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Clinocopathological Conference Report

Pelviureteric Necrosis–Rare Reason for Graft Loss in Simultaneous Pancreatic–Kidney Transplant

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Case history

A 23-year-old male admitted for simultaneous pancreas–kidney transplant

Background History

A 23-year-old male was admitted for simultaneous pancreas–kidney transplant. The patient had type 1 diabetes mellitus for the past 10 years and was on insulin therapy. He had developed end-stage renal disease (ESRD) due to diabetes. One year before transplant, continuous ambulatory peritoneal dialysis (CAPD) catheter was inserted for ESRD and he was on peritoneal dialysis and listed for combined pancreas–kidney transplant. Combined pancreas–kidney transplant was planned for him and was on the waiting list.

Present Admission

A 23-year-old male on CAPD for ESRD was admitted for simultaneous pancreas–kidney transplant planned for his end-stage kidney disease due type I diabetes mellitus. The deceased donor was available, and preoperative clearance from brain dead committees was available. Simultaneous pancreas–kidney (SPK) transplant was performed on dopamine infusion > total operative time was 7 hour. Dopamine infusion was subsequently tapered off and he was extubated after 6 hour. His intra-abdominal pressure (50 mm Hg) was recorded to be high and after 8 hours of operation he developed high blood pressure (170/108 mm Hg) which was managed by nitroglycerine drip. He developed hypotension after few hours (12 hours post-op); consequently, nitroglycerine drip was stopped, and dopamine infusion was restarted. On a postoperative day 1, he had oliguria followed by anuria, for which he received two sessions of hemodialysis. He had fever and bicytopenia (thrombocytopenia and neutropenia) on day-2 of transplant with persistent hypotension and increased intra-abdominal pressure. He was investigated (Tables 1 to 4) and infection and DIC was excluded. He was given crystalloids, inotropes (dobutamine, adrenaline) and antibiotics (meropenem, piperacillin–tazobactam) without response. He was intubated and put on SIMV ventilator mode. Pre-terminally, he had metabolic acidosis, leukopenia, and thrombocytopenia. The patient developed cardiorespiratory arrest on 16/01/2016 at 5.30 pm from which he could not be revived.

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Investigations

Preoperative (14/1/2016)

- *CAPD fluid cytology:* acellular
- *Culture:* sterile

Postoperative

- *Echocardiography:* Normal ejection fraction, no right wall motion abnormality
- *Imaging*
- *Chest X-ray: Normal*

Course and Management

Case Analysis

So we have a 23-year-male, Type I diabetic on insulin therapy who had developed CKD and was on CAPD, awaiting transplant surgery

Table 1: Postoperative biochemistry

	15/1 (post-operative)	*15/1 4 PM*	*16/1 12 AM*	*16/1 10 AM*	*16/1 (Pre-terminal)*
Hemoglobin (g/dL)	12.1	14.6		13.9	12.2
Platelets (/mm^3)	111	79		64	37
TLC (/mm^3)	4100	4000		4000	700
Na (mmol/L)	142	141	139	137	–
K(mmol/L)	4.7	4.5	3.9	5.5	–
Cr(mg/dL)	7.6	8.28	6.5	6.9	–
Urea(mg/dL)	98	93	71	76	–
Amylase(U/L)	174	154	194	134	–
Lipase(U/L)	–	145	–	–	–
AST(U/L)	45	54	–	–	–
ALT(U/L)	18	20	–	–	–

was taken up for simultaneous pancreas–kidney transplant. He had raised intra-abdominal pressure with high blood pressure 6 hours after postoperative. Later he developed hypotension, oliguria, and anuria. On day 2 of post-transplant, he had a fever, bicytopenia with persistent hypotension and raised intra-abdominal pressure. Pre-terminally he developed metabolic acidosis.

The surgical complications which are considered include vascular leak and abdominal compartmental syndrome. Raised intra-abdominal pressure with pain and organ dysfunction, absence of hemorrhagic abdominal drain and fall in hemoglobin favors abdominal compartmental syndrome.

The cause for allograft kidney dysfunction is acute tubular injury due to renal artery stenosis, sepsis, intra-abdominal hypertension and postoperative hypotension leading or reperfusion injury (< 4 hours) or rejection. Renal artery stenosis is more likely which occurs most frequently within the first six months after kidney transplant and can present with worsening hypertension, fluid retention, and allograft dysfunction. However, still, rejection cannot be ruled out.

The cause for pancreatic graft failure can be due to allograft reperfusion pancreatitis, allograft pancreatic vein thrombosis or rejection. Absence of raised amylase and lipase with detectable C peptide levels, Cold ischemia time of less than 8 hours and normal vascular Doppler have ruled out reperfusion pancreatitis and pancreatic vein thrombosis.

