Computer Aided Diagnosis of Polycystic Kidney Disease Using ANN
Anjan Babu G, Sumana G, Rajasekhar M

Abstract—Many inherited diseases and non-hereditary disorders are common in the development of renal cystic diseases. Polycystic kidney disease (PKD) is a disorder developed within the kidneys in which grouping of cysts filled with water like fluid. PKD is responsible for 5-10% of end-stage renal failure treated by dialysis or transplantation. New experimental models, application of molecular biology techniques have provided new insights into the pathogenesis of PKD. Researchers are showing keen interest for developing an automated system by applying computer aided techniques for the diagnosis of diseases. In this paper a multilayered feed forward neural network with one hidden layer is constructed, trained and tested by applying back propagation learning rule for the diagnosis of PKD based on physical symptoms and test results of urinalysis collected from the individual patients. The data collected from 50 patients are used to train and test the network. Among these samples, 75% of the data used for training and remaining 25% of the data used for testing purpose. Further, this trained network is used to implement for new samples. The output results in normality and abnormality of the patient.

Keywords—Dialysis, Hereditary, Transplantation, Polycystic, Pathogenesis.

I. INTRODUCTION

POLYCYSTIC KIDNEY DISEASE (PKD) is a disorder in which clusters of cysts develop primarily within your kidneys. Cysts are noncancerous round sacs containing water-like fluid. The kidneys are two organs, each about the size of a fist, located in the upper part of a person's abdomen, toward the back. The function of the kidneys is to filter wastes and extra fluid from the blood to form urine and also regulate amounts of certain vital substances in the body. Cysts are filled with fluid when they form in the kidneys. PKD cysts can profoundly enlarge the kidneys which are out of normal structure, resulting in reduced kidney function and leading to kidney failure, the patient requires dialysis or kidney transplantation. About one half of people with the most common type of PKD progress to kidney failure also called end-stage renal disease (ESRD).

As the statistics says that as on December 2007, in the United States, about 600,000 people have cystic disease and it is the fourth leading cause of kidney failure.

Polycystic kidney disease is a common cause of kidney failure in Australia and equally affects men and women of different ethnic backgrounds. Men usually progress faster to kidney disease. There is currently no cure but the disease can be managed and research is ongoing for treatment options.

A. Forms of PKD [1], [2]

There are three forms of PKD: autosomal dominant PKD (ADPKD), autosomal recessive PKD (ARPKD) and Acquired Cystic Kidney Disease (ACKD).

Autosomal Dominant PKD is the most common form of the disease. It affects about 1 in every 1,000 births and symptoms usually appear in midlife. ADPKD follows a dominant inheritance, if either your mother or father has ADPKD, you will have a 50% chance of inheriting ADPKD. ADPKD is not a single disease. There are at least two genes that are associated with ADPKD. The Type 1 variant occurs when you inherit a defective copy of the PKD1 gene on chromosome 16. This form of disease accounts for about 85% of all cases of ADPKD. The Type 2 variant occurs when you inherit a defective copy of the PKD2 gene on chromosome 4. This form of the disease accounts for most of the remaining cases. Generally, the symptoms of both of these subtypes overlap completely.

Autosomal Recessive PKD is a very rare form of PKD causing symptoms in infants and young children. You may develop ARPKD if each of your parents carries at least one defective copy of the disease gene; in this case, you have a 25% chance of having the disease. If only one parent carries the defective gene, you cannot inherit the disease.

Acquired Cystic Kidney Disease occurs in children and adults, the cysts are more likely to develop in people who are on hemodialysis or peritoneal dialysis. The risk of developing Acquired Cystic Kidney Disease increases with the number of year’s people are on dialysis.

B. Causes [3]

Congenital Dysontogenesis or Genic Mutation: It is a giant cyst which has lead to the disarticulation and the restraint of the function of the two kidneys.

Toxins: Toxins can damage all kinds of cells, tissues and organs. Also it is one of the main reasons causing congenital dysontogenesis and genic mutation.

Infections: The common infections include: urinary tract infection, gastrointestinal tract infection, skin infection, upper respiratory tract infection, and trauma infection and so on can make the internal environment changes, for cyst genes to change causing the activity of inner factor of cysts will be increased, consequently the growth of cysts will be speeded up.

