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SUCCESSFUL SURGICAL CORRECTION OF ASCENDING AORTIC DISSECTION IN A KIDNEY TRANSPLANT PATIENT

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Cardiovascular disease is the leading cause of death in patients with a transplanted kidney and in graft loss. We present the first clinical case of successful surgical correction of ascending aortic dissection (DeBakey type I) in a young patient with a functioning kidney graft. The patient underwent the first cadaveric kidney transplantation (KTx), which was complicated by acute humoral rejection and suboptimal graft function. High blood pressure, anemia, elevated blood levels of triglycerides, phosphorus, parathyroid hormone, and uric acid were recorded. A repeat KTx was performed five years later; the patient's condition and kidney function were satisfactory. Three years later,the patient started experiencing severe pain along the thoracic and lumbar spine; his blood creatinine level was 408 μmol/L. Computed tomography and echocardiography diagnosed DeBakey type I aortic dissection (AD) with critical narrowing of the true aortic lumen at certain levels, dissection of aortic branches. Aortic resection surgery with prosthetic replacement of the ascending aorta according to David procedure with reimplantation of coronary artery orifices according to Kouchoukos technique, prosthetic replacement of the aortic arch with debranching of brachiocephalic artery and left common carotid artery were successfully performed as planned under endotracheal anesthesia, cardiopulmonary bypass and selective pharmacological cold cardioplegia. The peculiarities of the course, possible causes and outcomes of surgical correction of thoracic AD in the patient are discussed.

Keywords: kidney transplantation, vascular calcification, thoracic aortic dissection (DeBakey type I), prosthetic ascending aortic replacement.

INTRODUCTION

KTx is generally recognized as the best modality in renal replacement therapy to achieve satisfactory medical and social rehabilitation, improve the quality of life and increase the life expectancy of patients suffering from end-stage renal disease. Currently, about 1 million renal transplant recipients are registered in the world; they are more than 10,000 in Russia, and the figure is going up annually [1–3]. Cardiovascular diseases remain one of the leading causes of high mortality in kidney transplant patients and early kidney graft loss [4, 5].

Ascending AD (Stanford type A, DeBakey type I) occupies a special place in the structure of cardiovascular diseases. It is a rare, but very serious and dangerous condition, characterized by a high incidence of early fatal complications and mortality. There are very few population data on the prevalence of ascending AD and patient mortality from this disease. The disease is more common in older men, the leading risk factor is chronic high blood pressure [6, 7].

There has been information on proximal AD in patients with chronic kidney disease (CKD) in sporadic publications. It has been previously reported that AD has been found to be the most common cause of sudden cardiac death in dialysis patients [8]. A recent analysis by German researchers involving 14,911 patients presenting with acute type A aortic dissection for surgical intervention between 2006 and 2014 showed that 2,871 (19.3%) of them had CKD [6]. A just-published US study, which used a national database (US Renal Data System database), followed up 461 dialysis patients with type A aortic dissection from 1987 to 2015 [9].

There is even less information on surgical treatment outcomes, short- and long-term survival of patients with end-stage renal disease who underwent proximal aortic grafting, since there are very few such patients in large studies based on the Society of Thoracic Surgeons (STS) data [7, 10, 11]. Thus, according to Lee T.C. et al. [12], early mortality after surgical intervention for acute type A aortic dissection was 17.4%. International registry, which did not include patients with CKD, has documented a decrease in in-hospital surgical mortality from 25 to 18% in type A aortic dissection during the last two decades [13]. However, in the cohort of dialysis patients who underwent surgery for type A aortic dissection, perioperative mortality was 24.3% and 10-year mortality was 87.9 ± 2.2%. Age equal to or over 65 years, congestive heart failure and diabetes mellitus leading to end-stage renal disease were independent risk factors worsening the long-term outcomes of proximal aortic reconstruction [9].

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In our review of open access publications, we found no studies or descriptions of individual cases of ascending AD in patients with CKD who underwent renal transplantation. We present the first such clinical case: a young patient with a functioning renal transplant was diagnosed with DeBakey type I ascending AD; he underwent successful surgical correction.

