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Review

The analgesic action of desmopressin in renal colic

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Urolithiasis is a frequent problem causing a significant clinical, psychological and socio-economic burden. Analgesia remains the most important element in the medical treatment of renal colic. Nonetheless, both NSAIDs and opiates have a side effect profile which can cause further complications. As such, the use of desmopressin for renal colic has received increased attention in the last two decades. This paper provides an overview of current evidence on the use of desmopressin as an analgesic strategy in renal colic.

Keywords: Desmopressin, Renal colic, Urolithiasis, Analgesia, Renal stone

Introduction

Urolithiasis (or renal stone) is a frequently occurring problem which has a prevalence of 1 to 20% in the population depending on geographical, ethnical and genetic factors as well as on gender, climate and diet. A recent national cohort study from the United States has reported a prevalence of approximately 9% in men and 7% in women. This study suggests that whereas previously there was a significant male predominance in the presence of renal stones, this now appears to be gradually evening out. Although renal stones can occur from infancy to old age, the highest incidence has been reported to occur in men and women between the 3rd and 5th decade.

The pain experienced during renal colic is caused by the acute obstruction of a urinary stone in the urinary tract. This leads to ureteric dilatation, tensile stretch and spasmodyc activity. Contrary to common belief, it is not the stone size, but rather the extent of the obstruction, the location, the presence of spasmodic activity and the incidence of infection that are more relevant factors influencing the level of pain.

For uncomplicated stones up to 10 mm, a conservative approach with observation and analgesia is a valid option. If observation for spontaneous passage is selected as primary treatment, then this is usually tried for 4–6 weeks. Clinicians currently commonly use medical expulsive therapy (MET) in an effort to increase stone passage rate and decrease time to stone passage as well as pain caused by the stone. The concept of MET is to assist the fluent passage of the renal stone by increasing the urethral diameter through smooth muscle relaxation. Previously researched agents for MET include α-blockers, calcium channel blockers and prednisolone. Nonetheless, the evidence for this is not unanimous and some recent evidence would suggest that tamsulosin is not effective in decreasing painful episodes and reducing time to stone passage. Due to conflicting evidence, further research in this matter is still warranted.

Based on the pathophysiology described above, analgesia in renal colic aims to relax the ureteric smooth muscle and to decrease the flow within the urinary tract, i.e. decreasing the diuretic effect. Current management mainly encompasses a combination of NSAIDs (non-steroidal anti-inflammatory drugs) and opiates. NSAIDs are regarded as first line according to the guidelines of the European Association for Urology and work by inhibiting prostaglandin synthesis. They are far more effective than opiates in renal colic and comparatively have reduced requirements for further analgesic therapeutics. Although NSAIDs are the most efficient group of analgesia for renal colic, its use may be restricted due to the risk of side effects in certain patient populations, such as gastrointestinal bleeding, renal function impairment and cardiovascular side effects.

Opiates are often heralded as excellent centrally acting analgesic agents. In pain caused by renal colic, the effect of opiates has not been shown to be as effective as NSAIDs. Opiates do not reduce the inflammation which is associated with renal colic, and as a result, there is often
need for co-administration of other analgesic medication. The problem is that opiates do not counter the underlying cause of the pain and their central analgesic effect on its own is not powerful enough. The combination of opiates with NSAIDs on the other hand has been shown to have a superior analgesic effect in comparison with either drug alone. They also have a multitude of side effects which include nausea and vomiting, drowsiness, respiratory depression and addiction.

With the rationale that analgesia in renal colic can be achieved by relaxing the ureteric smooth muscle and by decreasing the flow within the tubes, i.e. decreasing the diuretic effect, antispasmodics are frequently incorporated in analgesic regimens for renal colic. Nonetheless, they have been shown to have limited to no beneficial effect on renal colic pain control in addition to standard analgesic treatment. Studies which explored the effect of hyoscine N-butyl bromide and phloroglucinol were not able to demonstrate a significant analgesic effect. Nitrates were studied in renal colic for their relaxing effect on smooth muscle; however, again no clear analgesic result was shown. However, other smooth muscle relaxants such as drotaverine, PDE4 inhibitors and papaverine were able to show encouraging results, showing a role for smooth muscle relaxation in analgesia.

