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Case Report

Forty-Eight Years Old Woman Who Received Blood Group Matched Living Kidney from Twenty-Five Years Old Daughter Having Persistent Anuria for 13 Days Since Ureteric Anastomosis Developed Spontaneous Renal Allograft Rupture Due to Acute T Cell Mediated Rejection Producing Hemoperitoneum and Shock on Day 13 After Transplant: A Case Report

Khin Phyu Pyar[1]*, Tin Moe Mya[2], Ohmar Hlaing[3], Yu Aye Latt[4], Chan Myae[5], Ye Min Hein[6], Lay Maung Maung[6], Lin Htet Aung[6], Myo Maung Maung[6], Moe Tun Zaw[6], Kyaw Thurein Lwin[6], Min Aung Hein[6], Chan Pyae Aung[6], Kaung Myat Oo[7], Htet Aung[7], Zin Phyo Hlaing[7], Sein Kyaw[8], Wai Zin Win[8], Sai Aik Hla[6], Aung Zaw Htet[6], Ye Htook Maung[6] & Myo Min Thant[6]

[1]Professor and Head, Consultant Physician, Department of Medicine/Nephrology, Defence Services Medical Academy/ No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

[2]Professor and Head, Consultant Pathologist, Department of Pathology, No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

[3]Professor and Head, Consultant Radiologist, Department of Radiology, Defense Services Medical Academy, Yangon, Myanmar

[4]Professor and Head, Consultant Anesthetist, Department of Anesthesiology, No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

[5]Consultant Intensivist, Department of Intensive Care, No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

[6]Nephrology Fellow, Department of Nephrology, No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

[7]Consultant Pathologist, Department of Pathology, Defense Services Medical Academy, Yangon, Myanmar.

[8]Consultant Radiologist, Department of Radiology, No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

***Corresponding author:** Khin Phyu Pyar, Professor and Head, Consultant Physician, Department of Medicine/Nephrology, Defence Services Medical Academy/ No (1) Defense Services General Hospital (1000-Bedded), Yangon, Myanmar

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Abstract

A 48-year-old female who received kidney from blood group matched daughter allograft recipient had persistent anuria (non-functioning graft) for 13 days since ureteric anastomosis in transplant surgery. Graft biopsy done on third post operative day did not show features of acute rejection or acute tubular necrosis. On 4th post operative day, she had hemoperitoneum with hypovolemic shock; active bleeding point was not seen in laparotomy. The consistency of kidney was slightly soft; wedge biopsy was taken, and the histology was the same as before. Later, the patient had sepsis and DIC which were fairly controlled. On 13th post-operative day, she had hypovolemic shock again due to hemoperitoneum; laparotomy revealed 5 cm laceration along the convex border of the graft, with normal artery, vein and ureter. Donor nephrectomy was done in view of non-functioning graft for 13 days with escalating anti-rejection therapy. Histologically the graft demonstrated severe acute cellular rejection. The patient had been on hemodialysis since first post-operative day.

Keywords: Non-Functioning Graft, Renal Transplant Recipient; Living Kidney; Rupture Allograft Kidney; Cellular Rejection

Introduction

The quality of life of patients with end stage kidney disease is better with the success of renal transplant. The rejection rate falls with modern immunosuppressive therapy. Renal allograft rupture is rare; however, a well-known complication of renal transplantation. This catastrophe needs an immediate diagnosis and aggressive therapy being a life-threatening situation. Its prevalence varies from 0.3% to 3.6% depending on donor characteristics, procurement and preservation conditions, recipient characteristic, surgical characteristics and medical characteristics. The usual timing for rupture is first few weeks after transplantation. The main predisposing factor involved is acute antibody mediated rejection (ABMR) producing increased intraluminal pressure [1]. Other reported a etiological factors are ureteral obstruction, acute tubular necrosis [2,3], renal biopsy, heparin therapy, complete lymphocytic ligation, renal vein obstruction and renal trauma.

The cadaveric renal allografts are more likely to be complicated by rupture than living kidney transplant. Moreover, high donor recipient age difference is found to be associated with rupture. In clinical review on over 300 renal transplants in late 20 century revealed that older recipient age, donor-recipient race mismatch (White donor to Black recipient), and dialysis requirement were statistically related with renal allograft rupture. Gender, race, cold ischemic time, transplant number, living related donor vs. cadaveric donor, peak or current panel reactive antibody (PRA), and total HLA and BDR mismatch were not statistically correlated with rupture. The important point they highlighted was that acute tubular necrosis (ATN) and rejection act synergistically to cause renal allograft rupture (RAR); therefore, early delayed function requires intensive and perhaps novel immunosuppression, especially in Black recipients [4].

The diagnosis of rupture is mainly clinical, pain, hemoperitoneum and hypovolemic shock. USG shows fluids around grafted kidney. Multi-detector CT scan (MDCT) is a useful investigative tool for the evaluation of suspected spontaneous renal allograft rupture; a swollen graft, disruption of the capsule, parenchyma, and/or hematoma should prompt the radiologist to consider this diagnosis. Early diagnosis is essential to save the life of patient. Although the procedure of choice for kidney allograft rupture was graft repair to preserve the allograft [5], there were reports on graft nephrectomy [6]. Successful salvaging of severely ruptured living-related renal allograft secondary to acute antibody mediated rejection with modern immunosuppression therapy and proven surgical procedures was mentioned in some reports [1]; and they were very inspiring. The success of salvage rate varies between 40% to 100%; and it is well worth trying to salvage spontaneously ruptured renal allografts. Graft nephrectomy should be considered as the only definitive treatment; it is indicated when the hemodynamic status cannot be stabilized by appropriate aggressive hemodynamic support therapy [7]. The allograft rupture can be prevented with the use of well-matched, good quality kidneys. In addition, reducing or managing risk factors that would predispose to delayed graft function are essential, ensuring a technically satisfactory transplant procedure with short cold and warm ischemic times, and avoiding large donor-recipient age gradients [8]. The persistent non-functioning graft (severity of acute rejection and anuria) since transplant surgery, re-anastomosis of ureter, was rarely mentioned in former reports.