CLINICAL CASE STUDY

First kidney transplantation

Patient A., 33 years old, has been observed at the kidney transplant department of Vladimirsky Moscow Regional Clinical Research Institute in Moscow since October, 2012. According to the patient, the family has no history of sudden death, thoracic aortic dissection/aneurysm. He has been overweight since adolescence. At the age of 22, proteinuria and increased blood pressure up to 170/100 mmHg were detected after a sore throat, and chronic glomerulonephritis was diagnosed (without histological confirmation). In the next three years, renal function deteriorated to the point of end-stage kidney failure. In the fall of 2012, the patient developed arteriovenous fistula on the left forearm and hemodialysis treatment was initiated. In the spring of the following year, a blood group and HLA-antigen-compatible cadaveric donor kidney was transplanted into the left iliac region. Early postoperative period had no complications, renal graft function was immediate. The patient was discharged three weeks later with the following readings: height 180 cm, weight 109 kg (BMI 33.6), blood pressure 125/80 mm Hg, creatinine 150 μmol/L, estimated glomerular filtration rate (eGFR) 53 mL/min, hemoglobin 101 g/L, albumin 40 g/L, cholesterol 4.5 mmol/L, glucose 5.6 mmol/L, triglycerides 3.0 mmol/L, uric acid 311 μmol/L, phosphorus 0.78 mmol/L, daily proteinuria 0.7 g. Three months later, he underwent surgery to close the arteriovenous fistula.

Deterioration of kidney graft function and increase in blood pressure to 140–150/100 mm Hg were observed since spring 2014. For this reason, repeated inpatient treatment was carried out: pulse methylprednisolone therapy, filtration plasmapheresis courses, nephroprotective and antihypertensive therapy. Renal graft function remained unstable (Table 1). Renal transplant biopsies were performed: report of May 21, 2014 – acute humoral rejection (BANFF2A); report of January 28, 2016 – focal global and segmental glomerulosclerosis with elements of collapsing nephritis, transplant glomerulonephritis (IgA nephropathy); report of October 17, 2017 – IgA nephropathy combined with chronic transplant glomerulopathy (predominance of C4d expression in the periphery of capillary loops). Blood pressure was maintained within the 130–150/80–90 mm Hg interval against the background of combined antihypertensive therapy. Dynamic laboratory examination revealed non-medically correctable slight anemia, as well as shifts in lipid and mineral-bone metabolism (Table 1).

Dynamic echocardiography (EchoCG) visualized dense dilated aorta, bigger left atrium and left ventricular myocardial hypertrophy (Table 2).

Repeat kidney transplantation

In April 2018, an arteriovenous fistula was formed in the lower third of the right forearm due to the progressive deterioration of the renal graft function; hemodialysis was resumed. Two months later, a cadaveric kidney was transplanted to the right iliac region and the first graft was removed. Kidney graft function was delayed, two hemodialysis sessions were performed. The patient's condition was satisfactory. A month later he was discharged for outpatient follow-up. Blood hemoglobin 90 g/L, creatinine 200 μmol/L, (eGFR 37 mL/min), uric acid 550 μmol/L, other biochemical parameters were within the reference interval, daily proteinuria

Table 1

Results of laboratory examination of patient A. after the first KTx

Indicator	First KTx on 26/03/2013						
	November 2013	May 2014	July 2015	January 2016	August 2017	November 2017	January 2018
Urea, mmol/L	9.2	10.7	10.3	20.0	12.2	20.9	13.7
Creatinine, μmol/L	140	170	240	210	270	310	420
eGFR, mL/min	58	46	30	35	26	22	15
Proteinuria, g/day.	0.6	1.5	0.6	2.7	4.6	8.0	10.8
Tacrolimus, ng/mL	7.1	6.4	6.6	6.1	5.3	4.9	8.2
Hemoglobin, g/L	107	99	104	103	108	98	89
Cholesterol, mmol/L	5.8	5.5	5.3	5.3	4.7	3.5	5.1
Triglycerides, mmol/L	2.5	3.2	2.7	3.1	2.6	2.9	2.2
Total calcium, mmol/L	2.53	2.69	2.33	2.42	2.49	2.23	2.26
Phosphorus, mmol/L	1.01	1.53	1.54	1.57	1.86	1.79	1.75
Uric acid, μmol/L	311	401	388	397	511	543	555
Parathyroid hormone, pg/mL	–	116	–	500	477	–	489

1.1 g. EchoCG: moderate left ventricular myocardial hypertrophy; left atrium enlargement; dilation and sclerotic changes in the ascending aorta (Table 5). Morphological examination of the removed graft: graft glomerulonephritis (IgA nephropathy); chronic graft arteriopathy; hyaline arteriolopathy; fibrous calcified atherosclerotic plaques in the renal artery.

In the next 2.5 years, the patient's condition remained satisfactory, renal graft function was stable, and there were no metabolic disorders against the background of medical correction (Table 3).

Diagnosis and surgical correction of DeBakey type I aortic dissection

The patient noted that his condition started deteriorating from early March 2021: increasing weakness, shortness of breath with little physical activity, lower appetite and 8 kg weight loss; from early April, he experienced severe and gradually increasing pain along the thoracic and lumbar spine with irradiation into the abdominal cavity, resistant to analgesics. The patient was admitted to the kidney transplant department of Vladimirsky Moscow Regional Clinical Research Institute on April 9, 2021 with 408 μmol/L creatinine.