Due to its antidiuretic effect and experience from rabbits that it inhibits spontaneous contractions of circular smooth muscle, there has been increased attention towards the analgesic action of desmopressin in renal colic. Desmopressin (1-desamino-8-d-arginine vasopressin) is a synthetic analogue of the anti-diuretic hormone (ADH) with a stronger and longer anti-diuretic effect and decreased vasopressor effect. It binds to the V2 receptor in the distal renal tubule and collecting duct leading to the reabsorption of water and eventually resulting in an anti-diuretic effect. As such it is also used in the treatment of nocturia and polyuria. The aim of this paper was to perform a review on the use of desmopressin in the management of acute renal colic.

Methods
A literature search was performed between October 2015 and February 2016 on PubMed, Medline and the Cochrane Library with the MeSH term search string ‘desmopressin renal colic” for English text articles. Eighteen articles were found of which 11 human studies, 5 review articles and 2 animal studies. For this review, we mainly focused our attention towards the 11 human studies as we wanted to review the existing human data on the use of desmopressin. We also used the animal studies and reviews for hypotheses on the possible mechanistic action of desmopressin in achieving an analgesic effect.

Results (also see Table 1)
Kimoto et al. reported on the effect of desmopressin on circular smooth muscle cells in an animal model in 1990. They demonstrated that desmopressin is able to reduce spontaneous muscle contraction in a dose-specific and region-specific manner. This study thus provided the first insight into the potential use of desmopressin for renal colic pain.

A first human pilot study was performed in 1995 in Qatar on 18 patients. The participants were administered 40 μg of intranasal desmopressin spray on admission for renal colic in the emergency department. Eight of the 18 patients had a noteworthy resolution of pain after 30 min of administration. A further 9 patients did not feel pain after a supplementary administration of diclofenac sodium. Constantinides et al. applied a similar regimen in a study with 108 participants and reported resolution of pain within 30 min of intranasal desmopressin administration in 58 patients. In 44 patients, there was an additional requirement of prostaglandin synthesis inhibitors, and in 6 patients, a further dose of pethidine (an opiate) was given. Although these were non-randomised, observational studies without a control arm, they both indicated the potential analgesic effect of desmopressin and pointed out the need for further studies.

In a prospective randomised controlled trial in 2001, Lopes et al. randomised a total of 61 patients in three intervention groups: group A was treated with 40 μg intranasal desmopressin monotherapy, group B with intramuscular diclofenac monotherapy and group C with the combination of desmopressin and diclofenac. The visual analogue score (VAS) was measured at baseline and at 10, 20 and 30 min after administration. Although the true effect of desmopressin on renal colic pain is hard to assess in the absence of a control arm, patients in group A did have a pain reduction 30 min after desmopressin administration. This effect, however, was significantly less potent at 30 min when compared to diclofenac monotherapy (group B) and diclofenac plus desmopressin combination therapy (group C). Complete pain resolution was observed more in groups A and C than in group B. The study did not provide statistical evidence that combination therapy with desmopressin is better than diclofenac; however, combination therapy reported the lowest pain scores and the least number of patients requiring additional analgesia. Bearing in mind that NSAIDs such as diclofenac are nephrotoxic in patients with chronic renal disease, these results suggest the potential of desmopressin as an adjunct to reduce the dependency of NSAIDs for renal colic.

Hazhir et al. measured VAS in 90 randomised patients to 3 groups: group A received 40 μg intranasal desmopressin, group B 100 mg intramuscular tramadol and group C both tramadol and desmopressin. There was no significant difference in VAS between the 3 groups. When looking at the requirement for further analgesia, only in 33.3% of patients in group A, a further dose of pethidine was required in comparison with 50% in group B and 46.7% in group C. Again here the difference was not significant, but the authors did note that desmopressin seemed to have an
Table 1  The results of 10 studies exploring the analgesic effect of desmopressin are summarised in this table. For the purposes of clarity, we have renamed some of the groups in the table when compared to the original studies as to fit under the type of therapy received. The study by Juul et al. was a retrospective analysis of desmopressin prescribing and has been left out from the table.

