8%)</td>
<td>121 (81.2%)</td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>72 ± 8</td>
<td>69 ± 8</td>
<td>.7</td>
</tr>
<tr>
<td>Female sex</td>
<td>4 (14%)</td>
<td>12 (10%)</td>
<td>.5</td>
</tr>
<tr>
<td>Smoking</td>
<td>21 (75%)</td>
<td>103 (85%)</td>
<td>.3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>19 (68%)</td>
<td>95 (79%)</td>
<td>.2</td>
</tr>
<tr>
<td>Cholesterolisma</td>
<td>16 (57%)</td>
<td>40 (33%)</td>
<td>.02</td>
</tr>
<tr>
<td>Stroke</td>
<td>1 (4%)</td>
<td>8 (7%)</td>
<td>1.0</td>
</tr>
<tr>
<td>MI</td>
<td>2 (7%)</td>
<td>9 (7%)</td>
<td>1.0</td>
</tr>
<tr>
<td>PAD</td>
<td>9 (32%)</td>
<td>20 (17%)</td>
<td>.07</td>
</tr>
<tr>
<td>Diabetes</td>
<td>8 (29%)</td>
<td>17 (14%)</td>
<td>.09</td>
</tr>
<tr>
<td>Neck diameter, cm</td>
<td>2.2 ± 0.2</td>
<td>2.1 ± 0.3</td>
<td>.7</td>
</tr>
<tr>
<td>Statin use</td>
<td>20 (71%)</td>
<td>70 (58%)</td>
<td>.2</td>
</tr>
<tr>
<td>Antiplatelet therapy</td>
<td>28 (100%)</td>
<td>121 (100%)</td>
<td>1.0</td>
</tr>
<tr>
<td>SCr, μmol/L</td>
<td>107 ± 35</td>
<td>79 ± 14</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>eGFR, mL/kg/1.73 m²</td>
<td>61 ± 20</td>
<td>75 ± 9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hb, g/dL</td>
<td>13.3 ± 2</td>
<td>13.2 ± 1.6</td>
<td>.2</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>30 ± 5</td>
<td>27 ± 4</td>
<td>.2</td>
</tr>
</tbody>
</table>

BMI = body mass index; eGFR = estimated glomerular filtration rate; Hb = haemoglobin; MI = myocardial infarction; PAD = peripheral arterial disease; SCr = serum creatinine.
107 ± 35 μmol/L to 166 ± 53 μmol/L and 129 ± 43 μmol/L for those who developed AKI within 24 and 48 hours, respectively (p < .001 in both cases). Mean eGFR (CKD-EPI formula) decreased from 61 ± 20 mL/min/1.73 m² to 39 ± 16 units and then 52 ± 20 units for those who developed AKI within 24 and 48 hours, respectively (p < .001 in both cases). Twelve patients (8%) had a drop in urine output below 0.5 mL/kg/hour for more than 6 hours over the initial 2 first post-operative days. On multivariate analysis, only hypercholesterolaemia at baseline was associated with AKI development (Table 3).

Mean follow up was 33 ± 11 months. A total of 11 patients (7.4%) died during follow up and a total of 20 patients (13.4%) developed complications as per the cardiovascular composite endpoint. Interestingly, nine (32.1%) of the patients who developed AKI died during follow up, and another 18 (64.3%) developed cardiovascular complications. Those who developed AKI were more likely to die (32.1% vs. 1.7%, p < .001) or develop cardiovascular morbidity (as per the “composite endpoint” 64.3% vs. 1.7%, p < .001) during follow up on univariate analysis; AKI was also independently associated with death and cardiovascular morbidity on multivariate survival analysis (Tables 4 and 5, Fig. 1). A total of eight type 2 endoleaks (5.3%) and two type 1 endoleaks (1.3%) occurred during follow up — all type 1 endoleaks occurred in patients who did not develop AKI.