Anjan Babu G. is with Sri Venkateswara University, Tirupati, India (phone: +919959168462; e-mail: gababu.apps@gmail.com).
Sumana G. is with Sri Padmavathi Mahila Viswa Vidyalaya, Tirupati, India (phone: +919247873911; e-mail: sumanaspmvv@gmail.com).
Rajasekhar Mamilla is with Sri Venkateswara University, Tirupati, India (phone: +919347036765; e-mail: mamillarajasekharr@gmail.com).
Diet and lifestyle: Unreasonable eating habit, drinking alcohol and smoking can speed up gene expression and the growth of cysts. Too much psychological pressure or bad mood: Bad mood can decrease people's immunity, so that the body is easy to be attacked by germs or virus. If a person carries cyst gene, he may never know he is a potential PKD patient; different people have different speed of growth of cysts, which is associated with the diet, lifestyle, living habit and environment. These cysts decrease the function of a kidney to a certain degree; dialysis or a kidney transplant will be recommended. Fig. 1 shows Healthy Kidney and Fig. 2 is a gross photographic image of the Kidney with PKD.

C. Symptoms [4]

Physical Symptoms:
- Pain in the back and the sides (between the ribs and hips)
- Severe pain in abdomen, headaches
- Dark urine (hematuria) or Foamy urine due to excess protein (proteinuria)
- High blood pressure (hypertension)
- Fluid retention (edema) with swelling evident in your face, hands, feet and abdomen
- Urinary infections
- Fatigue from anemia or kidney failure

Clinical Symptoms [5]

Kidney Cysts: Many cysts which are noncancerous (benign) sacs contain water-like fluid develops in one or both kidneys, and as they accumulate more fluid they can grow extremely large and vary in size. Normally, a kidney weighs less than one-third of a pound (approximately three-quarters of a kilogram), while a kidney containing numerous cysts can weigh as much as 20 to 30 pounds (9.1 to 13.6 kilograms) gradually lose their ability to eliminate wastes from the blood substances and to maintain body’s balance of fluids and chemicals.

Pregnancy Complications: Pregnancy is successful for most women with polycystic kidney disease. In some cases, however, women may develop a life-threatening disorder called preeclampsia. Women who have high blood pressure before they become pregnant are at risk.

Growth of Cysts in the Liver: The likelihood of developing liver cysts with polycystic kidney disease increases with age. Both men and women develop cysts, where as women often develop larger cysts by female hormones.

Development of an Aneurysm in the Brain: Localized enlargement of an artery in your brain can cause bleeding (hemorrhage) if it ruptures. People with polycystic kidney disease have a higher risk of aneurysm, especially those older than age 50. If you have a family history of aneurysm or if you have uncontrolled high blood pressure then the risk is higher.

Heart Valve Abnormalities: As many as one-quarter of adults with polycystic kidney disease develop mitral valve prolapse. When this happens, the valve no longer closes properly; this allows blood to leak backward.

Colon Problems: Weaknesses and pouches or sacs in the wall of the colon (diverticulosis) may develop in people with polycystic kidney disease.

Chronic Pain: Pain is a common symptom for people with polycystic kidney disease. It often occurs in your side or back. The pain can also be associated with a urinary tract infection or a kidney stone.

II. CLINICAL APPROACH FOR THE DIAGNOSIS OF PKD [6]

The diagnostic methods are available to detect the size and number of kidney cysts and to evaluate the amount of healthy kidney tissue.

Urinanalysis: A protein urine test measures the amount of proteins, such as albumin, found in a urine sample.

Blood Test: A blood test to measure the level of protein or albumin.

Ultrasound Examination: A device called a transducer is placed on your body. It emits inaudible sound waves that are reflected back to the transducer. A computer translates the reflected sound waves into images of your kidneys.

Computerized Tomography (CT) Scan: The patient lies on a movable table, and guided into a big doughnut-shaped device that projects very thin X-ray beams through the body to see cross-sectional images of your kidneys.

Magnetic Resonance Imaging (MRI) Scan: The patient lies inside a large cylinder, magnetic fields and radio waves generate cross-sectional views of your kidneys.

Renal Ultrasounds: To check size and shape of the kidneys and to determine whether there are any blockages in the urinary tract.
III. COMPUTATIONAL APPROACH

A. Artificial Neural Network (ANN) [7]

Artificial Neural Network (ANN) is an information processing Technique based on the way biological nervous systems, process information. The fundamental concept of neural networks is the structure of the information processing system, composed of a large number of highly interconnected processing elements or neurons, a neural network system uses the human-like technique of learning by example to resolve problems. The neural network is configured for a specific application, such as data classification or pattern recognition, through a learning process called training. The purpose of training a neural network to perform a particular function by adjusting the values of the connections (weights) between elements. There are two types of training process: supervised and unsupervised training. Supervised training (e.g. multi-layer feed-forward (MLF) neural network) that knows the desired output and adjusting of weight coefficients is done in such way, that the calculated and desired outputs are approximately as close as possible. Unsupervised training means, that the desired output is not known, the system is provided with a group of facts (patterns) and then left to itself to settle down (or not) to a stable state in some number of iterations.