Case Presentation

The patient was 48-year-old lady having end stage renal disease due to presumed diabetes kidney disease. She had been on maintenance hemodialysis 3 times/week for 4 years; she received two units blood transfusions in 2019 due to ESRD anemia. She also had a co-morbid medical disease such as hypertension for 2 years. Her residual urine output was 100 ml/24 hours. The donor was her daughter, 25 years old. She had one child, no history of blood transfusion. She did not have diabetes mellitus or hypertension. CDC crossmatch was as follows:

a. Normal B cell positive 1:8

b. Normal T-cell crossmatch-Negative

c. DTT-treated B-cell and T-cell crossmatch-Negative

d. AHG -treated B-cell and T-cell crossmatch-Negative

The following were donor HLA typing: HLA A02, B07, C03, DRB104, DRB3(+), DRB4(+). DQB103 and donor specific antibody (DSA) was negative. They were ABO compatible; blood group "A" Rh positive. The body weight of the patient was 45 Kg (BMI 20 kg/m2); and that of her daughter was 50.9 Kg. Induction was done with anti-thymocyte globulin in the dose of 3 mg/kg and triple immunosuppression was given with tacrolimus, mycophenolate mofetil, and prednisolone. The removal of the donor kidney, as well as implantation of transplanted kidney, was uneventful. The first warm ischemic time was 2 minutes, cold ischemic time was 18 minutes; and second warm ischemic time was 36 minutes (total 56 minutes). Renal vein was connected to the external iliac vein by side to side; and the renal artery was end to end anastomosed to the right internal iliac artery. Apart from slight disparity in size between the donor's renal artery and recipient's right internal iliac artery, the anastomosis was smooth. The arteries of the recipient were thickened with atheromatous plaques. Finally, ureter was implanted into bladder. The date of transplant was April 22, 2022. During operation, the urine output was less than 50 cc till immediate first hour after surgery although blood pressure was normal. As there was no gross feature of hyper acute rejection till reconstruction of ureter, augmentation of fluid infusion together with frusemide was tried and also albumin infusion was done to improve oncotic pressure. She was anuric-50 cc at first 6 hours. So, hemodialysis was initiated immediately for 4 hours duration with heparin free and the amount of ultra-filtration (UF) was 2 liters. Doppler USG of graft revealed normal cortical echo with intact cortico-medullary junction, normal color flow up to the cortex. The Resistive Index was 0.58; normal limit (normal range of Resistive Index was 0.5-0.7).

As Doppler USG excluded mechanical cause, the possibilities were accelerated acute rejection and acute tubular necrosis. Thus, methylprednisolone was extended up to 5 days, the local protocol being 3 days. The total urine output for the immediate post-op day was less than 200 cc, anuria. USG was repeated on next day (post-op day 1); the Resistive Index immediately increased to 0.8 from 0.58. There was further decreased in cortical echo at upper pole and color signal in upper pole cortex also reduced. Renal length increased to 9.7 cm; cortico-medullary junction was still intact and perinephric fluid collection was same. All the findings pointed to the possibility of vascular thrombosis at upper pole. Therefore, low dose of low molecular weight heparin (Enoxaparin 40mg/0.4ml) injection was initiated. Her fluid status was controlled with fluid restriction according to urine out-put status. Total urine out-put in first post-op day was 100 cc/24 hour. She had no more fever and vital signs were stable. As the blood pressure became 180/120 mmHg; parenteral GTN infusion was done together with oral antihypertensive drugs (Nifedipine 20mg stat and BD at 6 am, also added carvedilol 25mg BD at 10 am, Methyldopa 500mg BD at 2 pm and Duracard [Doxazosin] 4mg BD at 8 pm). Risks and benefits of transplant kidney biopsy were discussed. So, Enoxaparin injection was withheld for 24 hours for graft biopsy. On 1st post-operative day, full blood count was within normal apart from low hemoglobin (8.2 g/dl); electrolytes were normal. On post-op day 2, her blood pressure was controlled with parenteral GTN infusion and oral anti-hypertensive drugs (Nifedipine 20mg BD, Carvedilol 25mg BD, Duracard [Doxazosin] 4mg BD and Methyldopa 500mg BD). The patient was clinically stable though anuria persisted (urine output was 50-100ml per 24 hour).

Doppler USG findings were as follows:

a. Increased in transplant renal size 9.9 cm and cortical echo.

b. Minimal perinephric fluid collection at upper and lower poles.

c. Reduced perfusion in upper pole and

d. Resistive index was 0.78. Second time hemodialysis was done at evening with 4 hours duration, heparin free and uf 1500 cc. After that, two units of iv immunoglobulin g infusion was given with pre-medication as the had anuria for 48 hours.

The Renal biopsy was done on post-op day 3. It revealed as follows:

a. C4d negative for glomerular and peritubular capillaries (C4d0)

b. No evidence of acute rejection

c. No significant interstitial fibrosis and tubular atrophy

d. No arteriosclerosis

e. Ischemia of glomeruli noted (Figures 1-4). It was done after 33 hours of last dose Enoxaparin injection.

