Polycystic Kidney Disease

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INTRODUCTION

This nurse is currently employed at an outpatient ambulatory hemodialysis (HD) clinic. Nationally, statistics revealed more than 600,000 Americans being treated for end stage renal disease (ESRD) and at the state level, Ohio has 16,182 individuals who are on routine HD with the primary causes attributed to diabetes and hypertension (HTN) (National Kidney Foundation [NKF], 2017). This nurse discovered a small patient population ascertained ESRD from an intertwined blood disorder, particularly polycystic kidney disease (PKD). PKD is the fourth leading cause kidney failure and approximately 650,000 individuals have been diagnosed with PKD in the United States (NKF, 2017). PKD causes multiple fluid cysts to grow in the kidney, depleting the size and amount they can damage the kidney, decrease kidney function, and lead to renal failure (NKF, 2017).

There are three types of PKD: autosomal dominant polycystic kidney disease (ADPKD), autosomal recessive polycystic kidney disease (ARPKD), and acquired cystic kidney disease (ACKD). ADPKD, ARPKD, and ACKD are the most common inherited human renal diseases with mutations on two genes, PKD1 and PKD2, respectively. PKD1 accounts for 85% of ADPKD cases (Srivastava & Patel, 2014). The PKD1 gene is located on chromosome 16 and the PKD2 gene is located on chromosome 4.

Polycystic kidney disease (PKD) affects more than 100,000 people in the United States and more than 12 million worldwide. This disease is usually caused by mutations in two genes, PKD1 and PKD2. PKD1 accounts for 85% of ADPKD cases, while PKD2 accounts for 15%. These mutations lead to increased levels of cyclic adenosine monophosphate (cAMP), activation of protein kinase A, and an increase in sensitivity of collecting duct principal cells to the constant tonic effect of vasopressin. Approximately 650,000 individuals are diagnosed with PKD in the United States (NKF, 2017). This nurse discovered a small patient population ascertained ESRD from an intertwined blood disorder, particularly polycystic kidney disease (PKD). PKD is the fourth leading cause kidney failure and approximately 650,000 individuals have been diagnosed with PKD in the United States (NKF, 2017). PKD causes multiple fluid cysts to grow in the kidney, depleting the size and amount they can damage the kidney, decrease kidney function, and lead to renal failure (NKF, 2017).

SIGNIFICANCE OF PATHOPHYSIOLOGY

- Cyst development occurs from dilatations in intact tubules which are connected to nephrons.
- Increased cell proliferation and fluid secretion lead to cyst growth causing displacement and loss of the normal renal parenchyma.
- Increased cAMP decreases intracellular calcium signaling increasing the size of cysts by increasing fluid secretion.
- Improves renal function and leads to ESRD (Cronoe-Le Gall, et al., 2013).
- Renal cysts compress the renal vasculature resulting in intravascular ischemia activating the renin-angiotensin-aldosterone system (RAAS).
- Chronic pain caused by cyst rupturing or enlarged kidney compressing other structures (Ong, Devroy, Knellmehn, & Wals, 2015).
- Acute pain is associated with kidney cyst hemorrhage, infection, or stones.
- Urinary tract infections (UTI) are caused by infected renal cysts.
- Cysts block the renal tubules preventing normal drainage causing cysts to form and creating tumors.
- Hematuria is caused by the rupturing of cysts or of the small blood vessels in and around the cysts (Silverman, Dowst, & Lema, 2015).

SYMPTOMS

- Pain: can be present in back, chest, abdomen, or flank.
- Pain is reported in 60% of the cases of adults with ADPKD (Chethi & Torres, 2016).
- Hematuria: can occur when cysts rupture or small blood vessels in the kidney are injured. Hematuria is caused by the rupturing of cysts or of the small blood vessels in and around the cysts (Silverman, Dowst, & Lema, 2015).

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IMPLICATIONS FOR NURSING CARE

- Take patient history and perform assessment.
- Monitor vital signs, particularly blood pressure.
- Palpate bilaterally for flank masses (Trud & Humphregis, 2017).
- Monitor renal function and urine elimination, hydration, fluid and electrolyte balance.
- Provide fluids and foods based on the patient’s condition, encourage increased fluids if the patient has a urinary tract infection, and restrict fluids if the patient has renal failure.
- Assess for signs of systemic infection and sepsis.
- Encourage the child to have adequate fluid intake to maintain electrolyte balance.
- Refer the patient and his family to community and social services.

CONCLUSION

Although there is no treatable cure for ADPKD, understanding the mode of inheritance and the implications is paramount. Patients with PKD having a basic understanding how the disease is inherited and establishing an accurate family history can appreciate how conditions are passed on from a family and passed through generations. Managing the progression of the disease and the symptoms are incremental to slowing the growth of the cysts which may potentially lead to end stage renal disease (ESRD).

REFERENCES