Table 2. Renal outcomes for groups with and without acute kidney injury (AKI).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>1 day</th>
<th>2 days</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>AKI SCr, μmol/L</td>
<td>107 ± 36</td>
<td>166 ± 53</td>
<td>130 ± 43</td>
<td>117 ± 39</td>
</tr>
<tr>
<td>eGFR, mL/kg/1.73 m²</td>
<td>61 ± 20</td>
<td>39 ± 16</td>
<td>51 ± 20</td>
<td>58 ± 21</td>
</tr>
<tr>
<td>No AKI SCr, μmol/L</td>
<td>80 ± 14</td>
<td>82 ± 15</td>
<td>86 ± 16</td>
<td>80 ± 16</td>
</tr>
<tr>
<td>eGFR, mL/kg/1.73 m²</td>
<td>75 ± 9</td>
<td>75 ± 10</td>
<td>74 ± 11</td>
<td>75 ± 10</td>
</tr>
</tbody>
</table>

eGFR = estimated glomerular filtration rate (CKD-EPI formula); SCr = serum creatinine.

DISCUSSION

This cohort study based on prospectively collected data suggests that AKI after elective EVAR may be more common than suggested by previous data, with an incidence of...
18.8%. Acute kidney injury constitutes an important peri-operative complication, after any intervention, for a variety of reasons: impact on short and long-term mortality and cardiovascular morbidity, longer hospital stay, increased cost, and impact on long-term renal function.\textsuperscript{13–17} Previous data suggested an incidence of 3–19\% for elective EVARs — the main reason for the wide variation is that a uniform widely accepted definition of AKI has not been used consistently in the current literature.\textsuperscript{13–17} Most investigators have not even included post-operative urine output in defining AKI. Serum creatinine (SCr) has mostly been used as a marker of immediate post-operative renal dysfunction (a rise of 25\% or 50\%) and then been reported as “AKI incidence.”

Overall, immediate post-operative renal injury has a lower incidence in EVAR compared with open repair in the majority of series, using the aforementioned inconsistent criteria.\textsuperscript{15} However, three studies have documented a similar or more pronounced incidence following EVAR — two of these defined AKI as a rise in SCr of more than 50\% compared with baseline and one as >30\% compared with baseline. The Dutch Randomized Endovascular Aneurysm Management (DREAM) trial showed that peri-operative changes in SCr were similar with no statistical differences in the incidence of AKI (as per their definition) or need for dialysis.\textsuperscript{26} Another prospective, non-randomized study, evaluating 485 patients undergoing EVAR or open repair,\textsuperscript{27} disclosed a significant increase in SCr and a drop in creatinine clearance for EVAR (from 1.0 [0.9–1.3] mg/dL to 1.08 [0.9–1.36] mg/dL, and from 67.6 [51.3–85.10] mL/min to 66.7 [49.9–81.4] mL/min), but not for open repair. An analysis of the National Surgical Quality Improvement Program in the USA, including 6,516 patients (retrospective data), also showed that immediate post-operative renal injury (defined as >30\% increase in SCr concentration) in EVAR and open repair have similar occurrence rates.\textsuperscript{28} A more recent update of this analysis including 11,753 patients showed that “acute renal failure” is less common in EVAR (0.4\% vs. 2.7\%, \(p < .001\)), but it is unclear how “renal failure” was defined.\textsuperscript{29}

The mechanisms that may lead to renal dysfunction after EVAR include: contrast administration (contrast induced nephropathy or CIN\textsuperscript{30} - contrast administration leads to: increased vasoconstrictive forces, decreased local prostaglandin and nitric oxide mediated vasodilatation, a direct toxic effect on renal tubular cells by oxygen free radicals, increased oxygen consumption, increased intra-tubular pressure secondary to contrast induced diuresis, increased urinary viscosity, and tubular obstruction), renal micro-embolization during device deployment,\textsuperscript{31} complications directly relating to the renal arteries, such as dissection or coverage of the arterial orifice,\textsuperscript{32} lower limb ischaemia and subsequent ischaemia reperfusion syndrome,\textsuperscript{33} hypovolaemia, the presence of an inflammatory infiltrate (the actual aeurysm sac that is not excised such as in open aeurysm repair),\textsuperscript{34} and various pre-morbid cardiovascular risk factors.\textsuperscript{35}

