

# Lisinopril and small bowel obstruction;

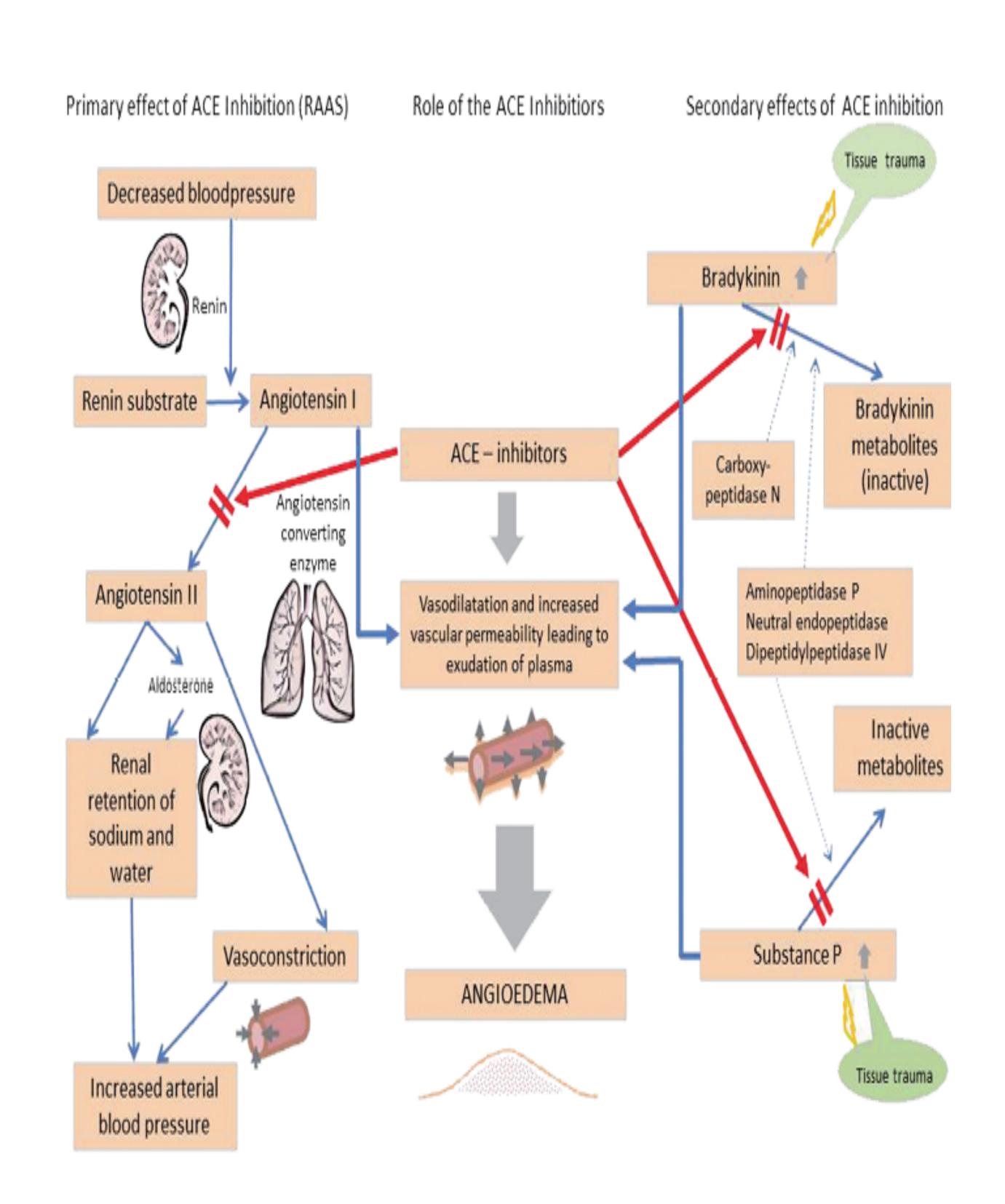
# a case report on ACE inhibitor-induced angioedema.

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### Introduction

Lisinopril is one of the most commonly prescribed medications in the United States. ACE inhibitors are a cornerstone for hypertension (HTN) management and are often given despite their well-known side effects. A dry cough is the most common, but seldom dangerous. More alarming is the risk of angioedema, which presents less often and is more difficult to differentiate. In this case, we will present how Lisinopril led to an acute complaint of small bowel obstruction, uncovering a lesser-seen adverse effect of this highly prescribed drug class. We will also demonstrate the vitality of efficient history taking in the setting of acute abdominal pain.