Table 2: Postoperative coagulogram

	15/1	*16/1*
PT (sec)	18	21
PTI (%)	72%	62%
INR	1.37	1.61
APTT (sec)	28	–
D-dimer	–	–

The cause of death is more likely sepsis because of the presence of fever, bicytopenia, and decompensated shock. Sepsis due to bacterial infection is more likely than fungal infections.

Final Clinical Diagnosis

- Type 1 diabetes mellitus,
- Chronic kidney disease (end stage renal disease),
- Post simultaneous pancreas-kidney transplant–
  - Early graft kidney dysfunction
  - Acute graft pancreatitis

Sepsis

- *Cause of death:* Abdominal compartmental syndrome, sepsis

Clinical discussion

- **Chairperson (Prof. Subhash Varma**): Thank you Ashutosh. Please join me here. Anybody from the treating unit would like to make a comment
- **Dr. Deepesh**: Good morning, as a treating unit, we were quite convinced that the patient had abdominal compartment

Table 4 : Postoperative fluid chart

Fluid (Intake/ output)	*15/1 MN to 8 am (mL)*	*15/1 to 16/1 8 am–8 am (mL)*	*16/1 (8 am–5 pm)*
Urine output	575	95	Nil
RT	100	240	275
Drain PN	810	1115	40
Drain PP	730	1365	350
Total	2125	2815	665
Intake	2375	4470	1640
Balance	150 (+)	1655 (+)	985 (+)

Table 3: Postoperative blood gas analysis

Parameters	*Postoperative*	*15/1*	*15/1*	*15/1*	*15/1*	*16/1*	*16/1*	*16/1*
Time		2 am	3 pm	7 pm	11 pm	7 am	10 am	4 pm
pH	7.323	7.329	7.326	7.31	7.332	7.28	7.29	7.209
HCO_3	18.4	19.5	19	19.8	18.4	15	15.1	17
pO_2	76.7	212.9	201.9	82.2	76.7	86.6	65.4	245
sO_2 (%)	98.1	99.7	99.6	94.5	93.7	94.6	89.1	99.6
pcO_2	35.4	37.9	38	40	35.4	32.7	31.6	43.4
BE	–6.6	–5.8	–7.1	–6	–6.6	–10.1	–10	–0.6
Na	137.9	136.9	136	136.4	137.9	127.3	131.3	131.8
K	3.3	3.9	4.1	3.6	3.35	3.6	4.7	5

syndrome. The plan was to go for surgical decompression. Before we could take for surgery, we have to stabilize the patient. Hence we planned to give a couple of sessions of hemodialysis, but in the meantime, he went into circulatory collapse. The abdominal compartmental syndrome is the mode of cardiac arrest. What we are interested in what caused abdominal compartmental syndrome. For us, the first diagnosis remains sepsis because the other kidney recipient had sepsis on day 2 or 3 and recovered after starting meropenem. The liver which was shared with R&R Hospital, Delhi, and that patient died 3 or 4 days due to sepsis. The other possibility is graft rejection because he had 10% positive crossmatch. Usually 10% we would not consider it as positive. There is some problem in reading crossmatch as the patients were on prednisolone which can interfere with the reading of crossmatch. The third possibility for us is allograft pancreatitis but does not look like as there was no marker to suggest that. So our first possibility is sepsis, then is a rejection and then allograft pancreatitis. Thank you.