<table>
<thead>
<tr>
<th>Study</th>
<th>Study type</th>
<th>Country</th>
<th>N</th>
<th>Group A/N (desmopressin monotherapy)</th>
<th>Group B/N (control therapy)</th>
<th>Group C/N (combination therapy)</th>
<th>Group D/N (high-dose desmopressin monotherapy)</th>
<th>VAS A 30’</th>
<th>VAS B 30’</th>
<th>VAS C 30’</th>
<th>VAS D 30’</th>
<th>Additional therapy/N</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>El-Sherif</td>
<td>Prospective observational</td>
<td>Qatar</td>
<td>18</td>
<td>Intranasal desmopressin 40 μg/18</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Diclofenac/9</td>
<td>NA</td>
</tr>
<tr>
<td>Constantines</td>
<td>Prospective observational</td>
<td>Greece</td>
<td>108</td>
<td>Intranasal desmopressin 40 μg/108</td>
<td>Intranasal desmopressin 40 μg/20</td>
<td>Intranasal desmopressin 40 μg/diclofenac 22</td>
<td>–</td>
<td>5.5</td>
<td>3</td>
<td>2.8</td>
<td>–</td>
<td>Prostaglandin synthetase inhibitor/44 and Pethidine/6</td>
<td>NA</td>
</tr>
<tr>
<td>Lopes</td>
<td>Non-blinded RCT</td>
<td>Portugal</td>
<td>61</td>
<td>Intranasal diclofenac 75 mg/19</td>
<td>Intranasal desmopressin 40 μg/diclofenac/22</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Desmopressin/7 (group B)</td>
<td>A-B: P &lt; 0.05</td>
</tr>
<tr>
<td>Hazhir</td>
<td>Non-blinded RCT</td>
<td>Iran</td>
<td>90</td>
<td>Intranasal desmopressin 40 μg/30</td>
<td>IM tramadol 100 mg/30</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Tramadol/2 (group C)</td>
<td>A-C: P &lt; 0.05</td>
</tr>
<tr>
<td>Roshani</td>
<td>Placebo-controlled double-blind RCT</td>
<td>Iran</td>
<td>150</td>
<td>–</td>
<td>Placebo + Diclofenac suppository 100 mg/70</td>
<td>Intranasal desmopressin 40 μg/diclofenac suppository 100 mg/75</td>
<td>–</td>
<td>5.7</td>
<td>3.7</td>
<td>–</td>
<td>–</td>
<td>Pethidine/10 (group A)Pethidine/15 (group B)Pethidine/14 (group D)</td>
<td>B-C: P &lt; 0.05</td>
</tr>
<tr>
<td>Kheirollahi</td>
<td>Open-label RCT</td>
<td>Iran</td>
<td>116</td>
<td>–</td>
<td>Buscopan alone/58</td>
<td>Combination: buscopan + desmopressin 20 μg/58</td>
<td>–</td>
<td>7.26</td>
<td>5.95</td>
<td>–</td>
<td>–</td>
<td>Morphine, no specific data on numbers</td>
<td>B-C: P = 0.001</td>
</tr>
<tr>
<td>Masoumi</td>
<td>Placebo-controlled Double-blind RCT</td>
<td>Iran</td>
<td>120</td>
<td>Desmopressin 40 μg + IM diclofenac 75 mg/60</td>
<td>Diclofenac 75 mg + placebo/60</td>
<td>Combination therapy/24</td>
<td>–</td>
<td>5.68</td>
<td>4.52</td>
<td>–</td>
<td>–</td>
<td>Morphone/23 (group A) Morphone/27 (group B)</td>
<td>P = 0.02</td>
</tr>
<tr>
<td>Kumar</td>
<td>Non-blinded RCT</td>
<td>India</td>
<td>72</td>
<td>Desmopressin 40 μg/24</td>
<td>Diclofenac 75 mg/24</td>
<td>–</td>
<td>–</td>
<td>7.5</td>
<td>3.75</td>
<td>4</td>
<td>–</td>
<td>Diclofenac/24 (group A) Desmopressin/2 (group B) Diclofenac/3 (group D)</td>
<td>NS</td>
</tr>
<tr>
<td>Keshvari</td>
<td>Single-blind RCT</td>
<td>Iran</td>
<td>81</td>
<td>Morphine 0.1 mg/kg + desmopressin 60 μg/41</td>
<td>Morphine 0.1 mg/kg + placebo/40</td>
<td>–</td>
<td>–</td>
<td>8.83</td>
<td>7.13</td>
<td>–</td>
<td>–</td>
<td>No data provided</td>
<td>A-B: P &lt; 0.05</td>
</tr>
<tr>
<td>Pricop</td>
<td>Single-blind RCT</td>
<td>Romania</td>
<td>249</td>
<td>IM Ketorolac + placebo/71</td>
<td>Desmopressin 60 μg/62</td>
<td>Combination: IM ketorolac + 60 μg desmopressin/59</td>
<td>Desmopressin 120 μg/62</td>
<td>4.3</td>
<td>3.8</td>
<td>3</td>
<td>3.2</td>
<td>Tramadol or JJ stent/21 (group A) Tramadol or JJ stent/7 (group B) Tramadol or JJ stent/12 (group C) JJ stent/9 (group D)</td>
<td>B-C: P = NS</td>
</tr>
</tbody>
</table>

