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A Case Report: Painless Type A Aortic Dissection with Cardiac Tamponade as Initial Presentation

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Abstract A 70 years old male with past medical history of hypertension, remote history of colonic and prostate cancer and ischemic stroke came to the hospital with generalized weakness, fatigue, increased shortness of breath and decreased exercise tolerance for the past week. Bedside echocardiogram revealed large amount of pericardial effusion with signs of pericardial tamponade. Patient successfully underwent echocardiography-guided pericardiocentesis with improvement of symptoms and remained stable and 900 cc of bloody fluid was drained. Blood tests revealed markedly elevated D-dimer, elevated liver enzymes and acute kidney injury. Chest X-ray showed widened mediastinum. CT chest without a contrast indicative of ascending aortic dissection, was confirmed by magnetic resonance imaging (MRI) of the chest. During and prior to admission to our hospital patient had reported no chest pain. Cardiac tamponade was the only clinical finding, which is very unusual for acute aortic dissection to be diagnosed. Patient was transferred to another institution for surgical intervention, which revealed sealed ruptured aortic dissection into pericardium, with successful repair of the dissection, and later was discharged in stable condition.

Keywords: *painless, type A aortic dissection, cardiac tamponade, atypical presentation, sealed rupture*

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1. Introduction

Aortic dissection, especially ascending aortic dissection is rare but can be catastrophic if not diagnosed and treated promptly. The presenting signs and symptoms of acute aortic dissection are diverse, and yet an early recognition and timely surgical intervention is essential for survival. The classic presentation is acute – onset of chest pain that is sharp and tearing in quality radiating to the back in the intrascapular region. [1] Hemorrhagic cardiac tamponade has been reported in patients received anticoagulant therapy [2] and radiofrequency ablation. [3] However, spontaneous rupture of aortic root associated with hemorrhagic cardiac tamponade without precipitating risk factors is rare in clinical practice. The treatment for cardiac tamponade is pericardiocentesis, however, the indication for this procedure in the context of aortic dissection remains controversial. Here we presented a case of ascending aortic dissection complicated by rupture into pericardium with cardiac tamponade, acute liver failure, and acute renal injury but no pain as initial manifestation.

2. Case Report

A 70 years old male came to a hospital with generalized weakness, fatigue, increased shortness of breath and decreased exercise tolerance for the one week. Patient reported cough and low-grade fever (100.4F) at home. He denied chest pain, abdominal pain, nausea, vomiting, diarrhea, leg pain or swelling. No recent travel. One week prior to coming to our institution he was seen at another hospital for abdominal pain, where abdominal CT scan was done but did not reveal any significant abnormality.

His medical history is positive for hypertension, cerebral vascular accident (CVA) with left-sided residual weakness, prostate cancer that was treated surgically 13 years ago and colon cancer with surgical intervention 2 years ago.

Upon arrival to emergency department, he was afebrile, hypotensive with blood pressure of 85/49 mmHg. He was mildly tachycardic, tachypneic and desaturated at room air. Coarse rhonchi throughout lung was noted by ED staff. Patient was placed on bilevel positive airway pressure (BIPAP) and oxygen saturation improved. Blood work was significant for leukocytosis (WBC of 16.2, with 89% of neutrophils), acute kidney injury (BUN/Creatinine: 43/2.81, baseline 22/1.48), transaminitis/shock liver: ALT/AST: (547/>1500); Bilirubin total/direct (2/1.3); creatine phosphokinase of 101, troponin of 0.102, N-terminal pro b-type natriuretic peptide: 5170, lactate of 5.1. D-dimer of 6000. Bedside echocardiogram revealed large pericardial effusion with tamponade physiology. 12

lead electrocardiogram (EKG) showed supraventricular tachycardia (SVT) with heart rate of 144 beats per minute with low voltage. (Figure 1A). Emergent pericardiocentesis was performed with removal of about 900 milliliters of bloody fluid. Patient's symptoms, blood pressure and oxygen saturation significantly improved, and he was transferred to Coronary Care Unit (CCU) for further management. Post-procedural echocardiogram was performed and revealed left ventricular ejection fraction (LVEF) of 35-40%; Chest radiography showed cardiomegaly and widened mediastinum. (Figure 2).

