What is ADPKD?

Autosomal polycystic kidney disease (ADPKD) is the most common inherited condition that affects the kidneys(1). It is a dominant disease(2), meaning that only one dominant allele is needed for the patient to have the disease(3), because the dominant trait is always expressed as the recessive allele is masked by the dominant allele(3). This is why it is the most common inherited renal condition.

The faulty gene that dominates results in a faulty PKD1 or PKD2 gene, which are the two main genes involved in this disease, occur in 85% and 15% of patients suffering with this condition, respectively(1). ADPKD can be life-threatening, because the PKD1 gene encodes for polycystin-1(PC1) (4). A faulty PKD1 will lead to a reduction in PC1(5), which can lead to early development of cardiovascular disease (CVD) (6), the most common cause of mortality in humans(7). PC1 plays a vital role in mechanosensing, such as movement or pressure(19). PC1 is also crucial for modulating osteoblastic gene transcription, which is important for bone-cell formation(20). A mutation can, therefore, affect bone-cell formation. The mutation can result in 10% of cases where a family history is not present(52).

A reduction in PC1 can harm vascular endothelial cells, leading to endothelial dysfunction(8). Vascular smooth muscle cells can also be affected, with their functions being altered(9). These factors can increase the blood pressure (10, 11), leading to CVD. This is because endothelium-dependent vasorelaxation is impaired - with vascular endothelial cells, their reduction causes a reduction in the nitric oxide bioavailability, and therefore endothelium-dependent relaxation decreases(12), and with vascular smooth muscle cells, calcium ions are decreased, due to the inactivation of L-type calcium channels. MLCP then decreases, causing the impaired vasorelaxation(13). Increased blood pressure can then lead to damage to the endothelium of an artery (14). This can lead to atheroma formation, because there will be an LDL infiltration, as well as an inflammatory response, allowing foam cell and fatty streak formation. Plaque formation will result as a hardening of the atheroma. This causes reduced blood flow through the coronary artery, leading to heart diseases(14), which can lead to cells being deprived of oxygen, and therefore part of the coronary muscle dies (15). These cells may then undergo necrosis. A process of cell death due to pathological processes(16).

ADPKD occurs when there are many fluid-filled cysts that develop around the kidneys(17). The cysts develop when the cells lining the kidney tubules begin to grow at a rapid rate, resulting in a bulge, which separates from the tubule, and can fill with fluid over time, causing swelling of the cyst, **Figure 1** (18). This can affect the filtration of the kidney, because the cysts can lead to fibrosis, which can damage the nephron, and therefore reduce the kidneys' filtration rate(18).