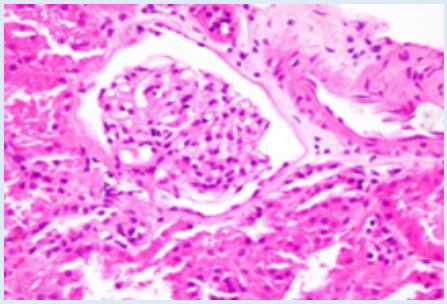

Figure 1: H & E stain of needle biopsy showing normal glomeruli, tubules, interstitial cells and endothelium. No evidence of acute rejection.

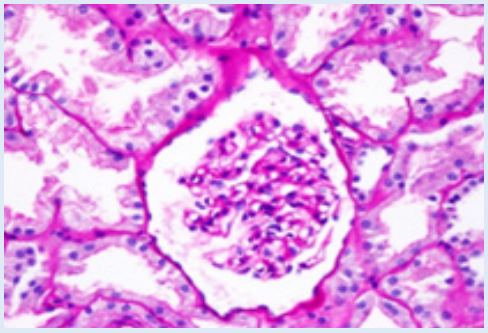

Figure 2: H & E stain of needle biopsy was normal.

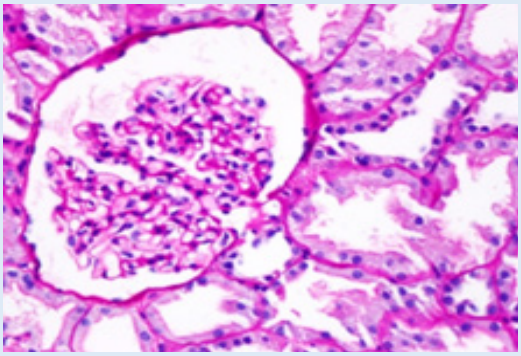

Figure 3: PAS stain of needle biopsy was normal.

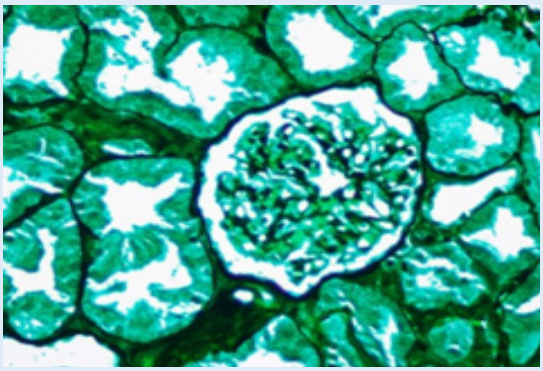

Figure 4: Silver stain of needle biopsy was normal.

And CT renal angiogram was done for graft renal function status. It showed transplant kidney in right iliac fossa with

reduced enhancement and there was no excretion even after 4 hours. Moreover, minimal perinephric fluid collection and minimal ascites were noted. Post contrast plain X-ray abdomen clearly demonstrated non-functioning status of grafted kidney as there was no contrast in bladder (Figures 1-4). Then, two units of IV immunoglobulin G infusion were repeated with pre-medication as rejection was presumed to be antibody mediated rejection (ABMR). In the evening, renal function was maintained with hemodialysis; heparin free and ultra-filtrated 600 cc. At midnight, drainage tube fluid increased suddenly to 350 cc over 4 hours, brownish red blood. The patient felt pain at right iliac fossa. On post-op day 4 morning at 06:00 hour, the blood pressure rose suddenly to 200/110 mmHg; thus, nitrate infusion was initiated again. An hour later, it dropped to 90/60 mmHg suddenly. The patient was paper white; extremities were cold and clammy.

So, IV Noradrenaline and IV Dobutamine infusion titration were given initially to maintain blood pressure. Oxygen saturation decreased to less than 90%. The drainage tube's total amount was increased to 500 cc, fresh red blood. The impression was hypovolemic shock due to hemoperitoneum as a result of blood leakage from either anastomosis site or renal biopsy site. Urgent laparotomy was done; huge perinephric hematoma about 1 liter was evacuated. The strange thing was that there was no active bleeding site; the grafted kidney looked healthy, but it was slightly soft. The vascular anastomosis site and renal biopsy site were clean, no active bleeding. Minimal ascites was noted; then, wedge renal biopsy was taken from the lower pole. The histology of wedge biopsy was the same; ischemic changes in some glomeruli and congestion of some glomeruli were seen. There was no evidence of glomerulitis. There was no evidence of tubulitis or tubular fibrosis. The interstitium was normal. Both arteries and capillaries were normal. No evidence of acute rejection (Figures 5- 8).

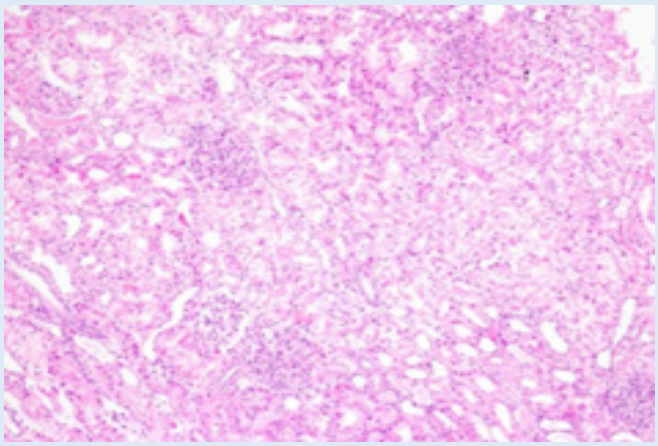

Figure 5: H & E stain of cone biopsy was normal.