The RIFLE criteria were the first universally accepted criteria for AKI in 2002.\textsuperscript{36} However, more recently, the Acute Kidney Injury Network (AKIN),\textsuperscript{22} Kidney Disease Improving Global Outcomes (KDIGO),\textsuperscript{19,20} and National Institute for Health and Care Excellence (NICE)\textsuperscript{17} criteria have been introduced. In this study AKI was defined using the AKIN and KDIGO definitions to reveal an incidence of 18.8\%, which makes AKI a common complication after EVAR. Interestingly, the main predictors of AKI development were eGFR levels and hypercholesterolaemia. Contrast load was not directly associated with AKI, which means that contrast induced renal injury is not the only underlying mechanism. Also, none of the patients were found to have microemboli into the renal parenchyma on the follow up CT scans. This may be because none of the patients had a device with suprarenal fixation. Suprarenal fixation can lead to deterioration of renal function over the longer term after EVAR, based on previous data\textsuperscript{38}; however, sufficient data are not available currently regarding renal function in the immediate post-operative period after implantation of a device with suprarenal fixation.

One of the most important findings of the study is that AKI is more prevalent among those with impaired renal function before EVAR (baseline). Those who did go on to develop AKI in this series had a significantly lower eGFR at baseline (61 ± 20 vs. 75 ± 9 units, \(p < 0.001\), Table 1), even though on multivariate analysis, eGFR or SCr levels were not associated with the incidence of AKI. AKI development was also associated with mortality and cardiovascular outcome over a mean follow up of 33 months. It has been shown previously that baseline eGFR levels are directly associated with post-operative outcome after EVAR.\textsuperscript{39} Overall, this study cannot specify whether AKI per se is the main determinant of less favourable outcome in this subset of the population undergoing EVAR. Indeed the patients who did develop AKI were more likely to develop morbidity, but their baseline renal function was also decreased in comparison. In any case, it is important to optimize the patient’s renal function in the peri-operative period, especially those with reduced renal function at baseline. Unfortunately, there are a paucity of data regarding the prevention of AKI in EVAR.\textsuperscript{40} Two significantly underpowered randomized pilot studies have reported no benefit from N-acetylcysteine\textsuperscript{41} or hydration with sodium bicarbonate.\textsuperscript{42} Targeted renal therapy\textsuperscript{43} and regional anaesthesia\textsuperscript{44} may be of benefit, but both have drawbacks and have not been widely adopted. Aggressive hydration therefore remains the most important intervention to prevent AKI in EVAR. Further studies are urgently required to assess further methods as well as the optimum way of providing hydration to this population.

The impact of AKI on longer term renal function is another important point. In this series, those with AKI dropped their eGFR by a mean of 3 units (Table 2), whereas patients without AKI had a stable eGFR 6 months after the repair. It has been shown previously that EVAR may be associated with a significant drop in eGFR, up to 6 units per annum for the first post-operative year.\textsuperscript{28} This reduction in renal function slows down after the first year. As a result, alleviating the peri-operative insult will improve longer-term function overall as well.\textsuperscript{25} This study is limited by...
the fairly small number of patients included and the fact
that even though it is based on prospectively collected data,
it represents a retrospective analysis. However, it has
employed consistent and widely accepted AKI definitions
to assess the incidence of the pathology after EVAR in a sys-
tematic manner. It is also not confounded by the use of
suprarenal graft fixation in part of the population under
investigation; previous studies have combined the two
modes of fixation in their analyses, which can skew find-
ings. The study is also limited by the urine output mea-
surements, after removal of the urinary catheter, not being
adequately documented in 100 patients, who were subse-
quently excluded. This may have introduced selection bias
at baseline.

Acute kidney injury after EVAR is common and is asso-
ciated with increased mortality and morbidity. Further
studies are urgently required to assess the impact of AKI on
longer term results and examine preventive strategies.

CONFLICT OF INTEREST
None.

FUNDING
None.

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