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## Acute antibody-mediated rejection of kidney allograft; mind the fibrin thrombi

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### *Implication for health policy/practice/research/medical education:*

Acute antibody-mediated endothelial damage and subsequent disseminated intravascular coagulation and thrombotic microangiopathy are life-threatening conditions that can affect not only graft but also the patient's survival significantly.

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### Acute antibody-mediated rejection of kidney allograft; mind the fibrin thrombi

A 45-year-old multiparous woman with chronic renal failure because of diabetic nephropathy and on hemodialysis for two years received her first cadaveric renal allograft. The blood group was compatible. She received induction therapy with rabbit anti-thymocyte globulin. Immediately after transplantation, her allograft started functioning well and she was discharged with a serum creatinine of 1.7 mg/dL. She was started on mycophenolate mofetil, tacrolimus, and prednisolone immunosuppressive therapies. During the second week of her discharge, she was re-admitted because of a sudden drop in urine output. The laboratory investigations revealed thrombocytopenia (platelets, <30 000/ $\mu$ L), elevated lactate dehydrogenase (>3000 U/l), anemia (hemoglobin; 6 g/dL), and impaired partial thromboplastin time, with international normalized ratio >3. She started bleeding from the venipuncture site and from vagina. Doppler ultrasound revealed preserved main renal artery and venous flow, however an absence of diastolic flow within the arcuate arteries. Laboratory examination revealed peripheral blood schistocytes and decreased fibrinogen level compatible with thrombotic

microangiopathy (TMA) and disseminated intravascular coagulation (DIC). Finally, she underwent a lifesaving nephrectomy.

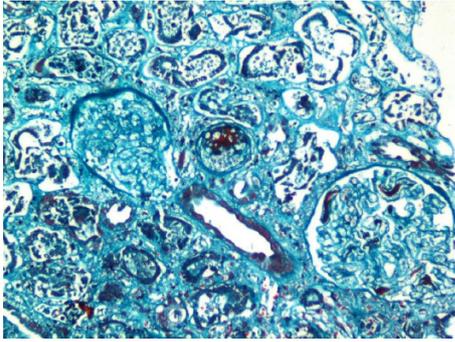
Acute antibody-mediated endothelial damage and subsequent TMA and DIC are life-threatening conditions that can affect not only graft but also the patient's survival significantly. A high index of suspicion should be exercised in cases of rapid graft shutdown in the very early post-transplant period. An aggressive multimodality approach could save the graft. Unfortunately, immediate allograft removal is reported in severe cases (1-3).

### Authors' contribution

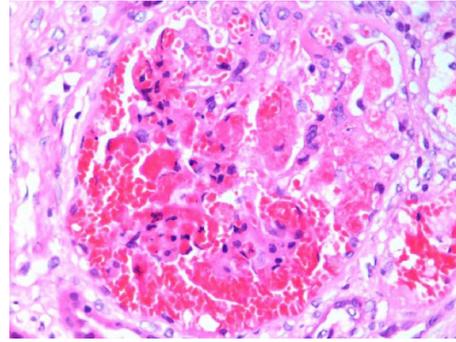
Conceptualization: MA. Methodology: MM, ShA and HRJ. Validation: DJ. Formal Analysis: MM. Investigation: MA. Resources: DJ and SS. Data Curation: DJ and SS. Writing—Original Draft Preparation: MA and SS. Writing—Review and Editing: DJ and SS. Visualization: DJ and SS. Supervision: MA. Project Administration: MA.

### Conflicts of interest

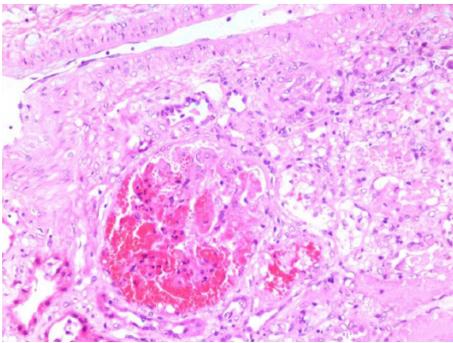
The authors declare that they have no conflicts of interest.



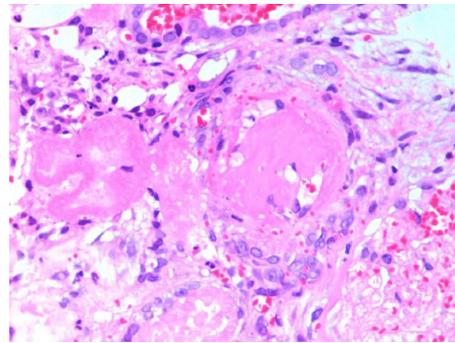
**Figure 1.** Low-power view showing two completely infarcted glomeruli. One of these contains fibrin thrombi. One arteriole also contains fibrin thrombus in the lumen. There is extensive acute tubular injury in the background. The above constellation of morphological findings is found in acute thrombotic microangiopathy (TMA), which may be caused by many insults, including alloimmune injury (Trichrome stain  $\times 100$ ).



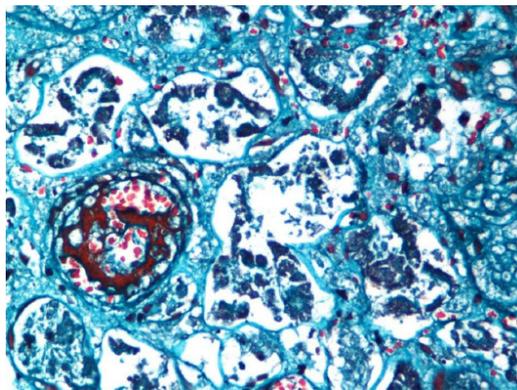
**Figure 3.** The same glomerulus as shown in Figure 2 at higher magnification. Complete occlusion of capillary lumens coupled with sledged red blood cells and fibrin thrombi is appreciated. A few neutrophils are noted (H&E stain  $\times 400$ ).



**Figure 2.** One glomerulus showing complete occlusion of capillary lumens with sledged red blood cells (RBCs) coupled with fibrin thrombi. The surrounding parenchyma shows severe acute tubular injury. RBCs can also be detected in the Bowman's space and one tubular lumen indicative of parenchymal hemorrhage. One interlobular size artery is included in the upper part of field and is unremarkable in this area (H&E stain  $\times 200$ ).



**Figure 4.** One small artery is seen in the center of the field with almost complete occlusion of lumen by fibrin thrombus. Many fragmented red blood cells can be detected in the wall of artery along with fibrinoid necrosis. An infarcted glomerulus can be visualized in the right lower corner of the field (H&E stain  $\times 400$ ).



**Figure 5.** OA small artery is demonstrated/stained, the lumen is filled with fibrin thrombus and admixed red blood cells. Fibrin stains red with trichrome stain. Severe acute tubular injury is seen in the background (Trichrome stain  $\times 400$ ).

**Ethical issues**

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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