Table 2

EchoCG results for patient A. before and after the first KTx

Heart chambers	First KTx on 26/03/2013				
	12/11/2012	12/04/2013	25/01/2015	1/07/2015	20/10/2017
Aorta	Dense	Dense	Dense	Dense	Dense
Aortic root diameter, cm	3.9	4.2	4.0	3.6	3.9
Ascending aortic diameter, cm	3.6	3.6	3.4	3.9	3.5
Aortic valve	Unchanged	Unchanged	Unchanged	Unchanged	Unchanged
Regurgitation	no	no	no	no	no
Left atrium, cm	4.3	4.2	4.0	3.9	4.1
Left ventricle					
EDD, cm	5.5	5.3	5.4	5.3	5.4
ESD, cm	3.8	3.1	3.2	3.3	3.2
EDV, mL	151	132	143	135	143
ESV, mL	61	37	40	62	40
EF, %	60	72	72	67	72
Right atrium, cm	3.7	2.1	2.1	2.1	2.1
Right ventricle, cm	3.7	2.8	2.8	2.8	2.8
Pulmonary artery, cm	2.7	1.9	2.7	2.8	2.5
Interventricular septum, cm	1.4	1.3	1.3	1.3	1.4
Mitral, tricuspid and pulmonary valves	Unchanged	Unchanged	Unchanged	Unchanged	Unchanged
Regurgitation	gr. 0–1 (+)	gr. 0–1 (+)	gr. 0–1 (+)	gr. 0–1 (+)	gr. 0–1 (+)

Note. EDD, end-diastolic dimension; ESD, end-systolic dimension; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction.

Table 3

Results of laboratory examination of patient A. after repeat KTx

Indicator	Repeat KTx on 04/07/2013			
	16/10/2018	12/08/2019	25/05/2020	10/03/2021
Urea, mmol/L	15.4	10.2	11.7	10.2
Creatinine, μmol/L	276	180	203	198
eGFR, mL/min	25	42	36	36
Proteinuria, g/day.	0.7	0.5	0	0.3
Tacrolimus, ng/mL	4.8	5.7	5.6	6.4
Hemoglobin, g/L	97	99	103	101
Cholesterol, mmol/L	4.2	5.4	4.4	4.1
Triglycerides, mmol/L	2.2	1.9	1.6	1.5
Total calcium, mmol/L	2.31	2.47	2.43	2.38
Phosphorus, mmol/L	1.91	1.47	1.23	1.16
Uric acid, μmol/L	550	401	381	366
Parathyroid hormone, pg/mL	193	153	147	–

On examination, he was in a state of moderate severity. Fully conscious and correct physique. Height 180 cm, weight 90 kg (BMI 27.8). The skin and visible mucous membranes were without features. There were no edemas. Body temperature was 36.7 °C. Respiratory and cardiovascular systems had no visible pathology. Respiratory rate was 19 breaths per minute. Heart rate was 80 per min. Blood pressure was 140/80 mm Hg. Systolic murmur over the apex of the heart. Clean and moist tongue. The abdomen was soft, painful in the epigastric and mesogastric regions. No peritoneal symptoms. Audible peristaltic murmurs. Normal stool. The liver and spleen were not palpated. Free urination. Kidney graft in the right iliac region, not enlarged, elastic, painless.

Laboratory examination revealed moderate anemia and drastically reduced renal graft function (Table 4).

The reasons for the severe pain and acute deterioration in graft function were not clear. The patient was referred for abdominal and thoracic computed tomography (CT). X-ray CT scan of the aorta with intravenous bolus contrast (Iomeron 400–99mm) was performed on a multislice CT scanner (256 slices) with ECG synchronization. Cardiomegaly (CTR 53%) was detected. There was fluid in the pericardial cavity with 10 mm layer thickness. The coronary arteries branch off projectionally from their respective sinuses. The aortic root diameter is 44 mm, the ascending aortic diameter is 44 mm, the aortic arch diameter is 37 mm, and the descending aortic diameter is 32 mm. DeBakey type I AD is detected (Fig. 1). The proximal border of the dissection is reliably visualized approximately 46 mm above the annulus level. The dissection extends further into the aorta throughout. There are no signs of intramural hematoma. The true lumen is narrow, about 6 mm wide at the level of origin of visceral branches. In the infrarenal region, there is local narrowing to a filiform level, then again about 6 mm wide. False lumen – maximum width of up to 33 mm in the ascending part and 28 mm in the descending part. No thrombotic masses are detected in the false lumen. The brachiocephalic arteries depart from the aortic arch typically, the left artery departs at the border of the true and false lumens. Transition of AD to the mouths of the left common carotid and subclavian arteries is not excluded. The right common subclavian artery branches depart from the true aortic lumen; the right common, external and internal subclavian arteries contrast homogeneously. The left common subclavian artery branches off from the true and false lumens, its dissection is visualized, its true lumen is narrowed to a critical level. The left internal subclavian artery branches off from the true lumen, the left external one – probably from the true and false lumens, its dissection and narrowing to the threadlike level of the true lumen are visualized, fenestration is detected in the distal part. Abdominal aortic branches: gastric and splenic trunk dissection and splenic artery are visualized; common hepatic artery has no signs of dissection. The superior and inferior mesenteric arteries and the right renal artery branch off from the true lumen, the left renal artery branches off from the false lumen. The artery to the renal graft is visualized from the right external iliac artery with irregular narrowing due to combined plaque (~50%). Conclusion.