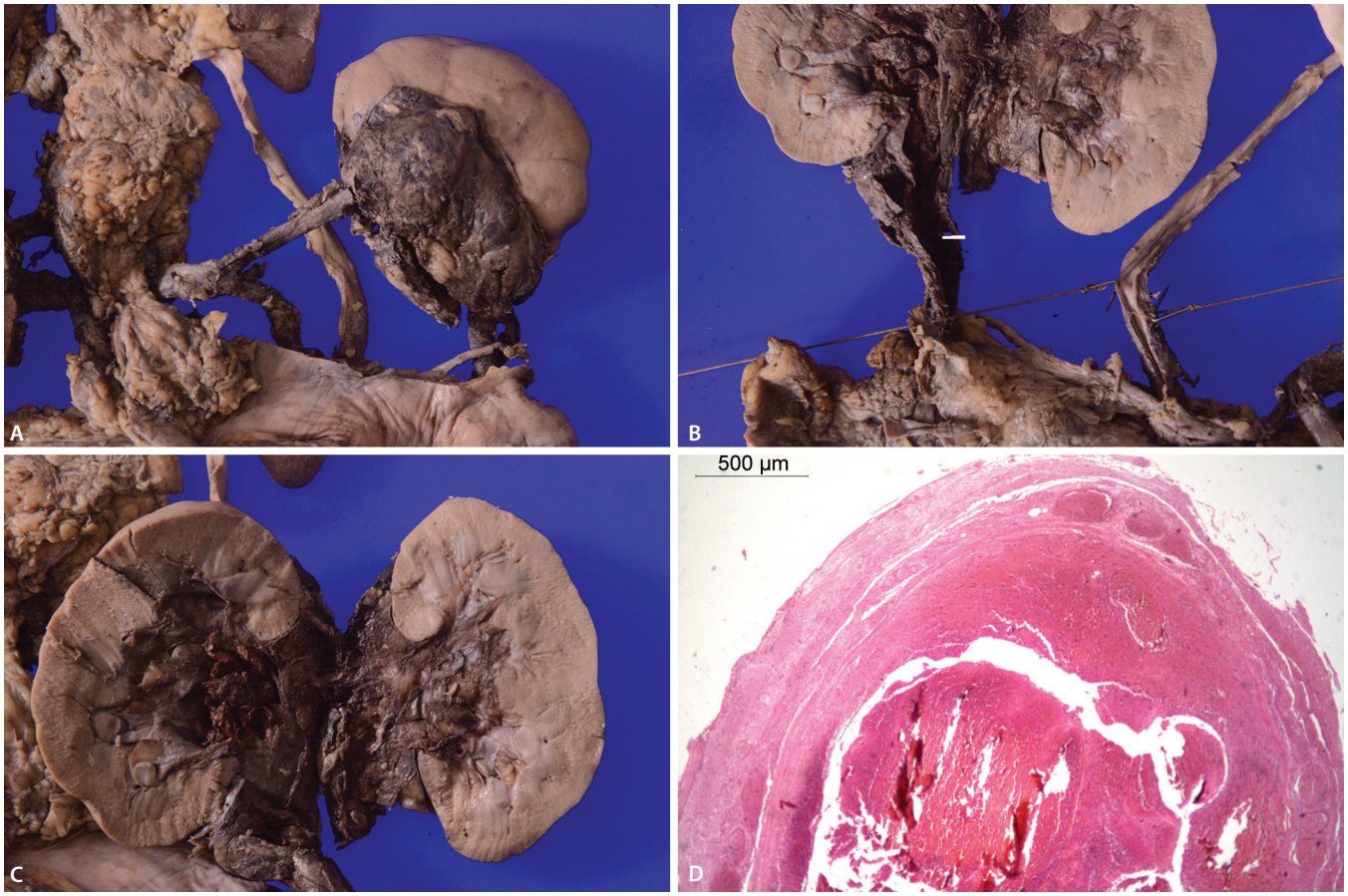
- **Prof. KL Gupta:** As far as graft dysfunction is concerned, I am thinking more in terms of acute tubular necrosis. It is not that thrombosis cannot occur; we have seen patients developing within one week. Often these were because of angioinvasive fungus. Thrombosis is more common in children. My possibility is more likely sepsis-related or hypotension-related acute tubular necrosis as the cause for renal dysfunction.
- **Prof. Anil Bansali**: One of the more reasons to favor acute compartmental syndrome (ACS) is the short stature of the patient. He had stunted growth due to uncontrolled diabetes for a long time which itself is a risk factor for developing ACS. My second comment is about the accelerated hypertension in the course of events possibly may be due to renal graft thrombosis because there was sudden hypertension followed by hypotension. The probability of reperfusion pancreatitis is very unlikely because there were brisk C peptide response which is not a feature of pancreatitis following graft rejection or absence of raise in amylase and lipase.
- **Prof. Ashish Sharma**: This is the first CPC of our pancreatic transplant. I must congratulate Dr. Ashutosh for presenting in an area for which he had minimal knowledge and digging out a lot of interesting aspects. A most frequent cause of death after pancreatic transplant is pancreatitis either because of reperfusion injury or handling. To begin with, he did not discharge urine which the other recipient did, so something was going on even before ACS was initiated. Oliguria was per se not because of ACS. I would disagree with Prof. Bhansali to say that C peptide is not a part of reperfusion injury. Whatever we have seen is that in our experience the patient who has C peptide levels in 20 or 30 have reperfusion injury, but the only odd thing was that all those patients had amylase levels in 2000 or 3000 which he did not have. But the patients with normal functioning pancreas and kidney, the C peptide levels are usually less than 10. One possibility can be that very severe pancreatitis or thrombosis can lead to the whole destruction of the pancreas. I think reperfusion injury was there. With regard to sepsis if you have a donor organ going to three patients and all of them were developing infections, then sepsis cannot be ruled out. One odd thing against ACS was that the patient was on CAPD therapy which means that the patient was used to get 3 to 4 liters of fluid. Clinically the abdomen was never tight, but the pressures were high. The second thing was during surgery we noticed lymphadenopathy along IVC. Sampling was done but the biopsy was reported as reactive. Maybe there was some infection which was not picked up at that time.
- ACS is usually a clinical diagnosis I do not expect much in the autopsy. Yes but organ changes will be there due to ACS
- **Prof. Sanjay Jain**; Is there a possibility to open the patient on the bedside rather than taking him to the theatre? The other thing is the patient had persistent hypotension. Is there any possibility of adrenal sufficiency, adrenal crises? But we don't have cortisol value. Many other autoimmune disorders are generally associated with type I diabetes.
- **Prof. Subhash Varma**: The question about bedside we will take it later.
- **Prof. Ashish Sharma**: Well. If we have ACS, presence of ACS per se can cause hypotension. What was the cause for ACS? surgical site infection. Pancreatitis is the most common cause of ACS. In this patient ACS still remains the most likely possibility.
- **Dr. Ashutosh**: The probability of adrenal insufficiency is unlikely. The first response to rejection or reperfusion pancreatitis is raised in amylase and lipase. Brisk C peptide response suggests that there is no insulitis.
- **Prof. Sanjay Jain**: ACS looks unlikely. We have two X-rays. There was no stomach or intestinal dilatation. The abdomen was soft. It can be because of only two things, either gas or fluid. The patient developed oligo-anuria on the table. What it meant, it is an event which happened peroperatively. More important is clinical finding. If the abdomen is soft then it cannot be ACS.
- **Prof. Subhash Varma**: Thank you. This is the first autopsy demonstration of combined kidney and pancreas transplant. Whether all this was related to raised abdominal pressure or something else was happening in this patient. I would also like to say this patient had a heart rate of 190. After age 20, a heart rate of 190 is not sinus tachycardia. We do not have EKG. Itis actually like paroxysmal atrial tachycardia like situation. There may be other things which we have not looked into carefully. May I request Dr. Ritambhra to tell us what happened to this patient?