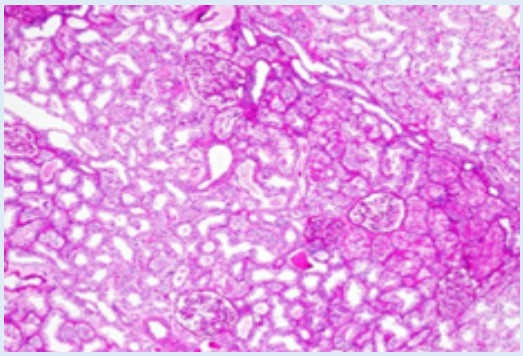

Figure 6: PAS stain of cone biopsy was normal.

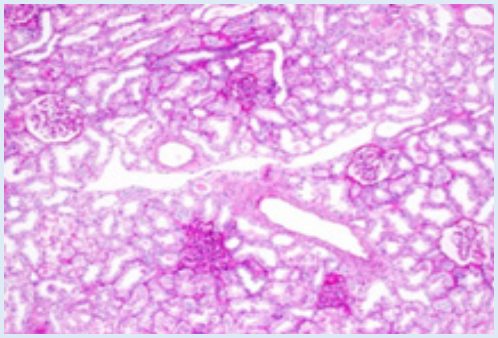

Figure 7: PAS stain of cone biopsy was normal.

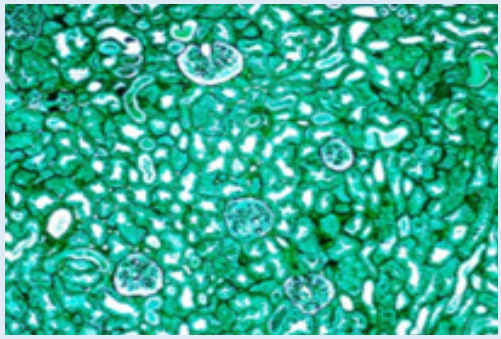

Figure 8: Silver stain of cone biopsy was normal.

After the operation, temperature was 99°F, vital signs were within normal limit with IV GTN infusion titration, added IV Azeptil (Tranxenamic acid) 250mg 8 hourly, IV vitamin C & K pair 12 hourly, IV Meropenam 500mg 12 hourly and total 4 units of whole blood were supplied in that day. Anuria persisted; urine output was 125 cc/24 hour. Extubated was done on 5:30pm of that day and the patient GCS was 15/15. Her hemoglobin was 3.5 g/dl, increased urea and creatinine level, other laboratory parameters (WBC, platelets and electrolytes) were normal. On Day 5; the patient's condition was stable with GCS 15/15, normal temperature, blood pressure was controlled with GTN infusion and oral anti-hypertensive drugs (Nifedipine 40mg BD, Cardivas 12.5mg BD, Duracard 4mg BD). The urine color and amount were much improved (Total 245 cc), and the drain tube color also improved (Total 40 cc). Two units of whole blood was given in that day. Hemodialysis was done on evening with 4 hours duration; heparin free with ultrafiltration 1500 cc. One unit

of fresh whole blood was transfused during dialysis. Doppler USG revealed as follows:

a. Renal size reduced to 8.9 cm

b. Normal cortical echo

c. Small amount of perinephric fluid collection on lower pole

d. Resistive index was 0.7; and,

e. Reduced doppler flow at upper pole. Methylprednisolone was changed to oral prednisolone and the remaining two immunosuppressive drugs (tacrolimus and mycofenolate mofetil) were continued according to transplant protocol.

On Day 6 morning, she complained of breathlessness at rest; oxygen saturation dropped to 92% with 4L of oxygen. Bilateral few basal crackles were detected. Portable CXR showed right upper lobe collapse due to mucous plug. (Figure 9) Thus, urgent bronchoscopy and bronchoscopic suction and washing were done. Bronchoscopic washing AFB and C&S were sent. Also sent Blood C&S, Urine C&S, Stool C&S for infection screening. WBC count significantly increased to 13.1×109/L; antibiotics was escalated with IV Moxifloxacin 400mg. In the evening, hemodialysis was done for 3hours 45minutes, ultrafiltration 1700 cc with heparin free. That day, total urine output was 297 cc and drain tube fluid total was 100 cc. On Day 7, temperature 100°F with stable vital signs; features of aspiration pneumonia were detected. So, IV Metronidazole was added. Tacrolimus trough level was 4.1; thus, the dose increased to 2 mg 12hrly. Urine output was 40 cc. Doppler USG recheck revealed as follows:

a) Renal size increased to 9.6 cm

b) Decreased cortical echo

c) Free fluid was present in hepatorenal pouch, perinephric region and pelvic cavity

d) Resistive index was 0.6 and

e) Reduced doppler flow at upper pole

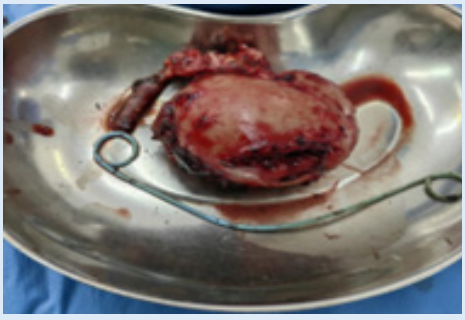

Figure 9: Allograft kidney showing tear 5 cm at lateral border.

