

## Representation of Membranous Nephropathy- A Commentary

Muhammad Nabeel Ghayur\*

Department of Medicine, St. Joseph's Hospital, McMaster University, Ontario, Canada

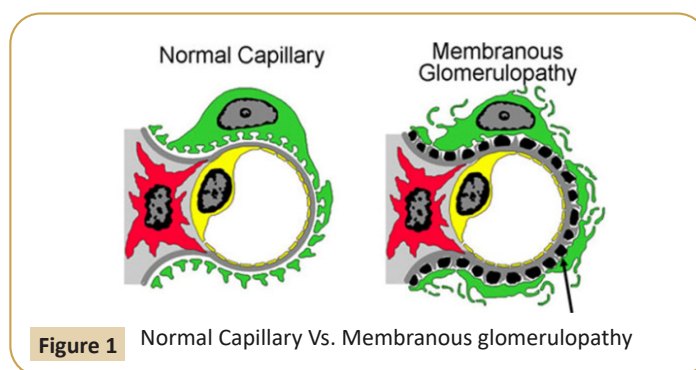
\*Corresponding author: Dr. Muhammad Nabeel Ghayur, Department of Medicine, St. Joseph's Hospital, McMaster University, Ontario, Canada, Email: nghayur@mcmaster.ca

**Keywords:** Obesity; Extent; Prevalence; Associated Factors; Uganda

### Commentry

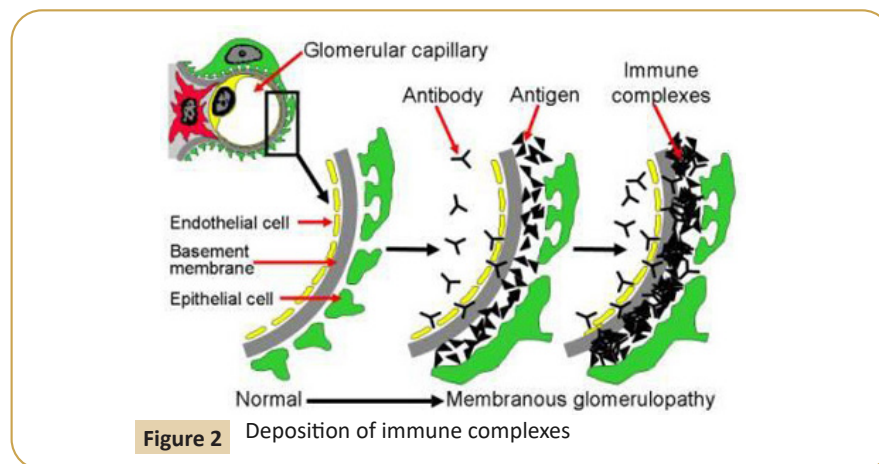
Membranous Nephropathy is a kidney disease that affects the kidney's philtres (glomeruli) and may cause urinary protein, as well as reduced function and swelling of the kidney. Membranous nephropathy can also be named as Membranous glomerulopathy. Membranous nephropathy in adults is one of the most frequent causes of nephrotic syndrome. Significant quantities of protein in the urine (at least 3.5 gm a day), low levels of blood protein (albumin) and swelling (edoema) include nephrotic syndrome. Membranous nephropathy may occur on its own (primary) or because of another illness or underlying cause (secondary). Membranous nephropathy is caused by the build-up of immune complexes in the philtres (glomeruli) of the kidney itself. The immune system usually produces antibodies to identify and bind to something (called an antigen). This is called an immune complex when an antibody binds to an antigen. Antigens, such as viruses or bacteria, are usually alien to the body. Often, however the body may create antibodies that identify and bind to anything not foreign) in the body itself.

