Document heading doi: 10.21276/apjhs.2016.3.4.23 Case report Polycystic Kidney Disease – A cause for Low backache

¹NS Neki, ²Amritpal Singh, ³Gagandeep Singh Shergill, ⁴Amanpreet Kaur

¹Professor, Dept. of Medicine, Govt. Medical College & Guru Nanak Dev Hospital, Amritsar, Punjab, India ²Senior Resident, , Dept. of Medicine, Govt. Medical College & Guru Nanak Dev Hospital, Amritsar, Punjab,

India

³Postgraduate Student, Dept. of Medicine, Govt. Medical College & Guru Nanak Dev Hospital, Amritsar, Punjab, India

⁴Consultant Gynaecologist, Civil Hospital, Fatehgarh Sahib, Punjab, India

ABSTRACT

Polycystic kidney disease is one of the common inherited diseases affecting the renal system, though it can be an acquired condition too. It can present as wide variety of symptoms and can also be incidental finding on abdominal imaging done for some other purpose. Polycystic kidney disease can be missed for a long duration if a strong suspicion is not there in the mind of physician based on the in detail knowledge of this condition. We here are presenting a short case report of a middle aged female who was having polycystic kidney disease but diagnosed considerably late.

Key words: Polycystic Kidney Disease, Low backache, Hypertension

Introduction

Polycystic kidney disease (PKD) is most common inherited disease affecting kidneys. Prevalence of PKD is estimated between 1 in 400 to 1 in 1,000 in general population[1]. Nearly half of the individuals go undetected during their lifetime [2], still 5-7% cases of end stage renal disease can be attributed to PKD[3]. Most of these patients with end stage renal disease require dialysis or renal transplantation in due course of the disease. PKD is of two types - autosomal dominant (ADPKD) and autosomal recessive (ARPKD). ADPKD is more common of the two with incidence of 1 in 8004 live births and is further divided into two types depending upon the gene involved. ADPKD-1 is caused by mutation in PKD-1 gene on chromosome 16 and account for 85-90% cases of ADPKD, mutations in PKD-2 on chromosome 4 accounts for rest 10-15%.4 ADPKD-1 is associated

Dr. N S Neki

with more severe disease manifesting clinically in first half of sixth decade of life as compared to ADPKD-2 which manifests after 70 years of age[5]. ARPKD is less common, incidence of 1 in 20,000 and causing death in fetal and neonatal period[4].PKD manifests with wide range of symptoms which may be due to cysts themselves and complications. As the cysts (and kidney) enlarge in size, they cause local pressure on the tissue and stretching of the renal capsule or traction of renal pedicle[6]. This leads to pain which may be felt in flank, in low back region centrally or on one side or as deep seated vague abdominal discomfort. Torsion of the cyst or any hemorrhage or infection in the cyst can also cause pain but this pain is usually acute and severe pain[7] and associated with other constitutional symptoms. PKD can manifest as hamaturia or albuminuria. Nephrolithiasis is also more common in ADPKD than general population, patient can present with symptoms from renal stones [8]. PKD is one of the etiology of hypertension, so all the symptomology of hypertension can be the first manifestation of PKD. Hypertension occurs in 3/5 of ADPKD patients and these patients are more prone to develop target organ damage[9]. Extrarenal manifestations from extrarenal features of PKD like cerebral aneurysms, cysts in liver, pancreas and other places can be the presenting complaints of PKD.

^{*}Correspondence

Professor, Dept. of Medicine, Govt. Medical College & Guru Nanak Dev Hospital, Amritsar, Punjab,India E Mail: drneki123@gmail.com

Case Report

A housewife of 45 years age presented to the outdoor section of Guru Nanak Dev Hospital and Govt. Medical College, Amritsar with chief complaints of low backache and headaches. To start with, she had low backache about one and half year back. The backache was like a continuous discomfort which was not severe enough to make her stop daily routine housework and it was nearly static since then. Only occasionally, she had to take brufen or some other painkiller to relieve pain on the suggestion of local practitioners. Once she was diagnosed to be having PIVD (prolapsed intervertebral prolapsed disc) despite a normal MRI scan and no history of any trauma. She was even prescribed lumbosacral brace. Then about two months back, she started having on and off headache. She took advice from some clinic which advised her to take painkillers when required but the headaches continued to come and go, she decided to get consultation in from some other practitioner where she was diagnosed to have migraine and put on flunarizine. But she was not relieved with the treatment, so she reported to us. She had no brother or sister and her parents had died. On examination, she was found to be hypertensive with blood pressure of 180/100 mmHg which was not known to her previously. Rest of the physical examination and laboratory tests including blood sugar, HbA1c, ECG, echocardiography showed no abnormality. High blood pressure with low backache, it was thought to rule out polycystic kidney disease and she was sent for ultrasonography of abdomen. Ultrasonograph revealed enlarged kidneys with multiple cysts in both the kidneys, with size of cysts ranging between 8mm to nearly 5cm in diameter (Pic. 1 and Pic. 2). Luckily, she had not any cyst in other abdominal organs. She was put on treatment for hypertension with ramipril and instructed to remain in followup. She was advised regarding avoidance of NSAIDs to relieve headaches and backache. On follow up, her blood pressure was under control and she had no headaches (which in all probability were due to high blood pressure).