Pathology Findings

A complete autopsy was performed. On opening, 300 mL hemorrhagic fluid was noted within the peritoneal cavity. Rest of the cavities was within the normal limits.

Kidney–Pancreas Complex

- ***Native Kidneys:*** Weighed 200 g. Grossly, the kidneys were small and shrunken with fine granularity on the capsular surface. Increased intra- and perinephric fat was noted. Microscopy showed varying degrees of globally sclerosed glomeruli. The viable glomeruli showed moderate mesangial expansion forming occasional KW nodule formation. Severe degree of tubular atrophy with accompanying fibrosis and lymphocytic infiltrate was noted. Blood vessels showed severe arteriosclerosis and hyperplastic arteriolosclerosis.
- ***Transplant Kidney:*** Weighed 320 g. In the left iliac fossa, hematoma measuring 3 cm was noted in the pelvicalyceal region with hemorrhagic cord like ureter extending right up to the bladder where it was bulging into the bladder mucosa (Fig. 1). Cut surface showed non-viable ureteric and pelvicalyceal wall with fresh intraluminal hemorrhages. The bladder had submucosal hemorrhages. The rest of the kidney parenchyma appeared grossly unremarkable.
- On light microscopy, there was hemorrhagic necrosis of the wall of pelvis and ureter. The lumen of the ureter was



Figs 1A to D: (A) Gross photograph of outer surface of kidney showing dilated pelvis and ureter with marked congestion; (B and C) Cut surface showing markedly congested pelivicalyceal system and ureter with fresh blood clot in the lumen of ureter; (D) Microscopy of ureter showing hemorrhagic necrosis of the wall causing luminal occlusion (H&E,10x)