On the next day patient developed multiple runs of SVTs, for which adenosine 6mg and 12mg were given. (Figure 1B). Subsequent EKG showed normal sinus rhythm. A few hours later, patient developed atrial fibrillation with rapid ventricular response and was started on amiodarone drip in settings of decreased ejection fraction. (Figure 1C). The amiodarone drip discontinued shortly due to liver dysfunction. Patient's heart rhythm converted back to normal sinus. (Figure 1D). On day 3 echocardiogram was repeated and revealed a dilatation of the ascending aorta with a diameter of 6.7cm. CT chest without a contrast (because of renal failure) was indicative of ascending aortic dissection, which was confirmed by MRI of the chest (Figure 3).

Patient was started on esmolol drip and transferred to another institution for surgical treatment. Patient had an uneventful surgery - it was found aneurysm ruptured into pericardial sac, however it was sealed. Patient was discharged to a rehabilitation facility in stable condition.

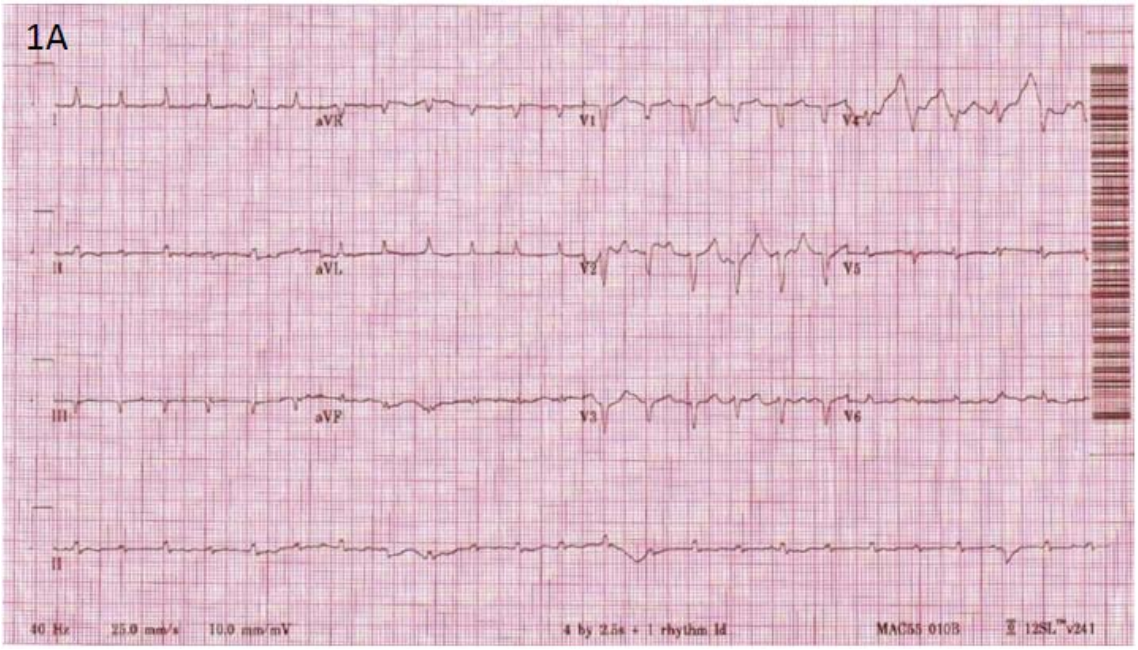

Figure 1A. EKG shows supraventricular tachycardia with low voltage (EKG was obtained before pericardiocentesis)