Discussion

As seen in our patient, PKD as the diagnosis can be easily missed if all the symptoms of the patient and examination finding are not put in a proper algorithm with strong suspicion of PKD. Early detection of PKD is very important as it will enable the treating physician to keep under control the medical complications arising from PKD like hypertension, cardiovascular risk and nephrolithiasis. Strict control of hypertension from the start is very important as PKD patients will progress to end stage renal disease more rapidly. There are two criteria to diagnose the 'at risk' individuals with renal cvsts based on the ultrasonography findings. Ravine et al proposed presence of at least two renal cysts (unilaterally or bilaterally) for the age group 15-29 years, at least two cysts in each kidney for 30-59 years and at least four cysts in each kidney for age ≥ 60 years.10 But this criterion is often inaccurate for PKD-2 patients. For PKD-2 patients Pei et al criterion is superior.11 According to this criteria, for those with family history of ADPKD of unknown genotype diagnosis requires the following: in age group of 15-39 years, the presence of three or more unilateral or bilateral kidney cysts; in 40-59 years age group, two or more cysts in each kidney; and in individuals ≥ 60 years of age, at least four cysts in each kidney.[11] So in light of the above criteria, our patient was clearly in 'at risk' group who needs an active intervention. Blood pressure control goal in PKD patients is <140/90 mmHg with no sign of target organ damage and <130-135/80-85 mmHg in those with target organ damage[12]. More intensive control of blood pressure in range of 120-125/80 mmHg results in reduction of albuminuria and left ventricular hypertrophy[13]. For control of blood pressure in PKD patients, ACE inhibitors and ARBs are first line drugs as they act on RAAS (renninangiotension-aldosterone system) which is overactivated in PKD-associated hypertension. Betablockers can be considered after ACEIs and ARBs. Diuretics are second line drugs as they activate RAAS and can deteriorate the overall disease. Similarly Calcium channel blockers are also third line drugs, only to be used in resistant hypertension[14].

Conclusion

As complications of the disease lead to progression of target organ damage more rapidly in PKD patients than general population, early detection of PKD is important to control the progression of complications arising from the disease and to some extent the disease itself. Rigorous control of blood pressure, detection of extrarenal manifastations of PKD and detection & control of co-morbidities like diabetes in PKD patients can significantly decrease the end stage renal renal disease and cardiovascular complications.



Fig 1 & 2: Ultrasonograph revealed enlarged kidneys with multiple cysts in both the kidneys, with size of cysts ranging between 8mm to nearly 5cm in diameter

References

- 1. Torres VE, Grantham JJ. Cystic diseases of the kidney. In: Taal MW, ed. Brenner and Rector's The Kidney. 9th ed. Philadelphia: Saunders; 2011: 1626–67.
- 2. Davies F, Coles GA, Harper PS, Williams AJ, Evans C, Cochlin D. Polycystic kidney disease reevaluated: a population-based study. Q J Med. 1991;79:477-85.
- **3.** Steinman TI. Polycystic kidney disease: a 2011 update. Curr Opin Nephrol Hypertens. 2012 Mar; 21(2):189-94.
- 4. Wilson PD. Polycystic kidney disease. N Engl J Med. 2004;350 :151-64.
- 5. Hateboer N, Dijk MA, Bogdanova N, et al. Comparison of phenotypes of polycystic kidney disease types 1 and 2. European PKD1–PKD2 Study Group. Lancet. 1999;353 :103–107.
- 6. Seeman T, Dusek J, Vondrak K, et al. Renal concentrating capacity is linked to blood pressure in children with autosomal dominant polycystic kidney disease. Physiol Res. 2004;53: 629-34.
- Elzinga LW, Barry JM, Bennett WM. Surgical management of painful polycystic kidneys. Am J Kidney Dis. 1993;22:532-7.
- Torres VE, Wilson DM, Hattery RR, Segura JW. Renal stone disease in autosomal dominant polycystic kidney disease. Am J Kidney Dis. 1993;22:513-9.

Source of Support: Nil Conflict of Interest: None

- **9.** Ecder T, Schrier RW. Hypertension in autosomaldominant polycystic kidney disease: early occurrence and unique aspects. J Am Soc Nephrol. 2001;12:194–200.
- Ravine D, Gibson RN, Walker RG, Sheffield LJ, Kincaid-Smith P, Danks DM. Evaluation of ultrasonographic diagnostic criteria for autosomal dominant polycystic kidney disease 1. Lancet 1994; 343:824–27.
- **11.** Pei Y, Obaji J, Dupuis A, Paterson AD, Magistroni R, Dicks E, et al. Unified criteria for the ultrasonographic diagnosis of ADPKD. *J Am Soc Nephrol* 2009; 19:205–212.
- **12.** Mancia G, Laurent S, Agabiti-Rosei E, et al. Reappraisal of European guidelines on hypertension management: a European Society of Hypertension Task Force document. J Hypertens. 2009;27:2121–2158.
- **13.** Zeltner R, Poliak R, Stiasny B, et al. Renal and cardiac effects of antihypertensive treatment with ramipril vs metoprolol in autosomal dominant polycystic kidney disease. Nephrol Dial Transplant. 2008;23:573–579.
- Laia Sans-Atxer, Roser Torra, Patricia Fernández-Llama. Hypertension in autosomal-dominant polycystic kidney disease (ADPKD). Clin Kidney J. 2013; 6(5): 457–463.