Table 4

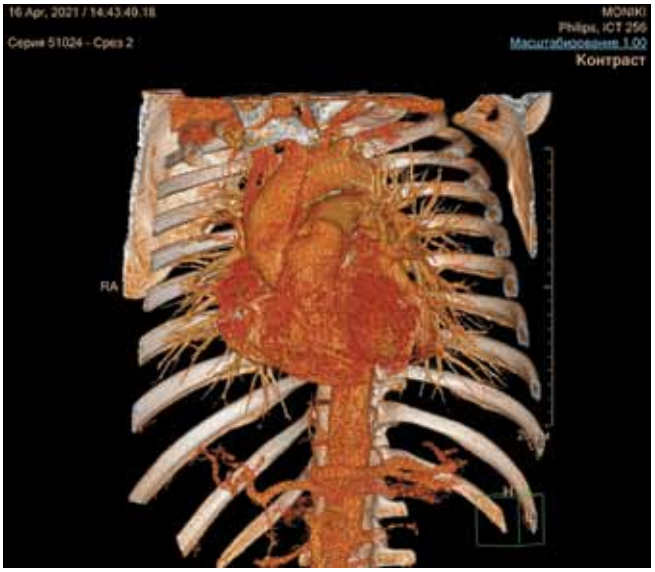
Results of laboratory examination of patient A. before and after surgical correction of DeBakey type I AD

Indicator	Prosthetic replacement of thoracic aorta on 1/06/2021		
	11/04/2021	26/05/2021	28/06/2021
Hemoglobin, g/L	91	105	98
Leukocytes, $\times 10^9$/L	7.8	12.2	8.4
Platelets, $\times 10^9$/L	480	338	370
Total bilirubin, μmol/L	7.3	13.7	16.2
Total protein, g/L	65	70	71
Albumin, g/L	39	32	39
Urea, mmol/L	25.6	15.1	11.1
Creatinine, μmol/L	462	218	168
eGFR, ml/min	14	32	46
Glucose, mmol/L	4.8	4.1	4.7
Potassium, mmol/L	4.5	4.8	4.6
Sodium, mmol/L	137	139	141
Total calcium, mmol/L	2.24	2.52	2.47
ALT, u/L	10	19	23
AST, u/L	15	37	30
Total cholesterol, mmol/L	2.7	2.8	2.9
Proteinuria, g/day	2.28	0.9	0.5
Tacrolimus, ng/mL	7.9	6.7	5.1
SARS-CoV-2 RNA	Not detected	Not detected	–

DeBakey type I AD with critical narrowing of the true aortic lumen at some levels, dissection of aortic branches. Variant of visceral branches originating from the aorta. Narrowing of the artery to the graft. Cardiomegaly.

Ascending AD was confirmed by echocardiography: visualized were dissecting aortic aneurysm (above the sinotubular junction to the visible part of the descending aorta), type 2 aortic insufficiency, dilated left heart chambers, left ventricular myocardial hypertrophy, moderate enlargement of the right heart chambers, dilated pulmonary artery trunk and branches (Table 5).

So, after CT and EchoCG findings, the patient was diagnosed with DeBakey type I AD with critical narrowing of the true aortic lumen at certain levels, dissection of aortic branches. The case conference decided that the patient be transferred to the cardiac surgery department for surgical treatment – aortic prosthesis. On June 1, 2021, under endotracheal anesthesia, cardiopulmonary bypass and selective pharmacological cold cardioplegia (Custodiol solution), the patient underwent aortic resection surgery with prosthetic replacement of the ascending aorta with VASCUTEK Gelweave 30 mm according to David procedure, with reimplantation of coronary artery orifices according to Kouchoukos technique, prosthetic replacement of the aortic arch with multibranched vascular graft VASCUTEK Gelweave 28 mm with debranching of the brachiocephalic artery and left common carotid artery. The surgery lasted for 375 minutes, cardiopulmonary pass and circulatory arrest of internal organs lasted for 197 and 20 minutes respectively. Intraoperative hypothermia was 30–26 °C, cerebral perfusion was bispherical. Histological examination of removed aortic segment: a fragment of muscular-elastic artery, lipid spots in the intima, fibrous atherosclerotic plaques; dissection at the medial layer level with formation of false lumen; focal fragmentation of elastic fibers in the area of dissection.