B. Multi-Layer Feed-Forward (MLF) Neural Networks [8], [9]

MLF neural networks, trained with a back-propagation learning algorithm. A MLF neural network consists of neurons that are ordered into layers (Fig. 3). The first layer is called the input layer, the last layer is called the output layer, and the layers between are hidden layers.

![Fig. 3 Typical Three layered feed-forward neural network](image)

The MLF neural network operates in two modes: training and prediction mode. For the training and prediction using the MLF neural network we need two data sets, the training set and predict set (test set). The training mode begins with arbitrary values of the weights - they might be random numbers – and proceeds iteratively. Each iteration of the complete training set is called an epoch. In each epoch the network adjusts the weights in the direction that reduces the error. As the iterative process of incremental adjustment continues, the weights gradually converge to the locally optimal set of values. Many epochs are usually required before training is completed. In back-propagation learning, we usually start with a training set and use the back-propagation algorithm to compute the synaptic weights of the network. A network is said to generalize well when the input-output relationship computed by network is correct (or nearly correct) for input/output patterns never used in training the network. When the learning process is repeated too many iterations (i.e. the neural network is over trained or over fitted), the network may memorize the training data and therefore be less able to generalize between similar input-output patterns.

C. Activation Functions [10]

Activation functions for the hidden units are needed to introduce nonlinearity into the network. Back propagation requires continuous, monotonic increasing activation functions, since these functions need to be differentiated when the gradient of the error surface is calculated during the weight update process.

The sigmoid function is desirable because of its simple derivative. The sigmoid function has the advantage of providing a form of automatic gain control. That is, for small signals (net near zero), the slope is steep producing high gain in the magnitude of the network's output and as the magnitude of net increases, the gain in the magnitude of the network's output decreases. In this way, large input signals can be accommodated by the network without saturation, while small signals are allowed to pass through without excessive attention [11]. The sigmoid function equation below is widely used as activation function and is a continuous function bounded in the range (0, 1).

The sigmoid function is expressed mathematically as follows:

\[ f(\text{net}) = \frac{1}{1+e^{-\text{net}}} \]

IV. CONSTRUCTION, TRAINING AND LEARNING OF ANN

A. Materials

The data required for this module is collected from the hospitals or from the Clinical Labs. The parameters that are used to diagnose the PKD are the Physical signs, symptoms, and readings that are obtained from the patients during physical examination and results obtained by urinalysis of the patient collected from the clinical labs. In the present study 50 samples are used. Each sample is having a set of 20 input nodes as given in the Table I and one output node. The input nodes represent the obtained physical, clinical evaluation features and the output node represents the diagnosis classification result in terms of abnormal or normal.
B. Construction of MLNN

A three layered feed forward neural network is constructed and Back Propagation Learning rules are applied. It consists of 20 input neurons in the input layer which is the Physical symptoms and the results obtained from the urinalysis collected from individuals, 10 hidden neurons in the hidden layer and one output in the output layer. The constructed architecture is as shown in Fig. 4. The network is trained using Back Propagation techniques. The activation function used in this model is the sigmoid logistic function. Once the network is trained, then it can be used to perform the diagnosis classification automatically for a new pattern.

