

dynamic process. Furthermore, if waits to see a physician in the ED, patients should be reassessed continually. However, this may be impossible in an ED not provided sufficient nursing staff.

Triage nurses who have responsibility for patients already triaged and waiting in the waiting room are often unable to re-evaluate patients simply because they are too busy with new patients. By rechecking vital signs and talking with the patient every two hours, patients should be re-triaged to a higher category should their conditions deteriorate. Another issue relates to changing chief complaints. In Case #1 the patient's friend complained that the patient developed chest pain two hours after arrival, yet these complaints were not acted upon immediately by the triage nurse because of overall overcrowding and dividing professional focus among too many patients.

Sorting out common illness from catastrophic illness can be difficult. In Case #2, the patient who presented with headache, fever, and vomiting appeared little different than others with URI symptoms and appeared appropriate for an "urgent" category. In most triage systems an initial increased respiratory rate of 24 in the absence of stridor would also be categorized as urgent. It is unclear if the history of trauma would have changed the triage category to emergent. In addition, the patient did not receive repeat vital signs two hours after presentation to the ED. In Case #1, the patient's respiratory rate was 24 and pulse was 116. If only EM physicians had been available and not busy with other patients. It is possible that a full triage re-assessment at two hours would have changed the patient's category to "emergent."

Triage in the ED is very high-risk, yet does not receive the attention, funding, or CQI reviews that would reflect its status as a high-risk activity. One of the major problems in large hospitals is that the triage nurse is pressured by long lines of patients, and may perform triage too briefly and too hastily to pick up subtle signs of high-risk disease. Questions have also been raised about the sensitivity and specificity of nurse triage. In a study performed at the University of New Mexico, investigators found that visual triage assessment by physicians significantly increased sensitivity in identifying those patients who had illnesses resulting in admission.⁵ In the United Kingdom, a five category triage system has been advocated to increase accuracy in identifying potentially ill patients. The rate of under-triage of patients is unclear, and has not been widely studied. The Accident in Emergency Department at the Kwong Wah Hospital in Hong Kong reported a 3.4% instance of under-triage.⁶ Although this number is relatively small, when one considers a very large ED with 50,000 patients triaged per year, potentially over 1,500 patients could be under triaged and sent to the waiting room with potentially serious and unrecognized conditions.

In conclusion, these two cases illustrate the difficulty of initial triage, and how disease states may progress rapidly after triage. Furthermore, that patients who truly require urgent intervention may not receive timely treatment in overcrowded EDs. To avoid potential unexpected outcomes, EDs must be provided with sufficient resources to prevent overcrowding and insure timely evaluation of all patients by emergency physicians.

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TOXICOLOGY REVIEW

Quinapril Overdose-Induced Renal Failure

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Background:

Angiotensin converting enzyme (ACE) inhibitor toxicity is infrequently reported and seldom produces significant clinical effects. Only 15 previous cases of intentional ACE inhibitor overdoses among adults have been published.¹⁻¹³ The most commonly observed clinical effect is hypotension,¹⁴ which is typically transitory and responsive to supportive measures and intravenous hydration. An additional 48 pediatric exposures were reported in a single case series, none of which resulted in any adverse effects related to the ACE inhibitors.¹⁵

Reversible impairment in renal function has been reported in 5 cases of ACE inhibitor overdose, but only in association with systemic hypotension (systolic blood pressure < 90 mmHg).^{3,5,12,13} We report a case of intentional quinapril overdose where the patient presented two days later with acute renal failure in the absence of clinical features of systemic hypotension. Additionally, this is the first reported case of quinapril overdose of which we are aware.

Case Report:

A 24-year-old man presented to the Emergency Department (ED) with complaints of bilateral flank pain and decreased urine output for one day. The patient admitted to intentionally ingesting 40 of his mother's 5 mg quinapril tablets two days earlier in an attempt to "get high." The patient's mother confirmed that approximately 40 of her quinapril tablets were missing.

The patient reported no early adverse effects, but had two episodes of non-bloody, non-bilious emesis and three loose stools the following day. He denied any dizziness, lightheadedness, near-syncope, or other orthostatic symptoms. On the day of presentation, the

patient developed intermittent bilateral flank pain without radiation, dysuria, or hematuria. He had voided a "small amount" of urine only once in the previous 24 hours. Past medical history was significant for schizophrenia for six years, for which he was treated with clonazepam, olanzapine, and valproic acid. The patient smoked and occasionally binged on ethanol, but denied abuse of psychostimulant and intravenous drugs.