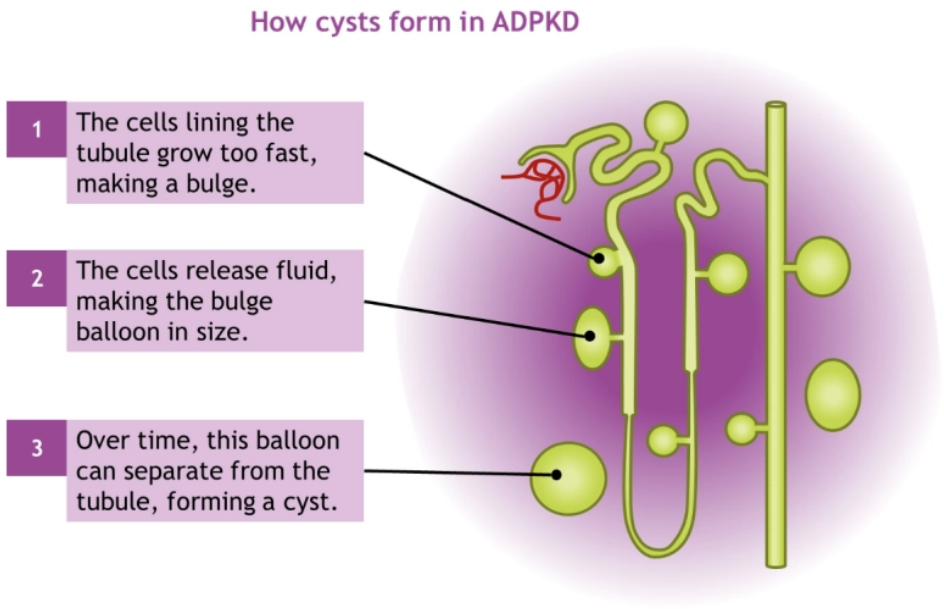

Figure 1 - The step-by-step process of cyst formation in kidney tubules (18).

Symptoms

Patients with ADPKD that is caused by the PKD1 faulty gene suffer with symptoms that are more severe than patients with the PKD2 faulty gene, for example, patients with the PKD1 mutations are more likely to suffer with hypertension than patients with the PKD2 mutation(21). This is because patients with the PKD1 mutation have larger kidneys than patients with the PKD2 mutation, and therefore, more clinically severe renal complications(22).

For patients with hypertension, antihypertensive drugs can be prescribed(23), for example ACE inhibitors, which are an excellent choice for patients with chronic kidney disease(24). ACE inhibitors decrease systemic vascular resistance(25), which is the resistance in the circulatory system which is used to create blood pressure(26), therefore decreasing the blood pressure. ACE inhibitors also tend to maintain glomerular filtration(27). Some side effects may include angioedema and a chronic dry cough, which can be followed by wheezing(27).

The Cure

Currently, there is no treatment that can fully inhibit cyst growth, and so ADPKD is currently incurable(28). Research is, however, still being conducted (29). For example, Mayo Clinic is conducting a study to discover biomarkers that are present in early stage ADPKD, meaning a broader knowledge of the disease can be obtained, so the biomarkers can better be targeted, and the possible treatment could be discovered(30).

Additionally, lifestyle changes can also help protect the kidneys for longer. For example, avoiding contact sports, such as rugby, to prevent the rupturing of the cysts, and losing weight, which can also lead to reduced proteinuria(31).

Tolvaptan may also be given to patients with ADPKD. This drug is proven to slow down kidney function decline(32). Tolvaptan blocks vasopressin V2 receptors(33), which has been directly linked to the regulation of cyst growth(34). A clinical trial with tolvaptan has been shown to reduce the kidney volume growth from 5.5% to 2.8%(34). However, some common side effects may include thirst, and a dry mouth(35).

When the kidney function of a patient can no longer function adequately on its own, the patient is diagnosed with end stage renal disease (ESDR)(36). A range of treatments can be used to provide sufficient filtration:

- Peritoneal dialysis(PD)
  The peritoneum lines the abdominal cavity. The peritoneal membrane has many tiny holes, making it porous, so waste products from the blood can be filtered(37). A catheter is placed into the peritoneal cavity(37). Clean dialysis fluid will be transported through the catheter(37). Peritonitis may occur, which is an infection of the peritoneal cavity(37). Prophylactic antibiotics should be administered prior to the insertion of the catheter(38).
- Kidney transplant
  Living-donor organ transplants have a significantly better survival rate than deceased-donor organ transplants(39). Even though both have a 90% survival rate after the first year, living-donor transplants drop to 80% after 5 years, whereas deceased donor-organs drop to about 65%(39). A major risk with kidney transplants is malignancy(40). This can be associated with immunosuppressants, because they decrease the immunological control of oncogenic viral infection and cancer immunosurveillance(40). A rare complication post-transplant can include renal vein thrombosis(41). Anticoagulants may be prescribed for the blood clot(42). If the thrombosis is due to an irreversible cause, life-long anticoagulants may be prescribed(42). Direct oral anticoagulants (DOACs) may be a form of anticoagulants prescribed. They inhibit thrombin(43), so the conversion of fibrinogen into fibrin will be inhibited (44), so blood clots will be inhibited. A side effect of this blood thinner is bleeding(45). This can especially occur when patients' kidneys or liver do not filter properly, so a higher concentration of DOACs builds up in the blood, so the doctor may prescribe a lower dose(45).
- Haemodialysis
  Before a patient undergoes haemodialysis, they might have to receive surgery for an arteriovenous fistula(46). During this surgical procedure, an artery is joined to a vein, most commonly on the arm(47). Aneurysms are complications of arteriovenous fistulae(48), which may be treated via an aneurysmectomy(49).
- Conservative treatment
  This is an alternative to dialysis for older patients with stage 5 chronic kidney disease and with multiple diseases(50). Conservative treatment is active medical management without dialysis, according to The Renal Physicians Association Shared Decision–Making Guideline(51).