Notes: NA – not applicable, NS – not significant, N – number of patients.
analgesic effect with reduced need of additional analgesia. These data should be interpreted with caution as there is considerable bias included in the study with the exclusion of 15 patients that needed rescue analgesia before 30 min after administration of study drug, the absence of pre-treatment pain scores and the lack of a double-blinded design.\textsuperscript{18}

In a double-blind placebo-controlled randomised controlled trial by Roshani et al., 150 patients were divided into 2 groups. The first group was given 40 μg intranasal desmopressin with 100 mg diclofenac sodium suppository and the second group 100 mg diclofenac sodium suppository with a 0.9% NaCl placebo spray. The study showed significantly better pain scores in group 1 when compared to group 2 at 15 and 30 min after administration and provided further evidence for desmopressin as a supplement for treatment in renal colic.\textsuperscript{19}

In an open-label two-arm randomised trial with 114 participants, Kheirrollahi et al. compared the combination of 20 mg hyoscine N-butylbromide and 20 μg desmopressin to 20 mg hyoscine N-butylbromide monotherapy. The VAS pain score was measured at baseline and at 30 and 60 min after administration. The pain scores were similar at baseline and were lower in both groups at 30 min; however, only in combination therapy, the pain score kept decreasing significantly at 60 min.\textsuperscript{20} This study again demonstrated the potential of desmopressin, but there were certain limitations which must be mentioned. First of all, the pain score at 60 min was measured against scores at earlier timepoints in the same group and not to pain scores in the other group. Furthermore, the lack of blinding in the design could have led to a variable experience of pain as patients knew which treatment they were given. Furthermore, there was no group of desmopressin monotherapy and there are known reservations on the use of hyoscine N-butylbromide as an analgesic agent in renal colic.\textsuperscript{4} And lastly, there was no numerical data on pain scores after 60 min.

Masoumi et al. performed a placebo-controlled randomised double-blinded clinical trial, including 120 patients between the ages of 18 and 55 years. Patients were randomised 1:1 to either combination therapy of 40 μg desmopressin and 75 mg intramuscular diclofenac sodium or to 75 mg intramuscular diclofenac monotherapy. The VAS pain score for all patients was measured at baseline and at 15, 30 and 60 min after drug administration. There was a statistically significant difference (p = 0.02) in pain sensation at each time point after administration in favour of combination therapy, demonstrating the benefit of adding intranasal desmopressin to intramuscular diclofenac in acute renal colic pain. The study, however, did not explore the use of desmopressin as monotherapy, and the effect was only studied up till 60 min after administration.\textsuperscript{21}

A recent study by Pricop et al. randomised 249 patients with renal colic to four groups: one group of 71 patients who received ketorolac tromethamine 30 mg IM and a sublingual vitamin C placebo, two groups of 57 and 62 patients, respectively, who received 60 and 120 μg of sublingual desmopressin, and one group of 59 patients who received combination therapy with 30 mg IM ketorolac and 60 μg sublingual desmopressin. The VAS pain score was assessed at baseline and thirty minutes after drug administration. Desmopressin monotherapy and combination therapy showed a decrease in pain intensity of 56% (P < 0.05) and 59% (P < 0.001), respectively, compared to a decrease of 47% with ketorolac alone. This suggested a potent analgesic action of desmopressin therapy, and a benefit of desmopressin as an adjunct to NSAIDs.\textsuperscript{22}