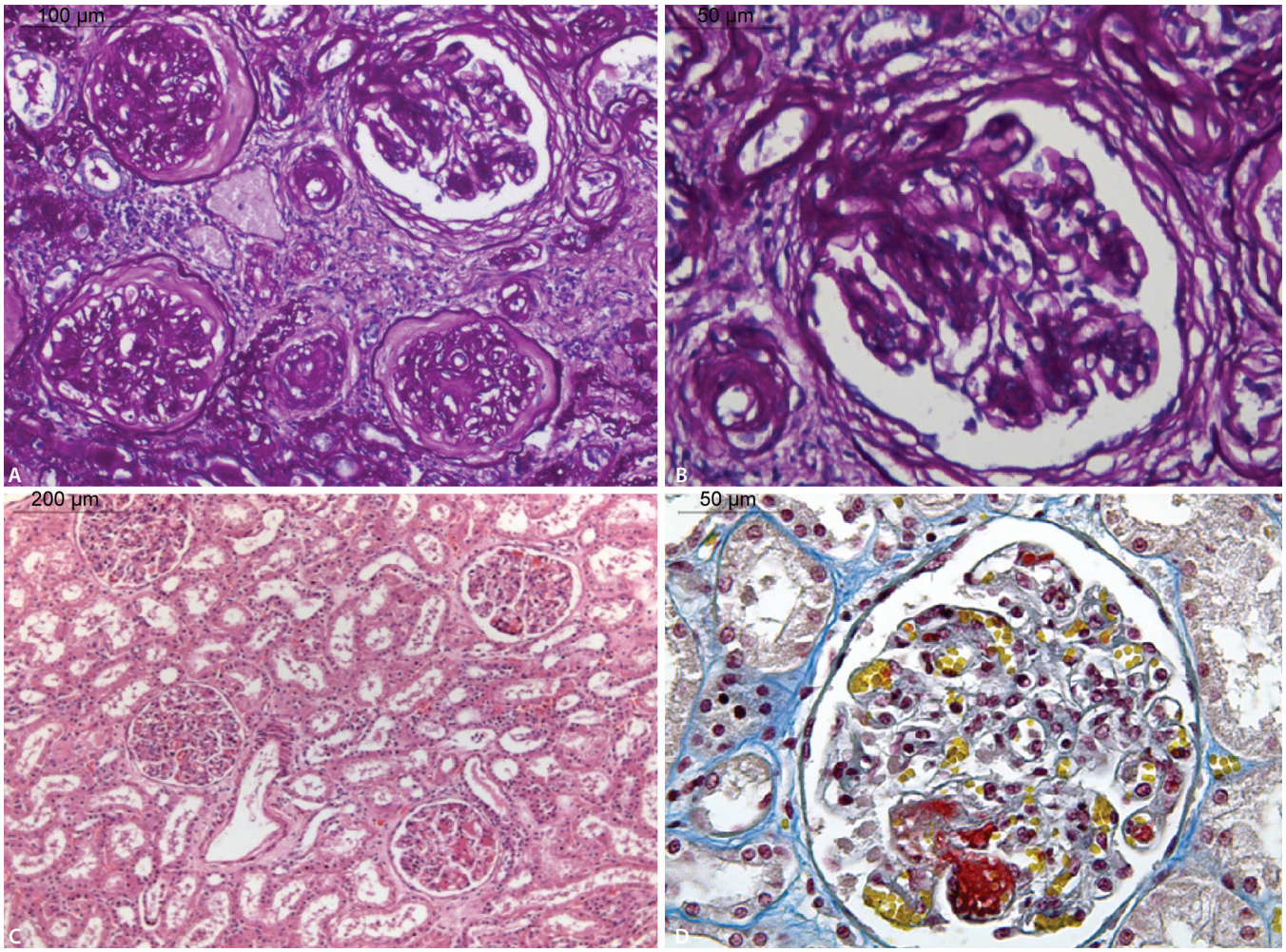
filled with fresh blood right from the kidney to the bladder. Adjacent fat showed hemorrhages. Occasional glomeruli showed a few thrombi within the glomerular capillaries and neutrophilic margination. Endothelial swelling was absent. Tubules showed mild acute tubular injury in the form of loss of brush border. There was no evidence of tubulitis or endothelitis. PTC dilatation and margination were absent. C4d was negative in the peritubular capillaries. There was no evidence of rejection or CMV/BKV inclusions or fungal organisms (Fig. 2).

- ***Native Pancreas*:** Weighed 100 g. Grossly, it was firm and fibrotic. Microscopically, mild interlobular fibrosis with fat infiltration was noted. Islets showed fibrosis and calcification. There was a relative decrease in islet number.
- ***Transplanted Pancreas:*** Weighed 320 g. It was bulky and located in the right iliac fossa with chalky white deposits on the external surface. Anastomotic sites were patent. Exocrine drainage was anastomosed to the small intestine which appeared hemorrhagic. Vascular anastomosis with aorta was patent. There was acute pancreatitis with areas of necrosis. Ductular inflammation, venulitis, interlobular, or inflammation were not seen. There was no evidence of rejection. Peripancreatic venous thrombosis and hemorrhages were noted.
- ***Lungs:*** Weighed 710 gm. Both the lungs were subcrepitant with brownish discoloration on the cut surface. Microscopy showed capillary congestion in the alveolar septa and focal areas of alveolar hemorrhages (Fig. 3).
- ***Spleen:*** Weighed 150 g. Gross and microscopy were normal.
- ***GIT:*** Grossly superficial hemorrhages.
- ***Heart:*** Weighed 410 g. All the valves were normal. Left ventricular wall thickness measured 1.6 cm indicating hypertrophy of the wall. Rest of the chambers was normal. Microscopically, the cardiac myocytes were unremarkable.
- ***Brain:*** Weighed 1485 g. Gross and microscopy were normal.