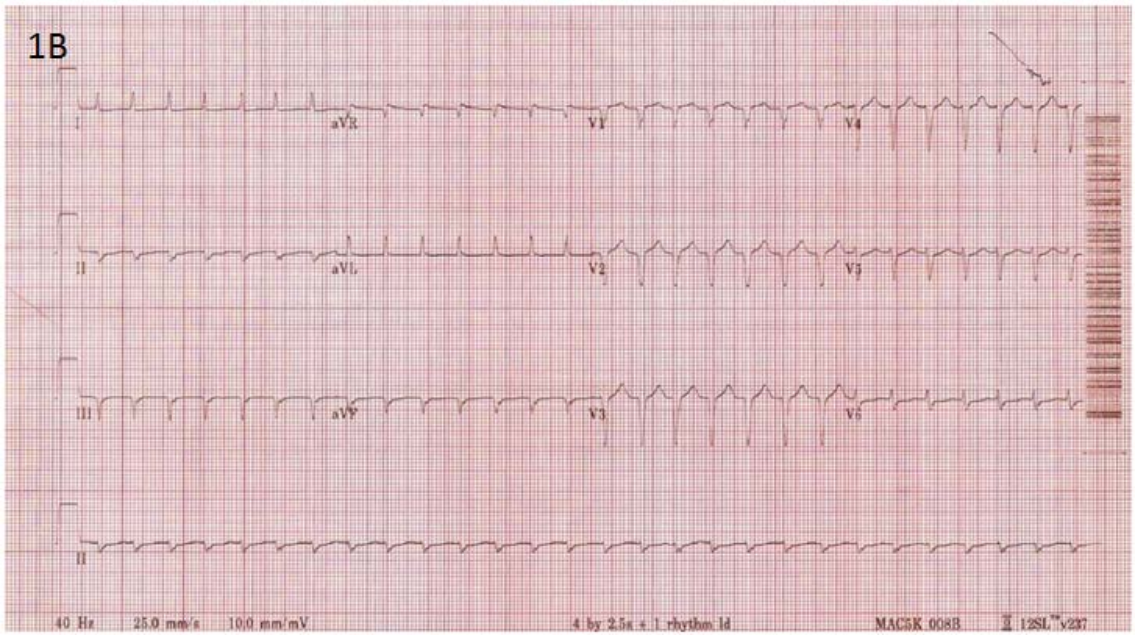

Figure 1B. EKG shows another run of supraventricular tachycardia, however there is no low voltage (EKG post pericardiocentesis)