Kumar et al. randomised 72 patients to three groups. Group A received 40 μg intranasal desmopressin, group B 75 mg intramuscular diclofenac and group C both desmopressin and diclofenac. Pain was measured again with the use of VAS scores. The study demonstrated no significant analgesic effect at 30 min in group A (desmopressin monotherapy). Both groups B and C experienced significant pain reduction, without a statistical difference between both groups. The authors concluded that intranasal desmopressin monotherapy did not provide an effective analgesic action and that it did not strengthen the effect of diclofenac sodium.\textsuperscript{23} This particular study did not seem to have a single- or double-blinded component to it, which made interpretation of the results less straightforward.

Keshvari et al. compared the use of sublingual desmopressin melt and morphine versus morphine and placebo in renal colic. The study was a single-blinded prospective randomised controlled trial of 81 patients and showed that the pain score was significantly higher after 30 min in the study group with desmopressin and morphine when compared to the control group with morphine and placebo. As the pain score was not measured after 30 min, the study did not provide any data on pain scales after this. Therefore, the authors recommended more and larger randomised controlled trials to clarify the potential of desmopressin in renal colic.\textsuperscript{24}

To explore the frequency of off-label desmopressin prescribing for renal colic, a retrospective study was performed of national epidemiological data of all prescriptions in Denmark between 2009 and 2011. The study was funded by Ferring Pharmaceuticals and was written by authors from the Ferring International PharmaScience Center in Copenhagen and from the Aarhus University Hospital. The authors based their analysis on the Danish National Prescription Registry which has been in existence since 1994 and records data on prescribing indications since 2004. As a result, the researchers were able to identify 888 prescriptions of desmopressin specifically for renal colic. These were for 95 patients with an average age of 65.4 years and an average treatment period of 159 days, out of a total Danish population of 5,580,134. This indicated that of all patients estimated to have renal colic from that population, and 0.2–2% were prescribed desmopressin. No statistical analysis was performed as the study had actual data for an entire population group rather
than a sample of that population. Nonetheless, as a result of the study design, it was difficult to suggest whether these prescriptions for desmopressin also related to concrete pain relief. The key finding from this study was that in Denmark, a minority of patients suffering from renal colic are prescribed desmopressin.25

Discussion

Patients with renal colic pain often require immediate diagnosis and treatment. Currently, NSAIDs and opioids are used as first-line agents; however, the potential side effects of current standard analgesic treatment for renal colic prohibit its use in certain patients.8 Therefore, it is important to study the effect of safe alternative treatments in order to reduce our dependence on NSAIDs.

The Danish study from 2011 indicates that there is off-label use of desmopressin among the medical fraternity and that this agent’s potential as an analgesic strategy in renal colic needs to be further explored. This has to do with the action of desmopressin itself, the advantages in the use of desmopressin and the encouraging results in published studies.

The exact method by which it may lead to an analgesic effect is still to be elucidated. What we do know is that desmopressin has a widely appreciated antidiuretic effect which in rat models has shown to reduce the intra-ureteral pressure.26 Kimoto et al. demonstrated that desmopressin inhibits the spontaneous contractions of the circular smooth muscle in the renal pelvis of rabbits, which reduces the peristaltic activity and hence decreases the triggers of pain.13 It is possible that a combination of these two peripheral effects of intra-ureteral pressure decrease and relaxation of the ureteric musculature in the renal pelvis leads to the analgesic effect of desmopressin. Additionally, it has been shown that desmopressin acts centrally by releasing β-endorphins from the hypothalamus.17

Desmopressin has multiple advantages which include an easy administration, few side effects and a biphasic effect. This does not only make it an ideal agent in the emergency department by its acute action which has been described in above studies, but also due to its prolonged effect which requires further research. In particular, the double-blinded randomised controlled trials19,21 are of high value due to lower bias.