On Day 8, temperature was 100°F with stable vital signs and bilateral basal crackles were audible. Blood C&S revealed Pseudomonas aeruginosa with sensitive to Cefoperazone/Sulbactam; therefore, it was added. Her blood pressure was controlled with four anti-hypertensive drugs and blood sugar was controlled with sc Insulin basal and correction bolus regimen. Hemodialysis was done in that day with heparin free, ultrafiltration 2000 cc and total 4 hours duration. Urine output was total 10 cc per 24 hours and drain tube was 80 cc. Fever increased to 103°F with high total WBC (11.7×109/L, neutrophil leukocytosis) and liver enzymes were increased (ALT 278 U/L, AST 134 U/L).

On Day 9, fever persisted (temperature 101°F) and vital signs were stable. Urine output was 85 cc the whole day. Stool C&S result revealed Burkholderia cepacia. That day antibiotics already given were IV Meropenam, IV Moxifloxacin, IV Metro and IV CS1. But total WBC was still high (11.9×109/L, neutrophil leucocytosis) and liver enzyme also high ALT 163 U/L, AST 60 U/L. On Day 10, fever was swinging; and the patient had loose motion (3 times). Clinically vital signs were normal ranges with crepitation in bilateral basal lung fields. Urine output was 75 cc in the whole day. Hemodialysis was done for renal function maintenance with heparin free, 4 hours duration and ultrafiltration 700 cc. Bronchial washing results were got; (Rt) Bronchus C&S revealed Klebsiella pneumoniae ssp and bronchial washing vocal cord revealed Acinetobacter baumannii complex. So, antibiotics add IV Elores (Ceftriaxone + Disodium edetate + Sulbactam) 1.5G 12hourly and stopped IV CS1 injection. Total WBC is still rising 12.25×109/L (neutrophil leucocytosis) and ALT still high 97 U/L.

On Day 11 and Day 12, intermittent fever (within 99°F to 103°F) still present with anaemia, bilateral basal crackle lungs sign. Urine output was still not improving with < 50 cc daily. Total WBC count became higher (14.14×109/L, neutrophil leucocytosis); and hypokalaemia set in. It was corrected with IV and per oral potassium. Recheck CXR (AP view) revealed improving of previous right upper lobe collapse and consolidation. (Figure 10) Blood pressure was controlled with four anti-hypertensive drugs, blood sugar controlled with Insulin basal and correction bolus regimen, four IV anti-biotic drugs and other supportive drugs. Fluid management done with fluid restriction according to daily urine out-put and also alternate day hemodialysis. On Day 13 Morning, the patient suffered abdominal pain following one shot of high blood pressure 200/110 mmHg. Then, blood pressure dropped suddenly to 80/60 mmHg with marked features of pallor; the colour of fluid from drain tube turned to red blood suddenly. Impression was recurrence of hemoperitoneum causing hypovolemic shock with septicemia and DIC; USG showed massive free fluid noted in the perinephric and pelvic cavity. Emergency laparotomy was done. Operative findings were as follows:

a. The color of graft was pink with a tear measuring about 5 cm at middle part of convex border.

b. There was active bleeding.

c. The graft was surrounded by a large hemorrhage and hematoma about 2.5 liters.

d. Renal artery, vein and ureter of grafted kidney were normal.

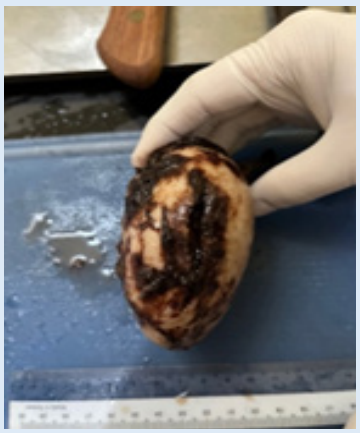

Figure 10: Allograft kidney showing tear 5 cm at lateral border after formalin fixation.

Donor nephrectomy was done in view of non-functioning kidney for 13 days with anti-rejection therapy. (Figures 11 & 12) Cut section showed distinct cortico-medullary junction compatible with shock kidney. (Figure 13) The cause of hemoperitoneum was from tear of allograft kidney. Hemodynamic parameters were stable after nephrectomy. Histologically the graft demonstrated acute cellular rejection. Histology revealed acute T cell mediated rejection as there were fibrinoid necrosis, tubulitis, interstitial oedema, hemorrhage, intimal hemorrhage in small and medium size arteries. (Figures 14 & 15). A total of whole blood 6 units, FFP 3 units, packed red cell 2 units, PRP 2 units (Total 13 units) were replaced for her blood loss. The patient had been on maintenance hemodialysis regularly. On Day 15, fresh blood coming out from drainage tube suddenly with falling blood pressure again. Fresh whole blood, fresh frozen plasma and platelet rich plasma were given as the possible source was DIC. After transfusion, she became stable on Day 20. She was on hemodialysis on regular basis.

Figure 11: Cut section of allograft kidney showing distinct cortico-medullary junction.

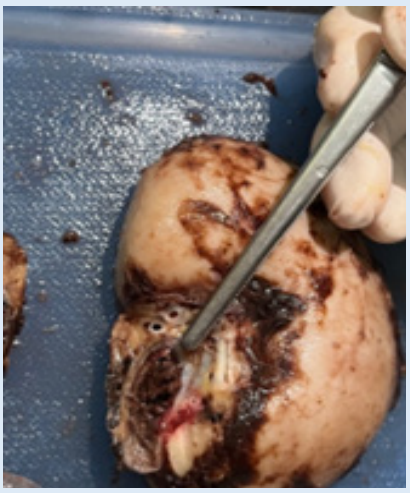

Figure 12: Allograft kidney showing a large blood clot 2X1 cm in proximal ureter with normal renal artery and vein.