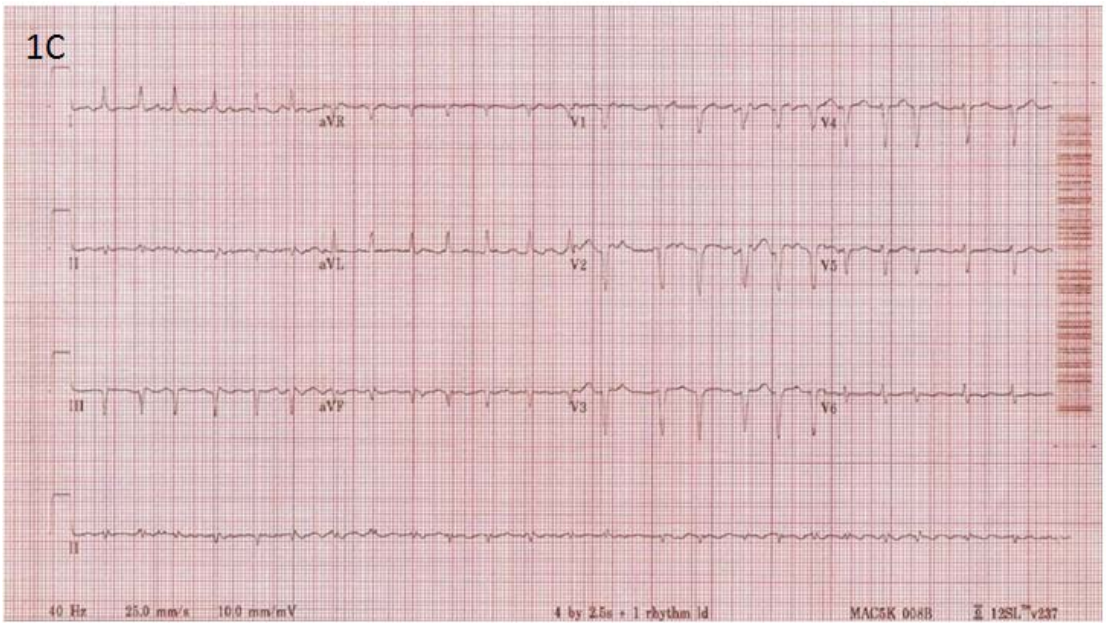

Figure 1C. Atrial fibrillation with rapid ventricular response

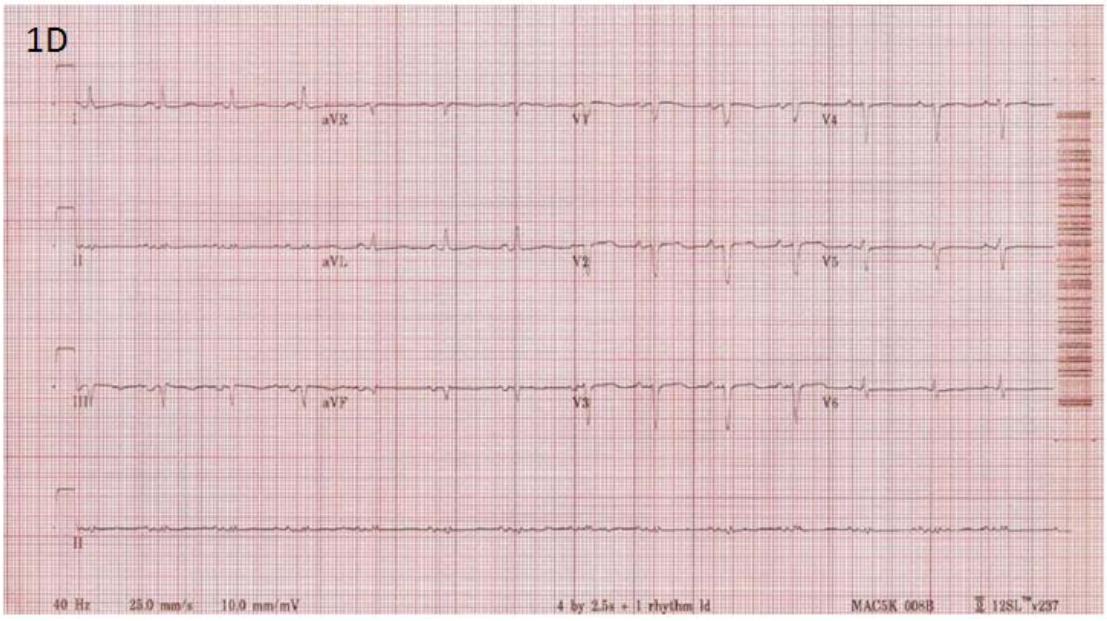

Figure 1D. Normal sinus rhythm with heart rate of 85 beats per minute. Same q in lead II, III, avF and poor r progression seen as in Fig 1A, 1B, and 1C). Inferior and anterior wall myocardial infarction, age indeterminate