In the ED, vital signs were: temperature 36.1°C, pulse 96/min, respirations 20/min, blood pressure 158/73 mmHg. The patient was a mildly obese, young adult male in no apparent distress. Except for moderate bilateral costovertebral angle tenderness, physical examination was unremarkable. Mental status examination demonstrated full orientation, but poor insight and judgement, and chronic complaints of visual and auditory hallucinations.

Routine serum chemistry evaluation showed: sodium 131 mEq/L, potassium 4.1 mEq/L, chloride 95 mEq/L, bicarbonate 22 mEq/L, BUN 59 mg/dL, creatinine 7.8 mg/dL, glucose 98 mg/dL. Complete blood count showed: WBC 10.5 k/mm³, hemoglobin 14.0 g/dL, hematocrit 41.5%, platelets 236 k/mm³. Urinalysis showed: specific gravity 1.010, pH 6.0, and 100 mg/dL protein, without glucose, ketones, nitrite, or any blood cells. Additional testing revealed: serum CPK 174 IU/L, valproic acid 30 µg/mL, and no detectable amount of ethanol, lithium, salicylate, or acetaminophen. A urine drugs-of-abuse screen was negative, while a comprehensive urine drug screen (utilizing thin-layer chromatography, high-pressure liquid chromatography, and gas chromatography – mass spectroscopy) was positive only for olanzapine and valproic acid; quinapril and other ACE inhibitors are not included in this screen. Chest X-ray and electrocardiogram were unremarkable.

The patient was admitted for acute renal failure. Renal ultrasound showed bilateral diffusely increased echotexture, without signs of obstruction or renal stones. Serum creatinine peaked at 8.3 mg/dL on hospital day 2, and the BUN peaked at 74 mg/dL on hospital day 3. Renal biopsy was scheduled, but the patient was transferred to another institution by his insurer on hospital day 3. The nephrologists at both institutions agreed that the acute renal failure was due to quinapril toxicity. The BUN and creatinine rapidly improved without hemodialysis, decreasing to 30 and 1.9 mg/dL respectively by discharge on hospital day 5, and therefore no biopsy was performed. At telephone follow-up 6 months after admission, the patient reported no long-term sequelae to his overdose.

Discussion:

Renal failure from ACE inhibitor overdose may occur by causing systemic hypotension and/or by reducing glomerular filtration pressure. The proposed mechanism for both effects is decreased angiotensin II levels, in the former case affecting the systemic vasculature, and in the latter case resulting in efferent arteriolar dilation. Lending support to this theory are two prior severe cases of ACE inhibitor overdose-induced hypotension, resistant to IV volume resuscitation and adrenergic vasopressor agents, which were successfully treated with angiotensin II infusions: one case from enalapril¹² and one from lisinopril.¹³ Therapeutic ACE inhibitor use may also cause renal failure by inducing glomerulonephritis,¹⁴ but this has not been reported in overdose situations.

Systemic hypotension has been present in all previously reported cases of ACE inhibitor overdose-induced renal dysfunction,^{3,5,12,13} although hypotension is not universally seen with over-

dose.^{6,8} Furthermore, in the prior cases with renal failure, the clinical and laboratory evidence of impaired renal function resolved coincident with correction of systemic hypotension.

Our patient did not present with systemic hypotension, nor had he experienced any orthostatic symptoms following his quinapril ingestion. We suggest, therefore, that his renal failure occurred as a result of decreased glomerular filtration pressure in the absence of systemic hypotension. If this theory is true, our patient appears to be the first victim of ACE inhibitor overdose to suffer renal failure without a "pre-renal" cause. Potential weaknesses in this supposition include lack of documented blood pressures prior to ED presentation, laboratory confirmation of the presence of quinapril or its metabolites, or serum angiotensin, aldosterone, or ACE activity levels. Nevertheless, we have no good reason to doubt the patient's history of acute quinapril overdose, especially as it was confirmed by his mother, and no other cause of acute renal failure was discovered by laboratory and radiographic evaluation. The patient's rapid and apparently complete recovery is most consistent with an acute toxic injury to the kidney, rather than any intrinsic renal disease.

Conclusion:

Overdoses with quinapril or other ACE inhibitors may occasionally result in renal failure. This effect is presumably due to reduced glomerular filtration pressure from efferent arteriolar dilation, and may occur in the absence of clinically evident systemic hypotension.

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