

## Solutions to [Test Your Knowledge: Transplant Glomerulopathy](#)

### 1D. All of the above

Glomerular double contours are characteristic of transplant glomerulopathy and are due to chronic antibody mediated rejection. Similar glomerular changes can be seen in various forms of thrombotic microangiopathy, such as thrombotic microangiopathy due to calcineurin inhibitors, recurrent thrombotic microangiopathy or antiphospholipid antibody syndrome. Glomerular changes are also similar in hepatitis C virus infection–associated chronic glomerulonephritis. In chronic antibody mediated rejection, there is sustained or episodic endothelial injury affecting kidney microcirculation, [including the glomerular](#) and [peritubular capillaries](#). This leads to structural remodeling of the capillary wall.

### 2D. All of the above

The [clinical manifestations](#) of transplant glomerulopathy include progressive loss of kidney allograft function, proteinuria, and hypertension. Early transplant glomerulopathy may have mild subnephrotic range proteinuria and an unexplained drop in GFR. [A number of cases](#) of transplant glomerulopathy are subclinical and detected only in protocol biopsies.

### 3C. Perform an electron microscopic examination of the tissue sample

Characteristic light microscopic features of transplant glomerulopathy, such as glomerular capillary double contours and peritubular capillary basement membrane lamellation occurs over time, and in the early stages these findings can be focal and mild. Some of the earliest changes, such as swelling of the endothelium, mild subendothelial widening along the glomerular capillaries and the lamellation of the peritubular capillary basement membranes may not be visible under the light microscope; they are discernible only by [electron microscopy](#).

In sensitized patients who develop new onset or worsening proteinuria in the setting of de-novo donor specific antibody, it is important to consider chronic antibody mediated rejection manifesting in transplant glomerulopathy.

### 4B. There is no proven effective treatment for transplant glomerulopathy

There is no proven effective treatment for transplant glomerulopathy. Some uncontrolled studies show that early transplant glomerulopathy may be stabilized with high dose intravenous immunoglobulin, plasmapheresis, and/or anti-CD20. [Proteasome inhibitors](#) and eculizumab are being studied as potential agents to prevent development of [transplant glomerulopathy](#) in sensitized patients; however, some early evidence is not favorable.

### 5B. At 5 years, she has about a 30% less likelihood of having a functioning graft than someone without transplant glomerulopathy

Transplant glomerulopathy is a strong predictor for poor long-term kidney transplant survival. Data from the Mayo Clinic showed a 62% graft survival for allografts with transplant glomerulopathy as opposed to 95% graft survival without transplant glomerulopathy at [5-years post-transplant](#).