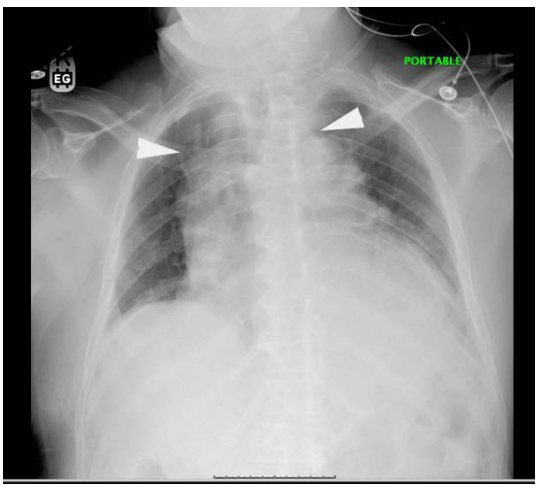

Figure 2. Chest X-ray with widened mediastinum

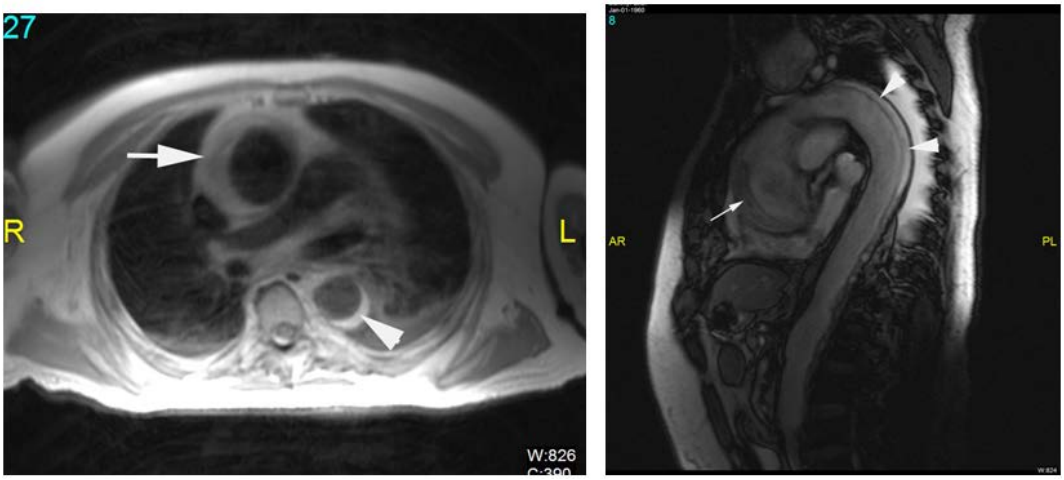

Figure 3. Axial MRI chest (Left) showing false lumen of ascending aorta (white arrow), and descending aorta (white arrow head); Sagittal MRI chest (Right) showing aortic dissection on the aortic root (white arrow) extending to the descending aorta (white arrow head)

3. Discussion

More than 250 years have passed since the first documented case of aortic dissection with rupture into pericardium. The report described dissection of the aortic arch with pericardial tamponade in king George II. [4] Despite of all progress and advances in diagnostic imaging and treatment techniques, aortic dissection remains a very ominous diagnosis with high mortality rate. Prompt and accurate diagnosis is essential to improve prognosis. In 2016 more than 9000 Americans died because of aortic dissection. [5]

Many classifications of aortic dissection have been proposed. [6,7,8] The Stanford system is the most commonly used in clinical practice. This system classifies all dissections upon two types. Type A is involving the ascending aorta and arch (proximal to the left subclavian artery) regardless of its extension. All other dissections are classified as type B. [6]

Typically, patient with aortic dissection presents with abrupt onset, sharp and tearing pain, [1,9,10,11,12] This symptom in some studies has been reported in more than 90% of cases. [10,11] Chest pain occurs more often in type A aortic dissection than type B. [9,10] Other common manifestations include murmur of aortic insufficiency (30-40%), pulse deficit (15-38%), stroke or other neurological deficit (5-15%), syncope (9-13%) and congestive heart failure (5-7%). [9,11,12] Most patients with dissection have a history of hypertension and are hypertensive upon presentation, [9-13] however in painless group normotensive or hypotensive patients are more typical. [11,14] The most common causes of death in patients with type A aortic dissection are aortic rupture, cardiac tamponade and visceral ischemia. [9] Mehta et al showed that advanced age (≥ 70), abrupt onset of pain, pulse deficit, abnormal electrocardiogram, kidney failure and hypotension/shock/tamponade are associated with worse survival and can serve as independent predictors of increased in-hospital mortality. [13]

Numbers of studies have been reported that acute aortic dissection can be painless. [11,13,14] Patient of our case did not report any pain. Painless presentation occurs more often in patients with type A aortic dissection and associated with higher mortality and more unfavorable functional outcome. [11,13,14] The prevalence of painless dissection has been reported in 6.4% - 17% of cases and it is not so uncommon as it was previously thought. Moreover, the rate of this entity may be even higher since autopsies are infrequently performed in the current era and significant amount of cases can be missed in life. [15]

Mechanism of development of painless aortic dissection is not completely understood, but several explanations have been proposed. Imamura et al in their study have noticed that patients with painless presentation had significantly higher rate of hypotension and pericardial effusions. They hypothesized that hypotension reduces pain because of low wall stress on the aorta. [14] Other authors speculated that slower dissection with less stretching causes no or less pain. [16,17] The sparing of the adventitial layer, the site of aortic innervation, may minimize pain. [16,18]

There are case reports that aortic dissections have been associated with different type of arrhythmias such as atrioventricular block, [19] atrial fibrillation, [20] and supraventricular tachycardia. [21]