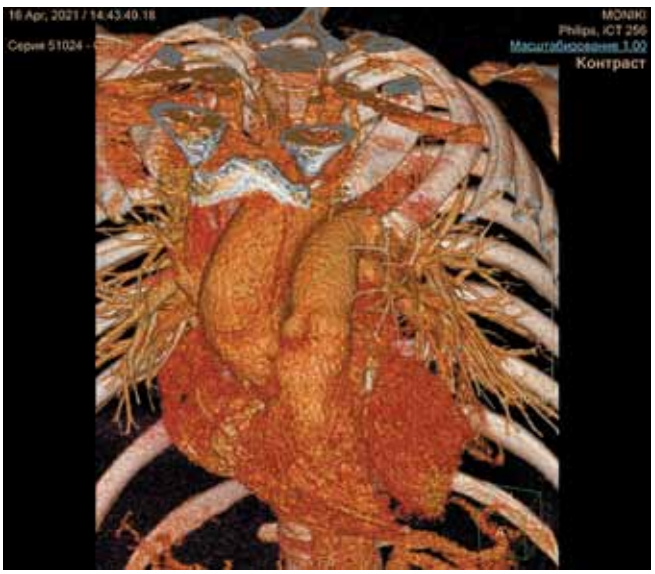


Fig. 1. Preoperative computerized tomography images in kidney transplant recipient with type I aortic dissection

After the operation, the patient was transferred to the intensive care unit, and the next morning, artificial ventilation was stopped (postoperative artificial ventilation lasted for 14 hours). The patient's condition remained stable. On day 7 after the operation, the chest X-ray and CT scans revealed pericarditis and bilateral hydrothorax. Drainage of the right pleural cavity removed 600 mL of hemorrhagic fluid. Due to hemorrhagic discharge continuing through the drainage, the patient underwent urgent resternotomy, revision and sanitation of the pericardial cavity (100 mL of liquid blood and 300 mL of blood clots were removed) and pleural cavities (150 mL in the left, 100 mL of serous hemorrhagic fluid in the right); the source of bleeding was not identified. On day 15 after operation, the patient was transferred in a satisfactory condition to the cardiac surgical department. Control transthoracic EchoCG three weeks after the operation showed decreased size of the left heart and aortic regurgitation (Table 5). Non-contrast-enhanced thoracic and abdominal CT scans: the prosthetic ascending aorta area is homogeneous; a dissection extending to the descending aorta is identified, the left common and external iliac arteries, without dynamics as compared with the previous study, was determined from the aortic arch level (Fig. 2). Kidney graft function remained stable (Table 4). One month after surgery, the patient was discharged for outpatient follow-up with a recommendation for dynamic monitoring and subsequent surgical correction of the descending AD.

The patient was examined on month 3 after surgery. Results of laboratory blood tests: Hemoglobin 115 g/L, urea 10.7 mmol/L, creatinine 190 μmol/L (eGFR 40 mL/min), uric acid 539 μmol/L, albumin 44 g/L, total bilirubin 16.3 μmol/L, total cholesterol 3.1 mmol/L, triglycerides 1.4 mmol/L, C-reactive protein 2.8 mg/L, phosphorus 1.53 mmol/L, other electrolytes and enzymes within

the reference interval, tacrolimus 6.1 ng/mL, no proteinuria. Thoracic and abdominal CT scans: the prosthetic ascending aorta area is homogeneous; with contrast enhancement, the release of the drug beyond the prosthetic aorta is not determined; dissection from the aortic arch level persists, spreading further to the descending aorta; the left common and external iliac arteries, without dynamics as compared with the previous study (Fig. 3). Recommendation of the cardiac surgeon: re-examination and hospitalization for surgical endovascular correction of the abdominal AD in 3–4 months' time.

DISCUSSION

Analyzing the patient's medical history, we paid attention to some peculiarities of its course. In our observation, thoracic AD developed in the young man, whereas such patients are much older both in the general population and in the population with end-stage renal disease. For instance, in the German study mentioned earlier, the mean age of patients with thoracic AD was 58.3 years, and the mean age of dialysis patients who underwent ascending aortic grafting was 64 years (53–73) [6, 9]. It is known that arterial hypertension is the most common predisposing factor for thoracic AD, but in our patient, it was hardly the only factor responsible for this condition. The patient also had no family history of sudden death and thoracic AD, detected in 20–40% of such cases, although he was not subjected to genetic testing [14, 15].