At the same time, it is important to note that most studies only analyse a selected group of patients and researchers remain very prudent in their inclusion criteria. This is understandable due to the experimental use of desmopressin, however, and gives us limited knowledge on the potential side effects such as hyponatraemia, fluid retention, hypotension, gastrointestinal upset and headache. From the above studies, Pricop et al. measured blood pressure, serum osmolality and serum creatinine at baseline and at 30 min after intervention, and did not find any changes.17 A study by Stoof et al. explored the side effect profile of desmopressin when used for bleeding disorders.27 Desmopressin was administered intravenously as a single bolus of 0.3 μg/kg or intranasally as 300 μg. Blood was sampled at baseline and at one, three, six and 24 h after administration. A total of 108 patients were studied, of which 4% had mild-to-moderate hyponatraemia, 38% were hypotensive and 9% tachycardic – all of which were temporary and mostly resolved at 24 h. About 71% of patients further reported headache, fatigue, flush and/or dizziness. The authors concluded that most of the side effects noted in the study were expected and related to the known vasomotor and antidiuretic effect of desmopressin and that desmopressin remains a safe acute therapeutic strategy for bleeding disorders with careful preparation such as fluid restriction and measurement of serum osmolality or sodium concentration prior to repeat administration.

A following point of note is that most studies are focused around the acute admission of the patient. This allows us to witness the immediate action and side effects of desmopressin, however does not provide us information on the prolonged effect and safety. This is especially important if desmopressin would be used during the conservative observational management of renal colic pain. The Danish study gave an insight into the possible side effects of long-term desmopressin use by highlighting the number of patients that were admitted with hyponatraemia to hospital.25 Their retrospective analysis showed that four of the 95 patients who were prescribed desmopressin for renal colic required treatment for the consequences of its hyponatraemia. This is the result of the dilutional effect of desmopressin due to the reabsorption of water. The risk of hyponatremia is an important consideration, especially in patients over 65 years old and those suffering from polydipsia or from comorbidities requiring medications such as selective serotonin re-uptake inhibitors and tricyclic antidepressants. This shows that the risks of desmopressin will require appropriate analysis and preventative strategy prior to its widespread use.

Another important point is that desmopressin reduces flow in kidneys and ureters. If this reduced flow remains for a long time, then there is a theoretically increased chance of stone formation. The Danish study found that stone formation occurred in 12 patients of 499 (2.4%) who were prescribed desmopressin for cranial diabetes insipidus.26 This does not mean that there is a causal link between the use of desmopressin and stone formation, but it does give strength to this theoretical hypothesis. ‘Breakthrough diuresis’ is therefore an essential element when prescribing desmopressin for CDI in the guidelines, and it may be required to consider this for renal colic as well.

Additionally, most studies where desmopressin is used as monotherapy show little to no effect.17,19,23 From this, it appears that intranasal desmopressin 40 μg cannot be used as a replacement therapy for conventional analgesia such as NSAIDs. Nonetheless, desmopressin does seem
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Current evidence, albeit limited and of insufficient quality, reflects that desmopressin may have potential in the treatment of acute renal colic. High-quality, sufficiently powered, double-blind placebo-controlled randomised controlled trials are directly needed to add proof to this hypothesis. Further analysis is required to compare desmopressin as monotherapy to combination therapy and control medication such as NSAIDs, opiates and α-blockers in MET. Additionally, a possible placebo effect in the use of desmopressin remains to be further studied. Only after these factors have been addressed, can desmopressin be considered for inclusion in the guidelines for the treatment of renal colic pain.

Conclusion

Current medical treatment for renal colic primarily encompasses NSAIDs, opiates and α-blockers. The use of these can be limited in certain groups of patients due to the higher risk gastrointestinal, renal, cardiovascular and neurological side effects. As such, desmopressin has been studied for its potential analgesic action against renal colic in small, local randomised controlled trials which have shown promising results. Since there is no large-scale clinical evidence for a singular solution, there is now need for larger, high-power and thoughtful randomised controlled trials and meta-analyses with regard to the use of desmopressin. Only then can we truly explore the action of desmopressin and perform a benefit-risk analysis for its widespread use.

Declaration of interest

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