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Case Report

A Rare Etiology of Gastrointestinal Bleeding: Polycystic Kidney Disease

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Abstract

The major extrarenal complications of autosomal dominant polycystic kidney disease (ADPKD) are aneurysms, hepatic and pancreatic cysts, cardiac valve disease, colonic diverticula, abdominal wall and inguinal hernia and seminal vesicle cysts. We aimed to present that a gastrointestinal bleeding caused by pseudoaneurysm of rupture of a gastroduodenal artery in a patient with ADPKD.

A 74 years old geriatric patient with history of ADPKD and chronic kidney disease (CKD) presented with gastrointestinal bleeding symptoms. The endoscopy and colonoscopy were performed after the patient was stabilized but we could not detect any bleeding focus. So that we planned computed tomographic angiography. There was pseudoaneurysmatic expansion and active extravasation from gastroduodenal artery (GDA). We also detected cortical renal cysts in kidneys reaching up to 5 cm compatible with ADPK. After computed tomographic angiography, the opening of the branch leading to extravasation was selectively catheterized Extravasation was not seen in control angiography. There was not hemoglobin decreasing and hemodialysis was continued routinely three times a week. After that the patient discharged.

We discussed that a life-threatening complication of ADPKD. This case is important due to see very rarely and being very mortally.

Introduction

Autosomal dominant polycystic kidney disease is a common disorder, occurring in approximately 1 in every 400 to 1000 live births [1-3]. It is estimated that less than one-half of these cases will be diagnosed during the patient's lifetime as the disease is often clinically silent [1]. The major extrarenal complications of ADPKD are aneurysms, hepatic and pancreatic cysts, cardiac valve disease, colonic diverticula, abdominal wall and inguinal hernia and seminal vesicle cysts [4-6]. An association between abdominal aortic aneurysms and ADPKD has been proposed [7]. Rupture of a gastroduodenal artery (GDA) pseudoaneurysm is an extremely rare and life-threatening condition [8]. Therefore, treatment of pseudoaneurysms is highly recommended because bleeding from pseudoaneurysms is associated with high mortality rates [9,10]. We aimed to present that a gastrointestinal bleeding caused by pseudoaneurysm of GDA in a patient with ADPKD.

Case

A 74 years old geriatric patient with history of ADPKD and chronic kidney disease (CKD) presented with hematemesis. In physical examination, arterial blood pressure was 90/60 mm/Hg, heart rate was 118 / min. There was no significal finding except melena with digital rectal examination. In laboratory, hemoglobin: 6.3 gr/dL, hematocrit: 21.6 %, platelet: 215000 / uL, urea: 218 mg/dL, creatinine: 2.5 mg/dl, potasium: 6.1 mmol/L and coagulation parameters were normal. Patient was

hospitalized to intensive care unite by gastrointestinal bleeding diagnosis and performed hemodialysis due to CKD related bleeding diathesis.

The endoscopy and colonoscopy were performed after the patient was stabilized by erythrocyte transfusion. We could not detect any bleeding focus. During the follow-up, hemoglobin levels decreased and the patient needed massive erythrocyte transfusion. Therefore, second endoscopy performed but no bleeding focus was detected again. So that we planned computed tomographic angiography due to investigate the bleeding focus. There was pseudoaneurysmatic expansion and active extravasation from a small branch of GDA (Figure 1).

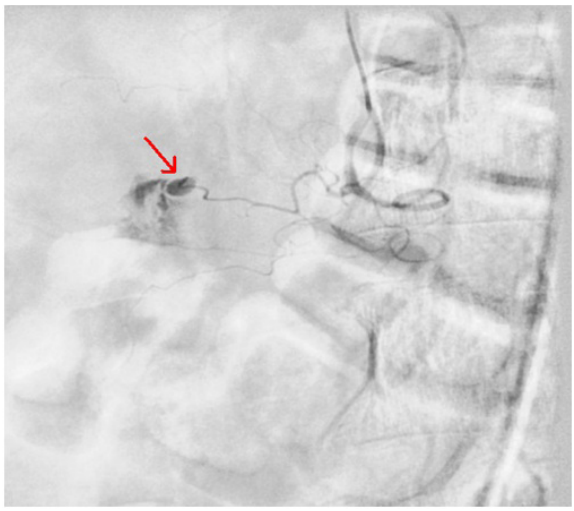

Figure 1: Extravasations marked with red arrow.

We also detected cortical renal cysts in kidneys reaching up to 5 cm compatible with ADPKD (Figure 2).