Final autopsy diagnosis (PM 27156)

Case of simultaneous pancreatic kidney transplantation for type I diabetes mellitus, end-stage kidney disease with chronic kidney disease.

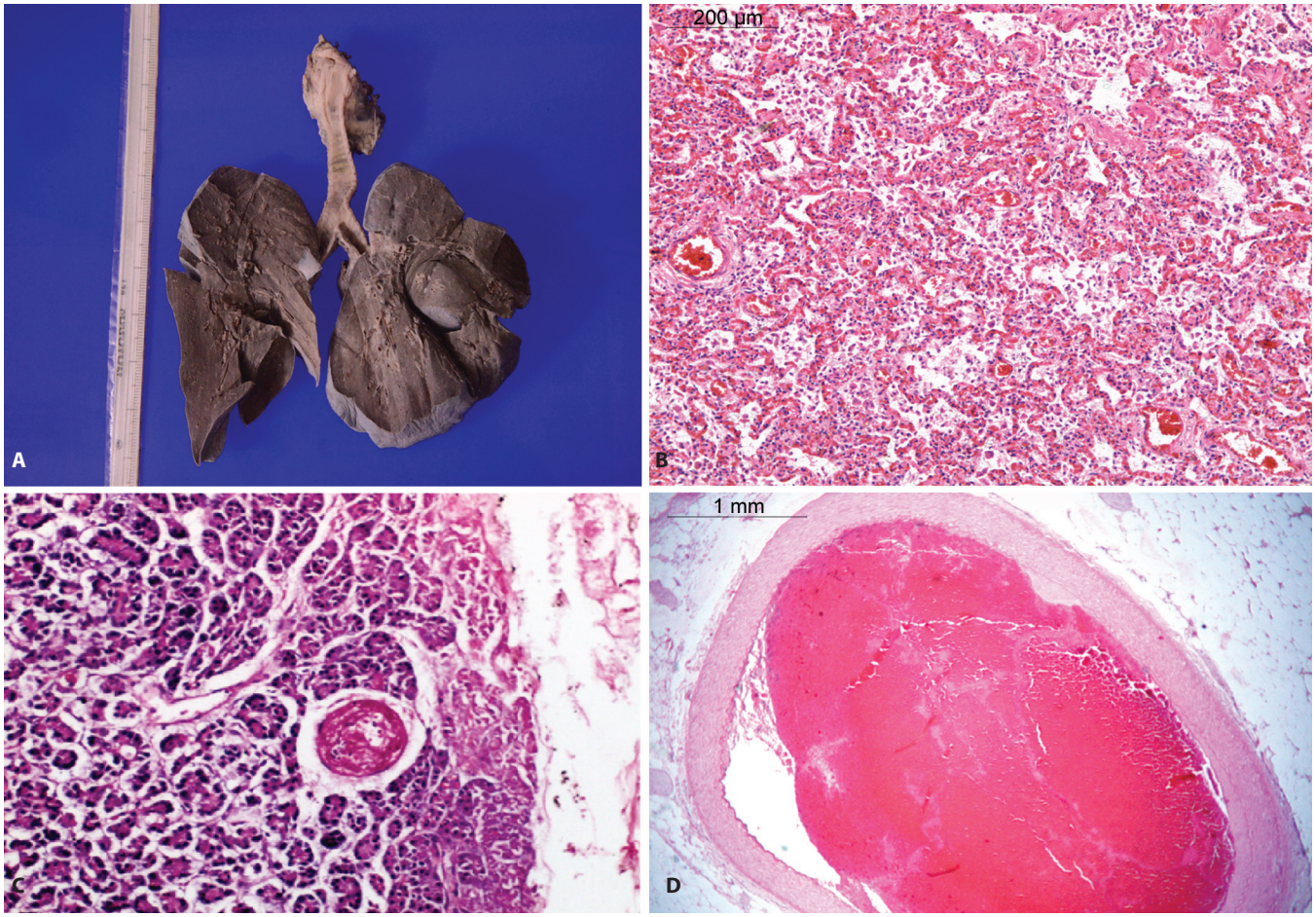
- Islet cell fibrosis and calcification, chronic pancreatitis in the native pancreas
- Acute pancreatitis with venous thrombosis in the transplanted pancreas
- Diabetic nephropathy–class IV with severe arterionephrosclerosis in the native kidney
- Acute pelvicalyceal and ureteric hemorrhagic necrosis with luminal bleed in the transplanted kidney.
- Left ventricular hypertrophy
- Pulmonary hemorrhage