To obtain further information about palliative care and conservative treatment, I was fortunate to be able to talk to Rebecca Owen – Clinical and Professional Development Facilitator. She explained how it was a challenge to manage the symptoms of patients with kidney diseases when pharmacological interventions are needed. She then added how the kidneys are involved in the metabolism and excretion of most drugs, and, with the kidney

function being so poor, there is a marked high risk of toxicity and sensitivity to some drugs, including side effects from the drugs being tolerated poorly by the person, emphasising that is was a real struggle to manage and control the pain adequately, without causing more harm to the patient. Especially in ADPKD patients, she found this to be an even bigger struggle due to the increased pain levels in patients who are under palliative care. Furthermore, she included how these challenges can be overcome with a mixed method approach, consisting of specialist knowledge around pain management, as well as complementary therapies and psychological support.This is all done via multiple healthcare professionals. Rebecca Owen then explained how these methods can be seen specifically in palliative care, because the main ethos is looking at the psychological holistic management of the person.

Linking Diabetes to Chronic Kidney Disease

Diabetes is the most common cause for CKD(54). This can lead to a reduction in the estimated glomerular filtration rate(eGFR)(55), because the blood vessels in the kidneys become damaged by the sugar over time(56). An early stage of diabetic nephropathy is a thickening of the glomerular basement membrane (GMB), followed by mesangial expansion, due to an increase in the mesangial matrix and an increase mesangial cell size, because of hypertrophy, causing a decrease in the filtration surface, leading to a reduced filtration rate(57). Due to the damage caused by sugar to the glomeruli, albuminuria may occur, which is when albumin leaks from the kidneys into the urine(58).

Budget Spent on Kidney Failure in the UK

Currently, 1 in 7 people in the UK live with chronic kidney disease, and therefore, the NHS spends roughly 3.2% of the funding on kidney care, which adds up to a total of roughly £7 billion per year(53). A high contribution to this cost is the cost of dialysis which is around £34,000 per patient per year(53).

How does AI help?

An AI tool was created based on an ensemble U-Net algorithm to measure the total kidney volume of patients(59). The ensemble U-Net algorithm, which can be used for medical image analysis(60), was created using the nnUNet approach(59), a learning-based segmentation method that automatically configures itself(61). The algorithm, which was trained by Principal Scientist Jonathan Taylor(62), used 454 kidneys and 227 scans, which came from 5 different MRI machines with strengths of 1.5T(59). At first, the kidney images were manually segmented by a single human operator (59), this allowed the AI tool to learn. The MRI scans that were used came from 4 European centres (from the CYSTic consortium)(59). The process of development, using the data from the MRI scans, can be seen in **Figure 2**.Some of the scans that were taken could not be used due to the fact that the kidneys were not in full view, or when the manual segmentations couldn't be drawn confidently(59).