These forms of antibodies are called auto-antibodies. Immune complexes are usually removed from the blood before causing any complications, but they can accumulate in various parts of the body under some circumstances. In membranous nephropathy, these immune complexes are trapped in the kidney philtres. In most cases of membranous nephropathy, the antigen that is part of the kidney philtre itself is formed by antibodies. These antibodies and antigens together produce immune complexes that get trapped in the philtre of the kidney and cause illness (Figure 1).

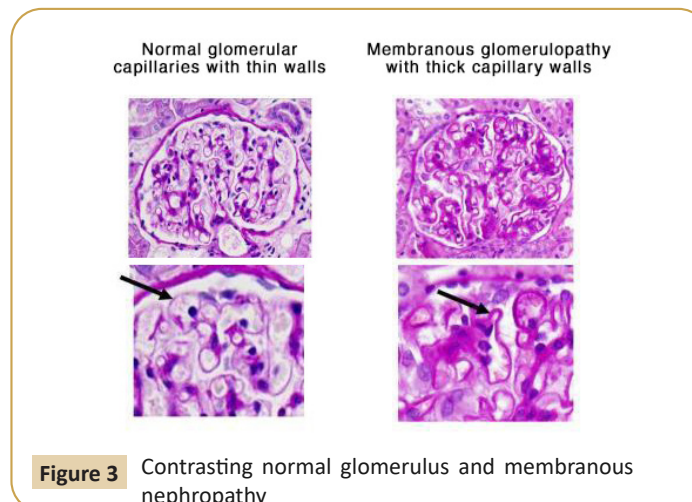


Recently, in about 70-80 percent of patients with primary membranous nephropathy, an antibody causing most cases of membranous nephropathy was detected and identified as anti-PLA2R is found in the kidney and/or bloodstream. The phospholipase A2 receptor (the antigen) binds to the anti-PLA2R antibody (short for anti-phospholipase A2 receptor antibody). A protein present in the kidney philtre is the phospholipase A2 receptor, specifically within a cell called the podocyte that makes up part of this philtre.

A diagram of how the immune complexes are deposited in the kidney is shown in Figure 2. A cross section of part of the kidney philtre is shown in the figure. This involves multiple layers, including the capillary blood vessel cells (endothelial cells, yellow), the basement membrane (grey), and the kidney cell layer (podocytes, green). Via these layers, blood within the capillary blood vessel is drained and becomes urine. In the blood stream, antibodies (Y-shaped, black in the image) bind to antigens (triangles, black in the image) and form immune complexes that get trapped and build up between the philtre layers. The immune system that induces inflammation is also activated by these immune complexes. As well as inflammation, the accumulation of these immune complexes causes the philtre to stop functioning properly which can lead to kidney damage. The philtre usually allows water, electrolytes and some waste materials to become urine, and bigger items such as blood cells and proteins are too big to move through the philtre, so that they remain in the blood. However, protein and blood cells will leak into the urine in this disease because the philtre does not function properly.



Part of a glomerulus is shown in Figure 3, contrasting a normal to one that is influenced by membranous nephropathy. On the right, collections of immune complexes (antigen-antibody complexes) are black spots or lumps (there is an arrow pointing to one). It gets thickened as more of these immune complexes build up between the layers of the philtres. The immune complexes and inflammation induced by the immune system harm the kidney cells that make up part of the philtre and stop functioning properly. You can see that the grey layer (basement membrane) has become thicker in the image on the right and has begun to fill in the gaps between the black spots/lumps. You can also see that the green cell does not look the same as on the left in the usual safe filtering loop (capillary loop).



The philtres in the kidney get thickened under a microscope, which is where the name of membranous nephropathy comes from. From 2 separate kidney biopsy samples, an example of this is shown below. These are cross sections, because the capillary blood vessels and philtres are cross sections of the loops. A typical glomerulus is on the left, and the loops in the glomerulus in someone with membranous nephropathy have become thickened on the right. The thickness of the capillary (small blood vessel wall) is pointed out by the black arrows in the photos. Note in a patient with membranous nephropathy (on the right) how much thicker this wall is.