Figs 2A to D: (A) Microscopy of native kidney showing varying degrees of sclerosed glomeruli with hyperplastic vessels (PAS, 20x); (B) High power view of a glomeruli with thickened basement membrane and occasional K-W nodule (PAS,40x); (C and D) Microscopy of transplant kidney showing acute tubular necrosis and occasional glomerular thrombi (H&E, 10x). High power view of glomeruli with occasional fibrin thrombi (MSB, 40x)

Final Discussion

- **Prof. Subhash Varma**: Thank you, Dr. Ritambhra, for showing very interesting and devastating pathology. Please join me here. Now both the clinical and pathological protocol is open for discussion.
- **Prof. Sanjay Jain**: Ritambhra, have you ruled out isolated ureteric rejection because you had done C4d only in the kidney but were not shown in the ureter
- **Prof. Ritambhra**: Sir, only the lower part of the ureter was viable. One has to look for the presence of inflammation into the ureteric epithelium which was not viable in the area sampled.
- **Prof. Sanjay Jain**: I was asking for cell-mediated, not antibody mediated.
- **Prof. Ritambhra**: Even for that sir, we need to see the structural integrity of ureter.
- **Prof. Sanjay Jain**: Otherwise, the veins were intact, that remains a possibility. Second, I could be a drug-induced vascular thrombosis. We have seen thrombosis both in transplanted pancreas and kidney. Although it may not be a classical HUS, could it be thrombotic microangiopathy?
- **Prof. Ritambhra**: Sir, the thrombosis was very limited to the transplanted kidney. Pancreas had just occasional focus of thrombosis that too in the peripancreatic region, it was not a very dominant component. That is why I did not even put it as a part of the rejection. The extent of thrombosis was too limited even with such a severe degree of acute pancreatitis.
- **Dr. Manish:** Good morning. I do not know why you are falling short of calling it as DIC. You have said that the extent of thrombosis is limited. It is possible in the autopsy that you may not be able to show extensive thrombosis. There was thrombosis in kidney, pancreas, and lungs. It can very well be DIC.
- Regarding ureteric necrosis, you have not shown the ureteric vessels with thrombosis. Can it be a part of intravascular coagulation leading on to ureteral necrosis? If it is intravascular coagulation, DIC can lead to pancreatic artery thrombosis leading on to pancreatic necrosis. On the other hand poor bedside pancreatic dissection increased ischemia, and pancreatic thrombosis can lead to intravascular thrombosis and DIC. Both ways it is possible.
- **Prof. Ashish Bhalla**: The most solacing is no finding of infection for the first time in the CPC. Otherwise, everyone is expecting sepsis. There are two comments. You showed pancreatic calcification in the native pancreas which is never a feature of type I diabetes. I think we must revise the diagnosis of type I diabetes. This might be secondary diabetes. Secondly, you hardly find any



Figs 3A to D: (A) Gross photograph of cut surface of lung showing focal brownish discolouration; (B) Microscopy shows desquamated alveolar epithelial cells with hyaline membrane formation (H&E,10x); (C and D) Microscopy of pancreas showing capillary and venous thrombi in the pancreatic and peripancreatic adipose tissue (H&E, 20x)

correlation between the structural and functional integrity of pancreas. You showed a lot of pancreatic, but still, the enzymes were normal. You always lack a correlation between structural disorganization and function integrity of the endocrine system. My third comment is on ureteric obstruction that may be the cause of intra-abdominal hypertension. There was a large hematoma. That's why Dr. Sanjay Jain told if it is a soft abdomen it denies but you have raised intrabdominal pressure measured by putting the Foleys catheter into the bladder that was very evident. Possibly this may be responsible for that hematoma.