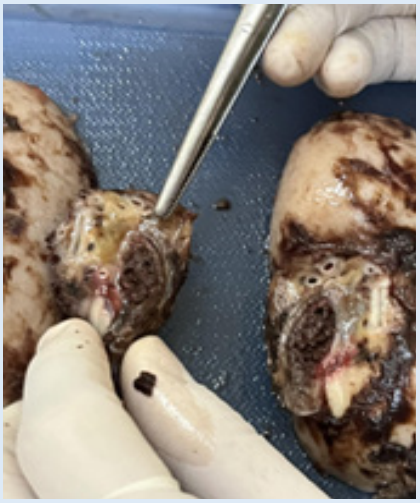

Figure 13: Allograft kidney showing blood clot in ureter in close up view with normal renal artery and vein.

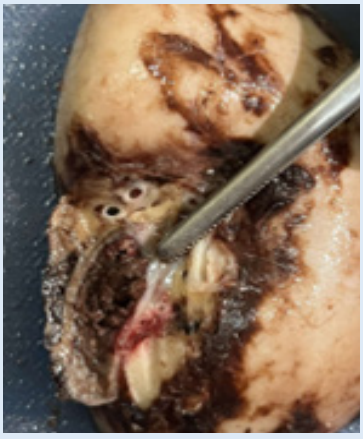

Figure 14: Allograft kidney showing blood clot in ureter in close up view with normal segmental renal arteries and vein.

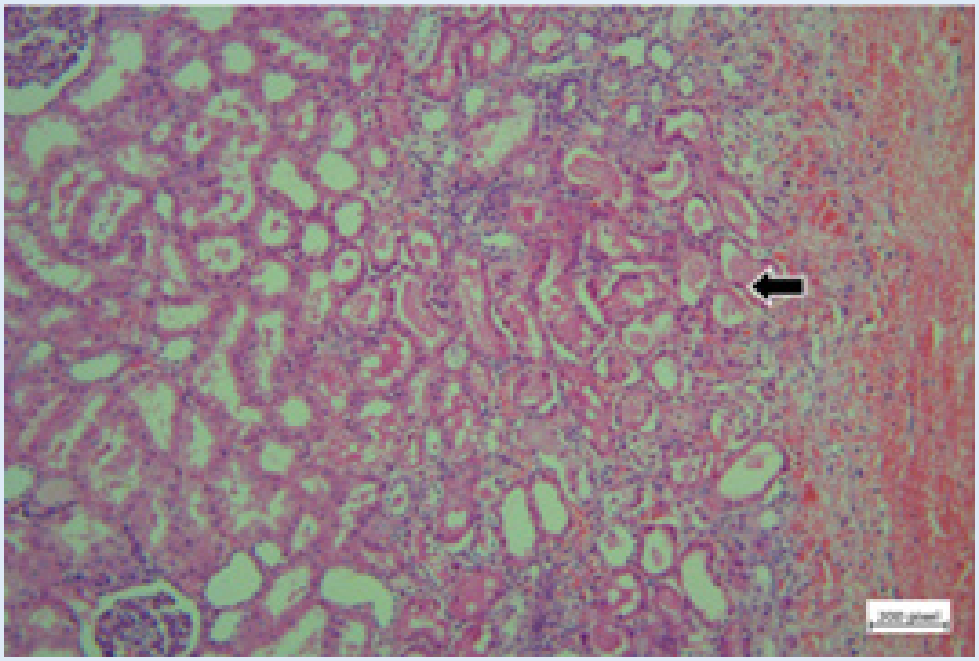

Figure 15: Flattening of proximal tubular lining cells with few casts, hemorrhages and necrosis at the site of rupture (H&E, 10x).

Discussion

The patient had oliguria (100 cc) immediately after transplant surgery; immediate non-functioning graft. The usual cause of oliguria during transplant surgery is hyper acute rejection provided that hemodynamic status is stable. In this patient, the surgical team did notice 50 cc of urine after ureter anastomosis; and there was no evidence of hyper acute rejection as the gross appearance of grafted kidney was normal.

The possibilities of oliguria immediately after anastomosis of ureter were as follows

a. Hyper acute rejection which usually happens at the time of vascular anastomosis of renal artery.

b. Accelerated acute rejection.

c. Acute tubular necrosis.

d. Mechanical causes like vascular thrombosis.

e. Fluid collection around the transplanted kidney.

As the doppler USG revealed normal cortical echo with intact cortico-medullary junction and normal color flow up to the cortex, mechanical causes were excluded. The Resistive Index was 0.58, normal limit (normal range of Resistive Index was 0.5-0.7). Therefore, the possibilities were accelerated acute rejection and acute tubular necrosis. Thus, methylprednisolone was extended up to 5 days, the guideline being 3 days. Graft biopsy done on third post operative day did not show features of acute rejection or acute tubular necrosis; only glomerular ischemic changes seen. The patient had persistent anuria for 72 hours with euvolemic fluid status clinically and stable blood pressure. What would be the likely aetiology for persistent anuria for 72 hours? Did we miss pathological features because of tiny needle biopsy specimen?