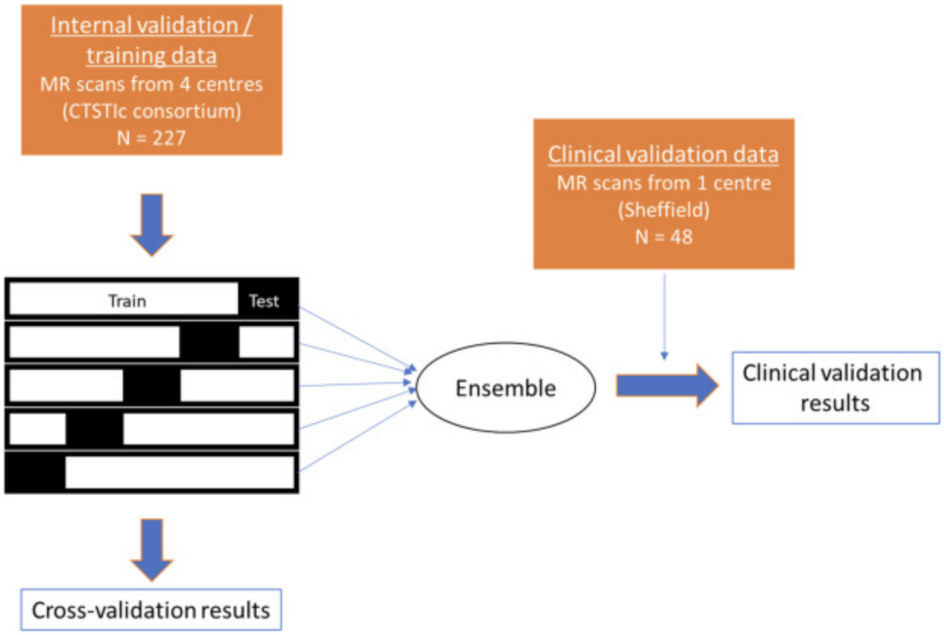

Figure 2 - The development of the algorithm, using the data produced by the MRI scans(59).

The AI tool can significantly reduce waiting times in hospitals, because the waiting time for a scan to be processed has been reduced from 1 hour to 9 minutes(59). The accuracy of the AI tool is also extremely high, with a DICE score of 0.96 on the clinical validation of the data produced by the AI tool(59).

The Dangers of AI

While there are many positives with the AI tool, it can also carry lots of negatives. Patients are fearing the loss of human connection in medical interactions(63). Patients also perceive that AI may overlook critical information, and lack the emotional intelligence required for effective care(63). Another concern includes the potential breach of patient data that is stored in the AI systems, leading to leaked patient health information(64). With AI developing for interpreting medical images, there is a risk of job displacement(64), for example radiologists.

Due to the fast development of AI, I wanted to discover a doctor's point of view. I am extremely grateful to be able to interview Dr Atif Khalil, a nephrologist at Noble's Hospital, Isle of Man. He discussed how AI might overestimate the progression of renal disease, such as ADPKD, so it may give the patient a false sense of confidence towards their kidney progression.

Further Kidney Discussions

Upon further discussions with Dr Atif Khalil, I discovered that the cysts can become calcified and lead to renal stones. He explained that this was because infections, caused by a build-up of fluid in blocked ducts, can raise the attenuation of the cyst contents, making it weaker, and induce dystrophic wall calcification, where the tissue becomes damaged. Dr Khalil then clarified that a process called percutaneous nephrolithotomy can be used to remove the renal stones, describing how it involved using a nephroscope (a thin telescopic instrument)

which is passed through a small incision in the patient's back and into the patient's kidney, which either pulls the stones out or breaks them using a laser. Dr Khalil also explained some of the side effects, including nausea, occasional vomiting and pain in the kidneys, abdomen, lower back and sides, within the first 24-48 hours.

Chloramphenicol and Bacterial Cyst Infections

Bacterial infections may occur in the cysts. A drug called Chloramphenicol can be used to treat this type of infection(65). Chloramphenicol acts on the 70S ribosome of bacterial cells and inhibits peptide bond formation by suppressing peptidyl transferase activity(66).

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