## **Case Report**

A 62-year-old female with a chief complaint of 10/10 generalized abdominal pain was treated in the emergency department (ED). Her symptoms began several days prior with an onset of upper abdominal discomfort following consumption of a heavy meal. Her generalized abdominal pain continued for several days, and the development of increasing RUQ pain accompanied by nausea prompted her to present to the ER.

She denied any vomiting, constipation, blood in stool, sick contacts, recent travel, or other gastrointestinal symptoms. Review of systems was negative for chest pain, cough, shortness of breath, rashes, or pruritis. The remainder of her review of symptoms, including GI and GU, was unremarkable.

The patient described multiple similar episodes of abdominal pain over the past decade, but none have been this severe, leading her to believe them to be "gastritis". She also noted an original hospitalization for abdominal obstruction attributed to lisinopril 10 years earlier, which was successfully treated non-surgically. Notably, her medication was continued despite this outcome. She even received a dose increase 1.5 years ago.

Past Medical Hx	Medications	Vitals	Labs	Past Surgical Hx
<ul> <li>Diabetes Mellitus</li> <li>Hypertension</li> <li>Hypothyroidism</li> </ul>	<ul> <li>Lisinopril 10mg Qday</li> <li>Synthroid 0.025mg Qday</li> <li>Gabapentin 300mg Qday</li> <li>Duloxetine 60mg Qday</li> <li>Meloxicam 15mg Qday</li> <li>Pravastatin 20mg Qday</li> <li>Metformin 500mg BID</li> </ul>	<ul> <li>BP 123/81</li> <li>Pulse 99</li> <li>RR 20</li> <li>T 99.3</li> </ul>	<ul> <li>WBC 17.61 with a left shift</li> <li>Lactate 2.4</li> <li>C-reactive protein 41.3</li> <li>Remainder of CBC, LFTs, serum electrolytes, and UA were all normal.</li> </ul>	• none

The patient appeared to be in mild discomfort with generalized abdominal tenderness and a focus at her RUQ. Murphy's sign was negative. Her abdomen was soft, notable for obesity, and without any rebound tenderness. No masses were appreciated.

Abdominal CT imaging with contrast indicated a normal gallbladder and liver. Areas of jejunal enteritis with associated edema of the mesentery and small volume ascites were present. These findings were consistent with angioedema of the small intestine.

A sepsis bundle was initiated. The patient was admitted for further supportive care and management. Lisinopril was discontinued. Sepsis was ruled out, and both leukocytosis and lactic acidosis resolved as her clinical condition improved over the ensuing 48 hours. The patient was discharged home 2 days later in good condition and instructed to stop taking her home lisinopril medication, but to resume all others. The primary diagnosis was allergic jejunal enteritis with associated ACE inhibitor use.

#### Discussion

ACE inhibitors induce angioedema in 0.1-0.7% of recipients. They account for approximately 35% of all prescriptions for HTN management in the United States, adding up to approximately 40 million patients. Angioedema can particularly affect the bowel wall, presenting as colicky abdominal pain often associated with nausea, vomiting, and/or diarrhea.¹ The exact mechanism of this is poorly understood, but a drastic increase in bradykinin was shown to play a role in the pathogenesis of this adverse outcome. Once the ACE inhibitor blocked the degradation of bradykinin, nonpruritic angioedema of the lips, larynx, face, and intestines ensued.²

The differential diagnosis of acute abdominal pain is extensive and challenging. Late presentation of angioedema secondary to ACE inhibitors has been reported in the literature. It is estimated that 20-40% of all ED visits for angioedema can be attributed to ACE inhibitors. GI manifestations of ACE-associated angioedema occurred within 72 hours of administration in over half of reported cases, however, other cases report presentation occurring after months or even years. The jejunum is the most often involved portion of the small intestine, followed by the ileum and duodenum. Physicians should be aware of this rare manifestation of ACE-inhibitor intolerance and consider it in the differential diagnosis of abdominal pain. Thorough history taking was essential to appreciating the potential role ACE inhibitor therapy played in this patient's clinical condition.

#### References

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