

Chronic Pancreatitis with Spontaneously Disappearing Calcifications

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ABSTRACT

Pancreatic calcifications, exocrine insufficiency, and endocrine insufficiency are hallmarks of chronic pancreatitis, and their prevalence increases with the duration of disease. We present a case of chronic pancreatitis in which a dramatic and spontaneous decrease in the burden of both parenchymal and intraductal calcifications was noted during longitudinal follow-up. We discuss the possible reasons for spontaneously vanishing calcifications, an entity rarely described in the literature.

INTRODUCTION

Pancreatic calcifications, diabetes, and exocrine insufficiency are considered to be hallmarks of chronic pancreatitis. The prevalence of calcifications generally increases over time, and they can be observed on imaging studies in 70–90% of patients with chronic pancreatitis.^{1,2} Calcifications are often classified as either intraductal (ie, within the main pancreatic duct or branch ducts) or parenchymal (ie, within the pancreatic tissue, separated from ducts).³

A decrease in the number of pancreatic calcifications can occur after a drainage procedure, such as the Puestow procedure (lateral pancreaticojejunostomy).² One would also expect a decrease or disappearance of pancreatic ductal stones after successful endotherapy with or without extracorporeal shockwave lithotripsy. However, a spontaneous decrease in pancreatic calcifications has only rarely been described.^{4,5} More recently, although not spontaneous, the disappearance has been demonstrated to occur following intraductal citrate administration as well as after oral trimethadione administration.^{6,7}

CASE REPORT

A Caucasian woman without a significant past medical history was initially diagnosed with acute pancreatitis at the age of 19 years. After a workup, including genetic testing, her pancreatitis was attributed to a combination of alcohol and smoking; she had been abusing alcohol since the age of 9 and had been smoking 1–1.5 packs of cigarettes per day from the age of 15. She stopped drinking at age 23 but continued smoking. She first presented to our facility at the age of 27 years with epigastric pain, nausea, vomiting, and weight loss. She was found to have intraductal calculi and extensive coarse pancreatic parenchymal calcifications (Figure 1). She also had endocrine insufficiency (A1c 7.4%).

A Frey procedure was planned, but by the time of surgery the pancreatic duct was found to have spontaneously decompressed to 2–3 mm. Due to the presence of a dense adhesion between the stomach and the anterior surface of the pancreas, a pancreaticogastric fistula was suspected to be the reason for ductal decompression; however, a fistula was not visually confirmed during surgery. A fistulectomy and limited wedge posterior wall gastrectomy was performed, and pathology subsequently confirmed the presence of an intramural fistula. At that time, she also was found to have a splenic vein thrombosis and pseudocyst at the body–tail junction, with the latter resolving spontaneously after a few months.

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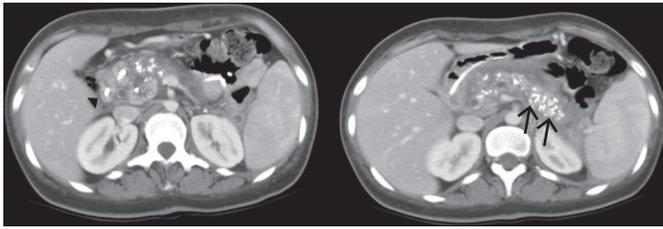


Figure 1. Contrast-enhanced axial computed tomography (CT) imaging from 2004 (age 27) showing intraductal calculi (arrow head) and extensive coarse pancreatic parenchymal calcifications (arrows).

Between the ages of 27 and 31, the patient's abdominal pain was well controlled with various opioid regimens, as documented during routine follow-ups in the pain clinic. During a hospitalization at age 32 for worsening abdominal pain, remarkably, a computed tomography scan showed dramatically reduced pancreatic calcifications (Figure 2), as well as chronic splenic vein thrombosis with perigastric collaterals, but no other local complications.

Between the ages of 31 and 40, despite decreasing smoking to one-half pack per day, the abdominal pain persisted with increasing opioid requirement, occasional exacerbations requiring hospitalizations, and worsening endocrine insufficiency (A1c peaking at 13.3%) and exocrine insufficiency ($<50 \mu\text{g}$ elastase/g stool). At age 35, magnetic resonance cholangiopancreatography revealed a ductal stricture in the pancreatic neck (Figure 3) with associated mild pancreatic duct dilation to 5.2 mm, and she underwent stricture dilation and placement of a dorsal duct stent (5 French \times 5 cm external pigtail stent). This did not alleviate her pain, and the stent was removed 1 month later. At age 38, endoscopic retrograde cholangiopancreatography for a rising alkaline phosphatase revealed a stricture in the ventral pancreatic duct in the head and lower third of the main bile duct. A 10-mm \times 4-cm covered metal stent (Figure 4) was placed in the distal common bile duct, with successful resolution of the stricture documented at the time of stent removal 4 months later. Computed tomography at age 39 for continued pain showed continued interval improvement of

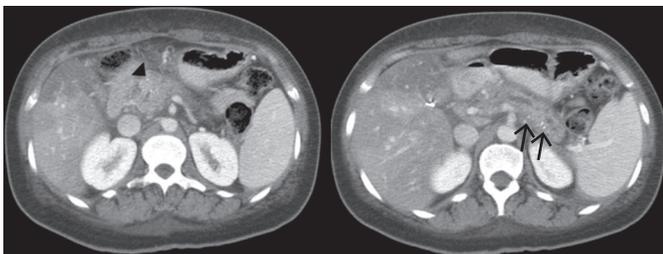


Figure 2. Contrast-enhanced axial CT imaging from 2008 (age 32) showing punctate intraductal calculi (arrow head) and few punctate pancreatic parenchymal calcifications (arrow).