It appears that vascular calcification – intimal (atherosclerosis) and medial (arteriosclerosis) – was the main pathophysiological process leading to dissection of the aorta and its branches in this patient. Simultaneous coexistence of both variants of vascular calcification in patients with CKD before and after KTx is real, but a more characteristic variant, a "specific feature" is the development of medial calcinosis. Factors that procalcify the arterial wall in CKD include male gender, high blood pressure, obesity, long-term dialysis treatment, metabolic disorders, shifts in mineral and bone metabolism, local and systemic inflammation, oxidative stress, uremic toxin

Table 5

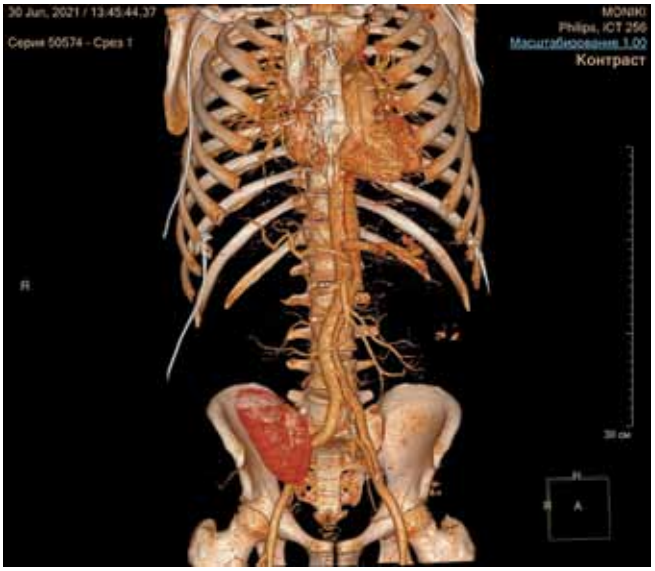
EchoCG results for patient A. before and after surgical correction of DeBakey type I AD

Heart chambers	Prosthetic replacement of thoracic aorta on 1/06/2021		
	12/09/2018	27/04/2021	23/06/2021
Aorta	dense		
Aortic root diameter	4.2 cm	4.5 cm	2.5 cm
Sinotubular junction diameter	–		
Ascending aortic diameter	4.0 cm	3.5 cm 4.7 cm (false lumen 3.0 cm)	3.0 cm (prosthetic part)
Aortic arch diameter	–	3.4 cm	
Descending aortic diameter	–	3.6 cm. Above the sinotubular junction, dissection of the aortic wall up to the visible part of the descending part is detected; a double contour is not clearly visualized in the area of origin of the left carotid artery	
Aortic valve			
Annulus diameter	–	24 mm	24 mm
Regurgitation	gr. 0–1 (+)	gr. 2 (+)	gr. 1–1.5 (+)
Left atrium	4.4 cm, volume 69 mL	4.2 cm, volume 74 mL	3.7 cm
Left ventricle	Hypertrophy	Hypertrophy	Hypertrophy 17 mm
EDD	5.6 cm	5.4 cm	4.7 cm
ESD	3.7 cm	3.1 cm	3.8 cm
EDV	152 mL	141 mL	127 mL
ESV	58 mL	40 mL	45 mL
EF	61%	71%	61%
Right atrium	44 mL	65 mL	46 mL
Right ventricle	31 mm	37 mm	30 mm
Pulmonary artery	Trunk 26 mm	Trunk 34 mm, branches 20 mm	Trunk 28 mm
Interventricular septum	13 mm; LVPW 12 mm	18 mm; LVPW 15 mm	17 mm; LVPW 16 mm
Mitral, tricuspid and pulmonary valves	Unchanged	Unchanged	Unchanged
Regurgitation	gr. 0–1 (+)	gr. 1–1.5 (+)	gr. 0–1 (+)

Note. EDD, end-diastolic dimension; ESD, end-systolic dimension; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; LVPW, left ventricular posterior wall.