Cardiac tamponade occurs in 20% of ascending aortic dissections and is the leading causes of death in patients with acute ascending aortic dissection, [9] with 54% of in-hospital mortality, which was more than double the mortality rate without cardiac tamponade. [22] Pericardiocentesis is the treatment of choice for cardiac tamponade caused by various underlying disease; [23] however, it is controversial in the context of aortic dissection. Some studies indicated that pericardial drainage and pericardiocentesis are contraindicated in case of tamponade complicated ascending aortic dissection, because a rebound increase in aortic pressure post evacuation may lead to recurrence of hemorrhage and recurrence of tamponade. [24,25] On the other hand, Hayashi et al reported that controlled pericardial drainage procedure produced better outcome. The total volume of drainage was approximately 40ml.[26] Based on that report, 2015 European Society of Cardiology guidelines recommended pericardiocentesis in the setting of aortic dissection with hemopericardium, with recommendation that controlled pericardial drainage of very small amounts of hemopericardium can help stabilize patients temporarily in order to maintain blood pressure at about 90mmHg [27]. The guidelines

emphasized controlled pericardial drainage of a very small amount, 40ml in the cited study. In our case, aortic dissection as the cause of pericardial tamponade was not recognized initially mainly because of patient's history of cancer. It was thought hemopericardium and widened mediastinum on CXR was secondary to metastatic malignancy. After drainage of a large amount of hemorrhagic fluid, patient hemodynamically improved without the recurrence of tamponade, which was unusual and it was due to sealed rupture as noted during surgical repair.

Acute hepatic dysfunction has been reported as presenting feature of acute pericardial tamponade secondary to aortic dissection, predominately resulted from right heart failure. There were 3 case reports that acute aortic dissection presenting with acute hepatic dysfunction by exhibiting dissection to the bifurcation or the aorta with obstruction of hepatic arterial flow secondary to extraluminal obstruction of the celiac and mesenteric arteries. All 3 patients died. [28,29,30] In Our case, MRI showed intact celiac trunk, although with dissection extending to descending aorta. (Figure 4) This indicated that the ischemic liver injury was not resulted from compromised hepatic flow, but directly from right heart which failed to expand at the pressure of tamponade.

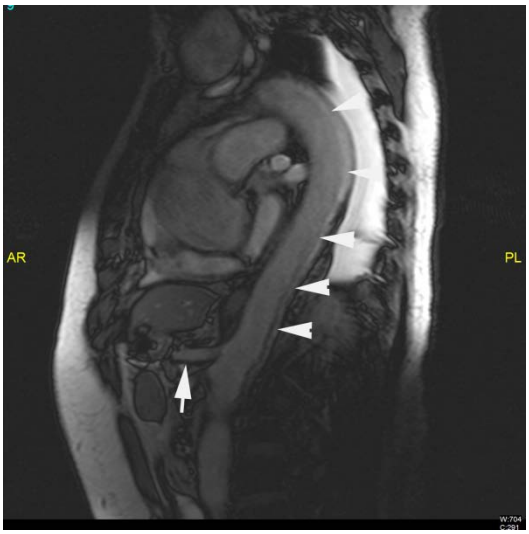

Figure 4. Sagittal MRI chest showing intact celiac trunk (white arrow); and dissection extending to descending aorta (white arrow head)

4. Conclusion

Aortic dissection is relatively rare but potentially fatal condition and can be painless. Atypical presentation can lead to delay in diagnosis and, as a result, delayed treatment and increased mortality. CT angiogram is one of the best imaging choice for diagnosis of aortic dissection; however, in patients with acute kidney insufficiency or contraindications for contrast its use may be limited. Cardiac tamponade is one of the complications of acute aortic dissection and leading cause of death in these patients and one of independent predictors of increased in-hospital mortality. Management of tamponade poses a real challenge to clinician. It may overshadow aortic dissection or clinician may omit exploration of the ascending aorta, which may not only [26] delay the diagnosis, regular pericardiocentesis may produce harm and lead to recurrent tamponade. We would like to emphasize through our case that in patients with hemorrhagic pericardial effusion, especially with large amount of fluid causing tamponade, a high index of suspicion of ascending aortic dissection is essential for prompt diagnosis and timely treatment to improve survival.

The uniqueness of this case lies in 1. Sealed rupture of dissection into pericardium; 2. Complete improvement in tamponade after complete drainage; 3. Survival of the patient to reach final decisive surgical repair; 4. This patient practically sustained most, if not all, potentially lethal complications of aortic dissection and yet survived.

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