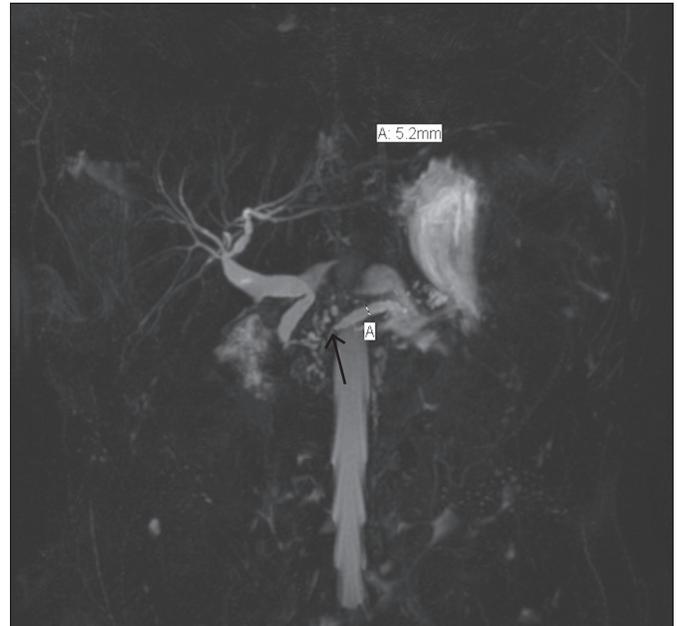


Figure 3. Contrast-enhanced coronal MRCP imaging from 2012 (age 35) demonstrating an abrupt termination of the pancreatic neck, consistent with a pancreatic neck stricture (arrow), with associated mild pancreatic duct dilation (A marker).

the calcifications, including disappearance of intraductal calcifications (Figure 5).

DISCUSSION

The disappearance of calcifications classically occurs in association with ductal drainage procedures, such as the Puestow procedure, either due to active removal of stones or postoperative passage of stones through pancreaticojejunostomy. Studies have also shown associations with intraductal citrate

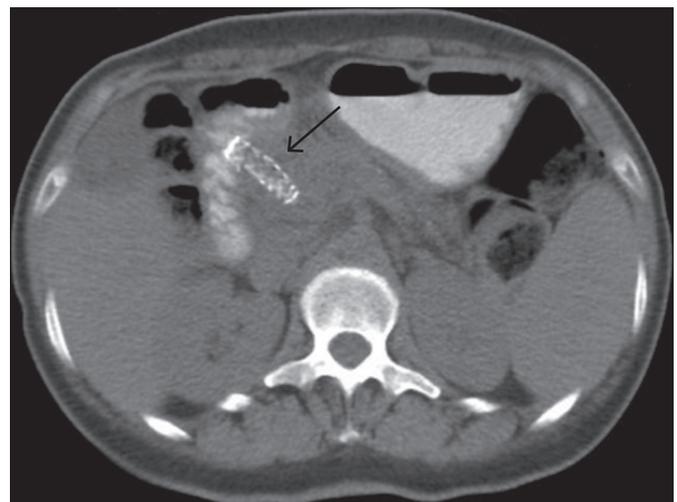


Figure 4. Contrast-enhanced axial CT imaging from 2015 (age 38) demonstrating a 10-mm \times 4-mm covered metal stent within distal common bile duct (arrow).

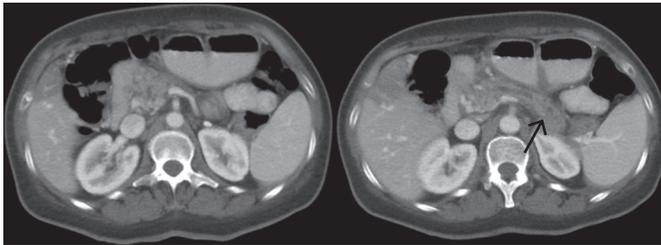


Figure 5. Contrast-enhanced axial CT imaging from 2016 (age 39) demonstrating scattered punctate pancreatic parenchymal calcifications (arrow) but no intraductal calculi.

administration as well as oral trimethadione administration.^{6,7} Spontaneous disappearances, as described in this case, are uncommon.

Although this patient did not undergo a surgical drainage procedure, it is possible that she underwent an “auto” ductal drainage via the suspected pancreaticogastric fistula; the fistula was seen at age 27 and the greatest decrease in calcifications occurred between the ages of 27 and 31. While endotherapy can also result in a reduction of pancreatic stones, it is important to note that endotherapy in this patient was only initiated after a dramatic decrease in calcifications had already occurred. An alternative possibility is that the stones dissolved via chemical dissolution. To our knowledge, the patient did not receive any pharmacologic agent capable of dissolving pancreatic stones; however, spontaneous dissolution of the calcium stones is theoretically possible in advanced chronic pancreatitis via decreased pH from reduced bicarbonate secretion.

It is also interesting that this case demonstrated a reduction of both parenchymal and intraductal calcifications, which has not been described in prior studies. It is worth noting, however, that prior studies of disappearing calcifications often did not use cross-sectional imaging. It is possible that the parenchymal calcifications were actually intraductal

calcifications, located within the small ductal branches deep within the tissue.

This case illustrates the fact that pancreatic calcifications, while a hallmark of chronic pancreatitis, may fluctuate in number and size and may in fact disappear in some patients spontaneously, independent of or with little correlation with disease activity.

DISCLOSURES

Author contributions: S. Friedberg wrote the article. A. Slivka, A. Dasyam, and D. Yadav edited the article. D. Yadav is the article guarantor.

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Informed consent was obtained for this case report.

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