The answer was "No" because histology of cone biopsy was exactly the same as needle biopsy. What would be the likely cause for persistent anuria for 4 days? On 4th post-transplant day, the patient developed hypovolemic shock and hemoperitoneum. As no active bleeding point was seen, the cause was likely to be due to DIC. The renal artery, vein and ureter of grafted kidney were normal. The kidney was congested, and the consistency was slightly soft. The only unusual finding was "slightly soft consistency of grafted kidney". We did not get a clue for persistent anuria for 4 days with "slightly soft consistency of grafted kidney". We were lost as the wedge biopsy showed no evidence of acute rejection or acute tubular necrosis. The patient was in state of anuria till day 13 though anemia, fluid and electrolytes status were managed well; and septicemia was handling our best. The patient was tried with anti-rejection regime; the clue was lost. On Day 13, the patient had second attack of hemoperitoneum causing hypovolemic shock with septicemia and DIC; the cause was rupture of grafted kidney. Spontaneous renal allograft rupture is one of the most dangerous complications of kidney transplantation requiring timely diagnosis and immediate surgical intervention. In this patient, we have to congratulate our team for getting early diagnosis of hemoperitoneum and possible graft rupture; because clinical diagnosis depends on pain, oliguria, hypotension and CT scan or USG finding showing peri-renal fluid collection and tear [9,10].

The graft nephrectomy was done for anuria for 13 days with maximum anti-rejection therapy and also for life saving. Histology was compatible with acute T cell mediated rejection. It was clear that the reason for rupture was cell mediated rejection; however, it was debatable that histological evidence of cell mediated rejection developed late- Day 13. It became overt with septicemia. Septicemia may be one of the predisposing factors for rejection. There has been a debate on the management of renal allograft rupture "graft nephrectomy versus salvaging" and the transplant team want to save both patient and grafted kidney with modern

immunosuppression therapy. Several case reports on salvaging the grafted kidney with successful results were mentioned [1,11,12]. However, none of the cases had immediate and persistent non-functioning graft (persistent anuria) since transplant for nearly 2 weeks. This patient had persistent prolonged anuria, DIC, septicemia, collapse right upper lobe followed by consolidation, and recurrent hemoperitoneum.

The most common causes of renal graft rupture are acute rejection and vein thrombosis, and rarely with acute tubular necrosis. Renal graft rupture may result from interstitial damage due to the prolonged warm ischemia time during the transplant and to post-transplant acute tubular necrosis in the absence of graft rejection [7]. In this patient, warm ischemic time was normal; thus, cell mediated rejection was the main culprit causing spontaneous rupture. To avoid allograft rupture, use of well-matched, good quality kidneys was recommended particularly in deceased kidney transplant. Moreover, a technically satisfactory transplant procedure with short cold and warm ischemia times was essential to produce good graft function. Mean cold ischemia time (CIT)in one study which involved over 300 recipients was 22 h, 28 min (range 15 h, 16 min to 40 h); and it was not related with renal allograft rupture [4]. HLA match was 1 antigen (AG) for 3, 2 AG for 8, and 4 AG for 1 (mean 1.9). Nine patients had delayed or declining renal function requiring dialysis. The panel reactive antibody was at peak, mean 47% (range 0-100%) and current, mean 18% (range 0-84%). Six of 12 had OKT3 therapy at time of RAR and six had biopsies. Day of RAR was mean 10, median 9 (range 4-21).

In addition, a large donor-recipient age gradients were related with delayed graft function [8,13]. In this patient, her age was 48 years and the daughter was 29 years. There were several debatable points for us, low resource setting area. It is questionable that "What if we did repeat third biopsy on day 7 and it showed acute cellular rejection?" "What if we did repeat anti-thymocyte globulin infusion though the patient had first dose prior to transplant?" "Was it safe to repeat anti-thymocyte globulin?" We gave IV immunoglobulin on Day 3 with the presumed diagnosis of antibody mediated rejection. Was the IV immunoglobulin aggravating acute cellular rejection? The patient was DSA negative initially. What should we do in resource poor setting? Repeating DSA could give good information like rising titer; however, it was expensive. The important point highlighted in one report was that acute tubular necrosis (ATN) and rejection act synergistically to cause renal allograft rupture (RAR); therefore, early delayed function requires intensive and perhaps novel immunosuppression, especially in Black recipients [4]. Early delayed graft function requires intensive and perhaps novel immuno suppression because it is a risk factor graft rupture. This patient had non-functioning graft which started during transplant surgery, and it was persistent for nearly 2 weeks- till rupture.

Conclusion

Spontaneous rupture of allograft kidney due to acute T cell rejection is relatively rare with the advance of newer immunosuppressants. Clinical awareness is important to get early diagnosis of hemoperitoneum and timely intervention to save both patient's life and graft kidney. Although transplant biopsy, histopathology (including immunohistochemistry and immunofluorescence studies), is still the key for diagnosis, acute cell mediated rejection is not seen in both needle biopsy and open wedge biopsy in this patient. Donor nephrectomy may save the life if the rejection is refractory to anti-rejection medication and recurrent hemoperitoneum endangering life. Septicemia may aggravate kidney rupture (Figures 16-32).

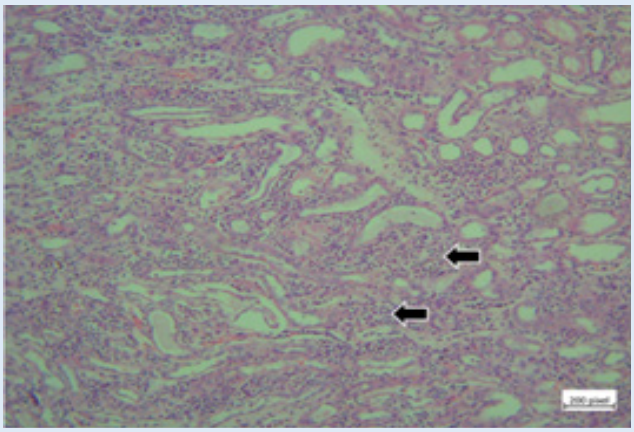

Figure 16: Moderate infiltration of mixed inflammatory cells into the interstitium with widening (H&E, 10x).