- **Prof. Ashish Sharma**: I think one thing is very clear there is no ACS. Only the transplanted organ is affected and the rest of the organs was not affected hence ACS is ruled out. Regarding the discrepancy between the enzymes and C peptide levels as I already pointed out that if you have massive necrosis there may not be increased in enzyme levels. It is the same in case of a liver transplant that you may not see transaminitis if you have complete thrombosis. But islets are known to survive. The main vessels I presume were patent because C peptide vessels were being released into the circulation. The only way it can come in through the main vessels. So what is the possible explanation? The only explanation is an ischemia-reperfusion injury either there is something wrong in the retrieval technique, or there is something wrong with the donor. We are pretty selective about the donors for pancreas donation. The only possibility which remains is the retrieval technique. As far as the infection is concerned we do not know why he went into leucopenia. I do not think there is any explanation for that. As far as the ureteric hematoma is concerned, it will not cause ACS. Why this necrosis had happened. I do not know. I have experience of close to more than 3000 kidney transplant and we have never seen such necrosis at any time. What we have seen is bland necrosis which can happen if you accidentally tie up the lower pole of the artery. I do not think that can cause hemorrhage. One possibility can be if the lower pole of the artery is damaged if this patient was on heparin and there was some ischemic area and which may be bleeding.
- **Dr Deepesh**: I do not know. Clinically the abdomen was soft. There was a drain *in situ*. There was nothing come out of the drain. As Dr. Jain rightly pointed out that there was no gas and there was no fluid. The only possibility is that tissue edema. This patient was on CAPD which means that the abdomen was used 4 to 5 liters of fluid which was hard to explain clinically. That is why we never thought of opening at bedside we thought of removing the pancreas. Unfortunately, the patient never gives us time.
- **Prof. KL Gupta:** Leucopenia could be because of the immunosuppression that he had. Sepsis may not always cause leucopenia. As far as ACS is concerned there is no doubt that the abdominal pressures were high. The patient had pancreatitis

which is the most common cause of ACS. The patient had hypotension and ARDS, so all these features fit into ACS.

- **Dr. Manish**: As a clinician when intra-abdominal pressure is very high >50 mm Hg, we have to think of ACS. It is not necessary that he should always have fluid or gas. Even if there is ileus. There are two transplanted organs which are major organs in a short child with a limited abdominal laxity. Though I agree with Dr. Ashish that he was on CAPD, so the abdominal walls were more complaint still ACS and hypotension should be considered when the pressures were high.
- **Prof. Sanjay Jain**: I think this is the point that can be discussed. There may be a problem in measurement. Sometimes we must be careful when we are interpreting the data here. If we have clinicomeasurement disassociation again, one needs to go back and see we what we were measuring is truly measured or not before you be dogmatic.
- First time when the intra-abdominal pressure was mentioned as 50 that time he was having hypertension not hypotension
- **Prof. Sanjay Jain:** Physicians are talking of surgical issues like ACS, but we have not seen once. Surgeons who are experienced should talk about that. Whatever we are talking is off the hand absolutely I can tell you know.
- **Prof. Subhash Varma**: We do not have time now. Anupam for the last comment.
- **Dr. Anupam:** It has been seen that patient who is on prolonged peritoneal dialysis may have adhesions and falsely raised interpretation of the intra-abdominal pressure. We have seen so many patients with CAPD for a long time with a lot of adhesions in the pelvis. They may have to encapsulate peritonitis kind of picture. That we have to consider that in this patient, he might have a false raise in intraa-bdominal pressure.
- **Prof Subhash Varma:** Thank you for the interesting discussion. There are lots of questions that have not been answered. But we have a very interesting demonstration today.

Summary

Ureteral necrosis remains a rather frequent complication after kidney transplantation despite improvement in surgical techniques and is ranging from 2–5% in the literature. Ureteral ischemia is the most common cause of ureteral necrosis. The other etiologies which may lead to ureteral necrosis include rejection and viral infections. Isolated ureteral rejection is very rare and is generally associated with rejection induced changes in the renal parenchyma. The viral infections which are also implicated in the development of ureteral necrosis include Cytomegalovirus and BKV infection. Although not well documented in the literature, older age and delayed graft dysfunction can also lead to ureteral necrosis. Old age has more frequent vascular alterations leading to ischemia and necrosis. In delayed graft dysfunction, ureteral edema can impair the venous drainage resulting in ischemia.

The pathogenesis of this complication probably involves (a) a primary deficit of blood supply from the renal vessels to the pelvis and ureter, (b) a failure to develop a new ureteral blood supply because of surrounding hematoma, (c) early swelling of the ischemic ureter resulting in oliguria interpreted as acute tubular necrosis, (d) resolution of edema resulting in diuresis, and (e) late patchy ureteral necrosis .

Development of this complication, however, has no effect on the patient or long term graft survival. Early detection and timely management is important to avoid significant morbidity to the recipient.

In the present case, the cause of ureteral necrosis is likely due to a vascular complication at the time of surgery rather than due to infection or rejection.

Suggested reading

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