C. Training and Learning of MLNN [12]

A neural network is built with 20 input nodes, 10 hidden nodes and one output node. The supervised network is trained using Back Propagation Learning techniques. The data collected from 50 patients are used to train and test the network. Among these samples, 75% of the data used for training and remaining 25% of the data are used for testing purposes. While testing the network, various data sets are applied to its input layer. Then the network generates the output which is compared with the desired outputs.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Attribute Name</th>
<th>Description</th>
<th>Allowed Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Age</td>
<td>Age of the Patient</td>
<td>Continuous</td>
</tr>
<tr>
<td>2</td>
<td>Gender</td>
<td>Gender of the Patient</td>
<td>Binary</td>
</tr>
<tr>
<td>3</td>
<td>Fatigue</td>
<td>Feeling very tiredness due to loss of blood</td>
<td>Binary</td>
</tr>
<tr>
<td>4</td>
<td>Nausea and vomiting</td>
<td>Vomiting sensation and uneasiness in the body</td>
<td>Binary</td>
</tr>
<tr>
<td>5</td>
<td>Hyper-tension</td>
<td>High blood pressure</td>
<td>Binary</td>
</tr>
<tr>
<td>6</td>
<td>Edema</td>
<td>Swelling of the body, especially noted in the face, hands, feet, and ankles</td>
<td>Binary</td>
</tr>
<tr>
<td>7</td>
<td>Loss of Appetite</td>
<td>Not willing to take food for longer period</td>
<td>Binary</td>
</tr>
<tr>
<td>8</td>
<td>Severe Back pain</td>
<td>Pain in the back and the sides (between the ribs and hips)</td>
<td>Binary</td>
</tr>
<tr>
<td>9</td>
<td>Abdomen Pain</td>
<td>Severe pain in lower part of the Abdomen</td>
<td>Binary</td>
</tr>
<tr>
<td>10</td>
<td>Headache</td>
<td>Partial Headache</td>
<td>Binary</td>
</tr>
<tr>
<td>11</td>
<td>Bloody Urine</td>
<td>Presence of RBC turn the urine reddish color</td>
<td>Binary</td>
</tr>
<tr>
<td>12</td>
<td>Urinary Infection</td>
<td>Severe pain during urination</td>
<td>Binary</td>
</tr>
<tr>
<td>13</td>
<td>Pyuria</td>
<td>Urine containing pus cells</td>
<td>Binary</td>
</tr>
<tr>
<td>14</td>
<td>Disturbed vision</td>
<td>Vision disturbance or giddiness</td>
<td>Binary</td>
</tr>
<tr>
<td>15</td>
<td>Diabetics</td>
<td>Abnormal sugar levels</td>
<td>Continuous</td>
</tr>
<tr>
<td>16</td>
<td>Weight gain</td>
<td>Abnormal gain of weight due to edema</td>
<td>Binary</td>
</tr>
<tr>
<td>17</td>
<td>Anemia</td>
<td>Less RBC count</td>
<td>Continuous</td>
</tr>
<tr>
<td>18</td>
<td>Create-nine</td>
<td>Excess creatinine levels</td>
<td>Continuous</td>
</tr>
<tr>
<td>19</td>
<td>Phosphorus</td>
<td>Excess Phosphorus levels in the urine</td>
<td>Continuous</td>
</tr>
<tr>
<td>20</td>
<td>Albumen</td>
<td>Excess albumen levels in the urine</td>
<td>Continuous</td>
</tr>
</tbody>
</table>

In the training process weights are adjusted till the target is reached. When the output result is matched with the original resultant with minimum error and then the training is stopped. The most iterative times were set to 1000 epochs, and the output error of the validation was set to less than 0.01. The output value is in between the range (0.0 to 1.0). If the obtained output value is near to 1.0 then the patient is normal person or the obtained output value is near to 0.0 then the person is with PKD. Once the Network is trained using these samples then it does the classification automatically for a new pattern. The Mean square error (MSE) is then calculated. The remaining 25% of the data is used for testing. The output results in normality and abnormality of the patient. The data collected from 50 patients are chosen randomly and are given as input data as shown in Table II, and resultant output data is compared with the actual resultant. Out of 50 Samples 31 samples were normal and the remaining 19 samples were diagnosed with PKD.

<table>
<thead>
<tr>
<th>Sample No.</th>
<th>Actual Result</th>
<th>Output Value</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>05</td>
<td>PKD</td>
<td>0.113</td>
<td>PKD</td>
</tr>
<tr>
<td>10</td>
<td>Normal</td>
<td>0.958</td>
<td>Normal</td>
</tr>
<tr>
<td>25</td>
<td>Normal</td>
<td>1.002</td>
<td>Normal</td>
</tr>
<tr>
<td>32</td>
<td>PKD</td>
<td>0.014</td>
<td>PKD</td>
</tr>
<tr>
<td>45</td>
<td>PKD</td>
<td>0.024</td>
<td>PKD</td>
</tr>
</tbody>
</table>

* PKD-Polycystic Kidney Disease

In overall training and testing only 10% of data is mismatched and the total accuracy is 90%. The result kept the accuracy in overall classification. The Mean Squared Error value decrease with the increase of the epoch values. The least MSE value of this approach is 0.010.
The graph plotted with computational results of each individual as shown in Fig. 5.

![Graph](image_url)

**Fig. 5** Graph plotted with training samples verses output result

**V. CONCLUSION**

Neural network with ability to learn by example has proven it is flexible and powerful decision support system for doctors in medical diagnosis. It proved that the experience from the expertise is not enough; by combining this opportunity with neural network the physicians can able to detect the patient disease at an early stages, there by having a chance for treatment otherwise at the later stages of PKD there is no treatment but only dialysis or the kidney transplantation should be recommended.

**REFERENCES**

[9] Introduction to multi-layer feed-forward neural networks by Daniel Svoził a, Vladimir KvasniEka b, JiE Pospichal b Received 15 October 1996; revised 25 February 1997; accepted 6 June 1997