accumulation and many others [16–19]. Interesting data on the possible role of uric acid in vascular calcification has been obtained [20]. Vascular calcification formed at the phase of end-stage renal failure is irreversible. After successful KTx, despite partial or complete resolution of the metabolic disorders inherent in CKD, recipients continue to be exposed to some procalcifying stimuli that contribute to progression of pre-existing vascular calcification or its development de novo [18, 21]. In particular, new post-transplantation risk factors for vascular calcification include immunosuppressive therapy with direct and mediated effect on the vascular wall through carbohydrate and lipid metabolism. Another potential factor in the post-transplant period is the often-diagnosed magnesium deficiency associated with inhibition of its tubular reabsorption under the influence of calcineurin inhibitors [22]. Finally, reduced renal graft function is a significant risk factor for vascular calcification [23, 24]. Maréchal S. et al. [25] observed 197 renal transplant recipients for 4.4 years and found a 4%-per-year increase in aortic calcification. In their study, risk factors for its development/progression were aortic calcification before KTx, high pulse pressure, older age, serum phosphorus, male gender, statin and aspirin therapy. A group of Italian researchers, performing multiple regression analysis, found the following predictors for aortic calcification in kidney transplant patients: body mass index, serum magnesium, age, systolic blood pressure, proteinuria, and dialysis duration [23].

Everything described above is applicable to our patient – after KTx, he had all of the above, as well as a number of risk factors atherosclerosis and arteriosclerosis. The duration of chronic renal failure, including the pre-dialysis phase and dialysis therapy, was short; there



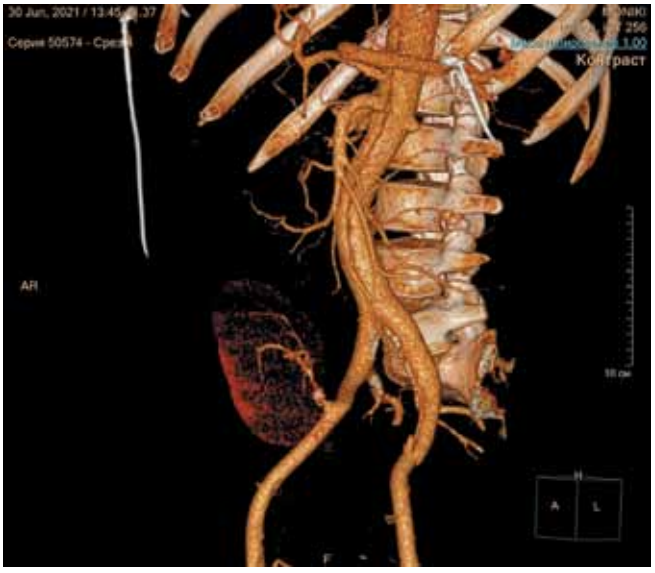
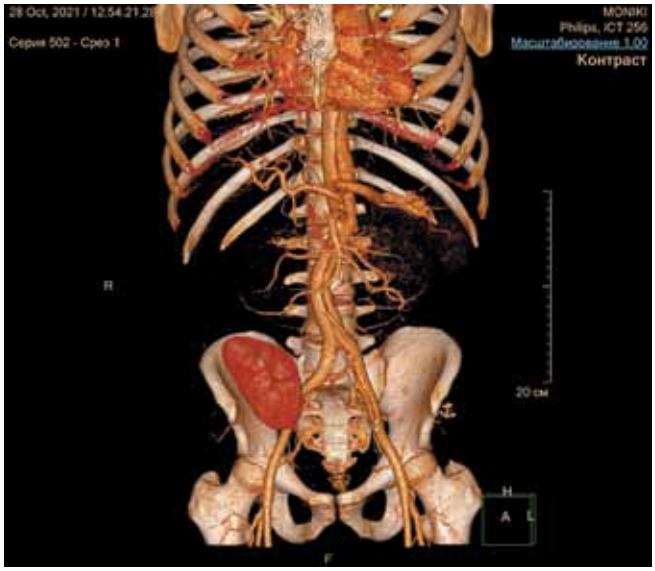


Fig. 2. Postoperative computerized tomography images in kidney transplant recipient with type I aortic dissection



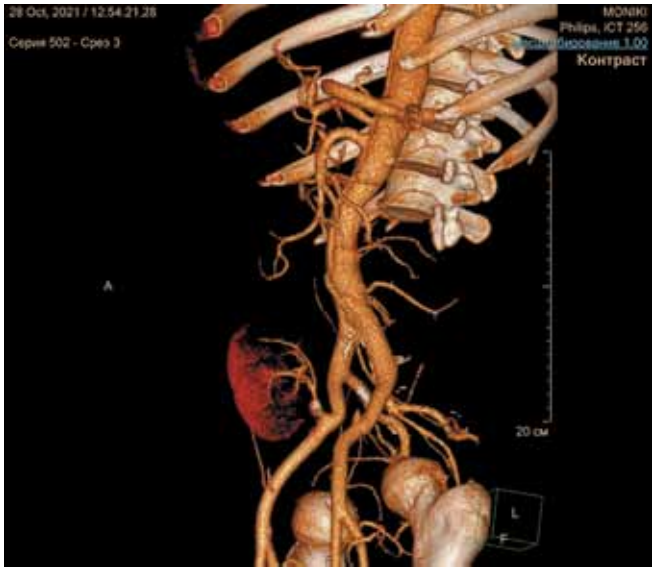


Fig. 3. Computerized tomography images in kidney transplant recipient with type I aortic dissection 3 months after intervention