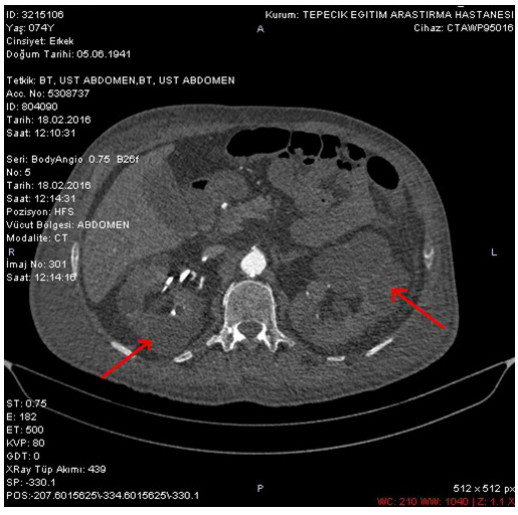

Figure 2: Bilateral renal cysts marked with red arrow.

After computed tomographic angiography, the opening of the branch leading to extravasations was selectively catheterized with a microcatheter, then the mixture of N-butyl cyanoacrylate and lipiodol were injected into the vessel. Extravasation was not seen in control angiography (Figure 3).

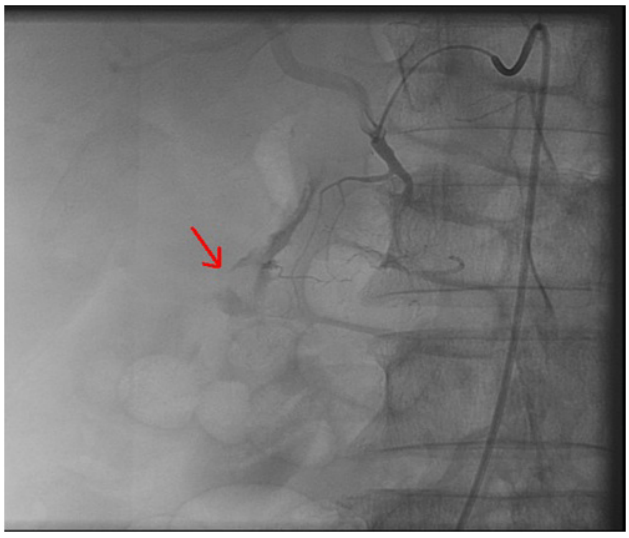

Figure 3: There is not extravasation in area marked with red arrow.

There was not hemoglobin decreasing and hemodialysis was continued routinely three times a week. After that the patient discharged.

Discussion

Autosomal dominant polycystic kidney disease (ADPKD) is characterized by cysts in the kidneys and, in many cases, is associated with cysts in the liver and pancreas that can be helpful in confirming the diagnosis. In addition, patients may have a variety of other abnormalities, many of which are consistent with a generalized defect in epithelial cell differentiation and/or extracellular matrix function as a primary expression of the genetic abnormality in this disorder [11-15]. Malformations of selected vasculature, including intracranial aneurysms and aortic root dilatation (normal diameter ≤ 35 mm), may be due to altered expression and/or function of the PKD gene in arterial smooth muscle cells [16]. One of the better studies evaluated the risk of recurrent aneurysm formation and/or an increase in size of an existing aneurysm among ADPKD patients with a history of either a ruptured or intact intracranial aneurysm. In this report, 20 ADPKD patients, 11 and 9 of whom had ruptured and intact intracranial aneurysms, respectively, were followed for a mean of approximately 15 years from the time of initial diagnosis. All of the patients with ruptured aneurysms and one with an intact aneurysm had undergone surgery at diagnosis [17]. Altough other antities as intracranial aneurysms more often, the visseral artery anevrism and rupture is very rare in literature. There is pseudoaneurysma in a small branch of GDA in our case but ruptures of pseudoaneurysms of the splenic (about 31%), gastroduodenal (about 24%), pancreaticoduodenal (about 21%), superior mesenteric, hepatic, or gastric arteries are reported with declining incidence [18,19].

The detection of an aneurysm mostly depends on CT or angiography [20]. CT should include an arterial phase with thin slices (1 mm) in axial and coronal plane. Pseudoaneuryms appear as sharply delineated lesion with homogenous and intense

arterial enhancement and an anastomosis to an artery. Angiography allows for a precise detection of bleeding arteries and pseudoaneurysms. As a result, the decision making, whether an embolization is technically feasible and safe, is facilitated [21,22]. In our case, CT angiography had performed and seen bleeding from pseudoaneurysma in a small branch of GDA. Therefore, we performed endovascular embolization percutaneously. Endovascular embolization has gained widespread acceptance for the optimal, initial treatment of unruptured or even of ruptured aneurysms because it is less invasive and more selective than traditional techniques. Compared with endovascular embolization of an aneurysm, the 'traditional' method of surgical clipping may be more invasive and difficult to accomplish in cases involving a large hematoma because the aneurysm itself can be missed during laparotomy [23-25].

Conclusions

We discussed that a life-threatening complication of ADPKD. This case is important due to see very rarely and being very mortally.

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