is no information about the presence of vascular calcification during that period. But it is known that the patient remained overweight for several years before and after KTx. There were high levels of triglycerides in the blood immediately after the first KTx and then for five years until the second KTx. Acute humoral rejection diagnosed one year after the operation, which required pulse methylprednisolone therapy, further reduction in kidney graft function and persistent increasing proteinuria contributed to the aggravation of arterial hypertension and appearance, in addition to hypertriglyceridemia, other metabolic anomalies – hyperphosphatemia, hyperuricemia, and increased parathyroid hormone levels. It is thought that hyperphosphatemia, which was observed for several years after the first KTx, is of the greatest importance in the development/progression of aortic calcification. Currently, high phosphorus levels are considered the main, unique inducer of vascular (medial) calcification in CKD, which is either passively deposited from extracellular fluid into the vascular wall in the form of calcium phosphate, or promotes the acquisition of an osteoblast-like phenotype by vascular smooth muscle cells [26]. All disorders diagnosed in this patient (obesity, long-term suboptimal renal graft function, multiple endocrine and metabolic disorders) most likely, were the pathogenetic mechanisms contributing to the progression, if the process appeared from the onset of CKD, or to the formation of de novo calcification of the aorta and its branches, which, in turn, caused their dissection. The existence of widespread atherosclerosis in the patient was convincingly confirmed by histological examination of the renal artery of the first graft, the removed section of the thoracic aorta, as well as by CT scan of the renal artery of the second graft. Known structural features of the root and ascending aortic wall could also be important for the formation of dissection in this area [27].

The outcome of surgical treatment of thoracic AD in our patient deserves a separate discussion. Surgery is presently the optimal solution for proximal AD, significantly reducing mortality [7]. Aortic root reimplantation surgery with preservation of the native aortic valve was proposed by David T.E. and Feindel C.M. 30 years ago. It now represents a generally accepted and widely used surgical option for proximal aortic aneurysm/dissection, which avoids many early and long-term postoperative complications [28, 29]. In the general population, perioperative mortality and long-term survival rates in proximal aortic grafting are quite optimistic [30, 31].

A group of German surgeons, having summarized more than 25 years of experience of such surgery in 732 patients with proximal aortic root aneurysm and dissection, convincingly demonstrated its high safety and low risk of perioperative complications and mortality. The authors also confirmed the excellent long-term outcomes of David's procedure and stable aortic valve function in the majority of patients [32].

In dialysis patients, both perioperative and long-term mortality in proximal aortic grafting were very high, which, apparently, is due to kidney failure and chronic dialysis with associated various shifts in homeostasis [9]. However, high perioperative and long-term mortality should not serve as a reason to delay urgent lifesaving intervention for acute type A aortic dissection in a patient with end-stage renal disease. There is no information about the immediate and long-term outcomes of surgical treatment of AD in kidney transplant recipients. It is known that, in general, patient survival after KTx is much better than in the dialysis population, and if a dialysis patient underwent KTx after aortic grafting, his life expectancy was longer than that of those who remained on dialysis [9].

In our patient, a complex operation to replace the ascending aorta and aortic arch, despite early postoperative complications (right-sided hydrothorax, pericarditis), which required repeated surgical intervention, ended successfully. Apparently, young age, relatively small aortic diameter, absence of severe heart failure, diabetes mellitus and serious homeostatic disorders with a satisfactorily functioning re-transplanted kidney contributed to a good surgical outcome.

CONCLUSION

Patients with CKD represent a high-risk population for cardiovascular disease. Thoracic aortic dissection/aneurysm is a rare vascular condition that nevertheless requires special clinical vigilance due to high incidence of fatal complications. Vascular calcification, which is common in CKD, is a predisposing factor for thoracic aortic dissection/aneurysm. Surgical intervention – aortic root grafting – is the treatment of choice for proximal AD, although immediate and long-term outcomes in patients with end-stage renal disease are not as optimistic as in the general population.

This present clinical case has demonstrated the successful outcome of scheduled surgical correction of proximal AD in a patient with end-stage renal disease, who has undergone two kidney transplants. The presence of comorbidity in CKD patients before and after renal transplantation requires a cautious approach to the management tactics for proximal AD, proper, careful selection and preparation of such patients, especially for a scheduled grafting.

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