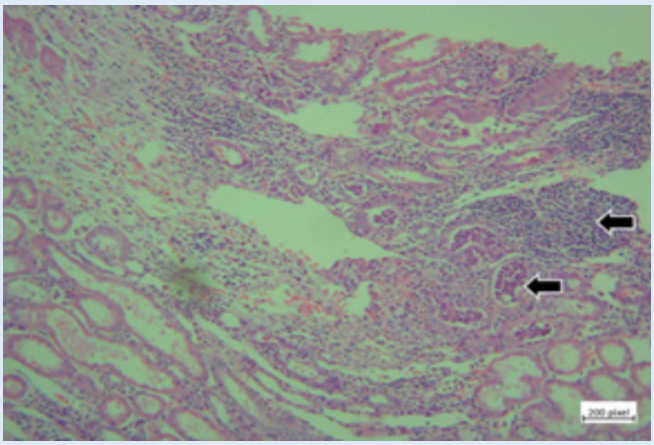

Figure 17: Lymphoid aggregates in the interstitium and neutrophil casts in the lumen (H&E, 10x).

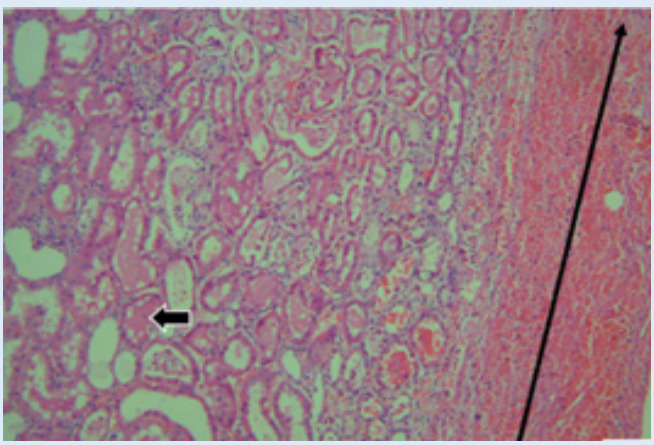

Figure 18: Massive hemorrhages and tubular casts at the site of rupture (H&E, 10x) [Although some flattening is noted, all tubular lining cells are viable].

Citation: Khin Phyu Pyar*, Tin Moe Mya, Ohmar Hlaing, Yu Aye Latt, Chan Myae, et al. Matched Living Kidney from Twenty-Five Years Old Daughter Having Persistent Anuria for 13 Days Since Ureteric Anastomosis Developed Spontaneous Renal Allograft Rupture Due to Acute T Cell Mediated Rejection Producing Hemoperitoneum and Shock on Day 13 After Transplant: A Case Report. J Clin & Commun Med 4(4)-2022. JCCM.MS.ID.000195. DOI: 10.32474/JCCM.2022.04.000195

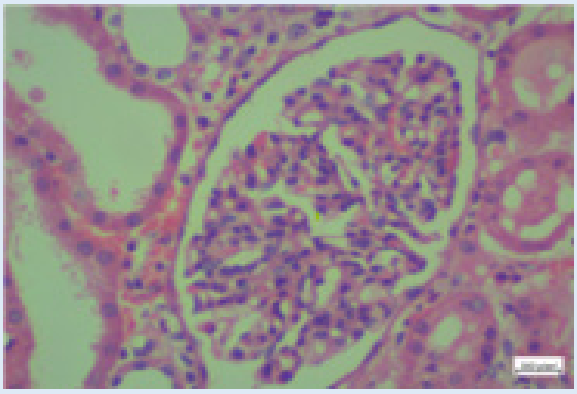

Figure 19: No/minimal change in the glomerular morphology and normal capsular space (H&E, 10x).

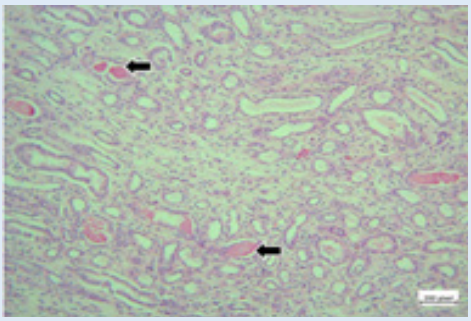

Figure 20: Protein casts are noted in some lumen of tubules in the renal medulla (H&E, 10x).

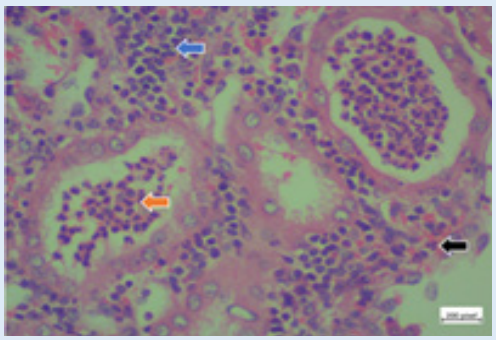

Figure 21: Intraluminal neutrophil casts, interstitial inflammation and peritubular capillaritis (H&E, 10x).

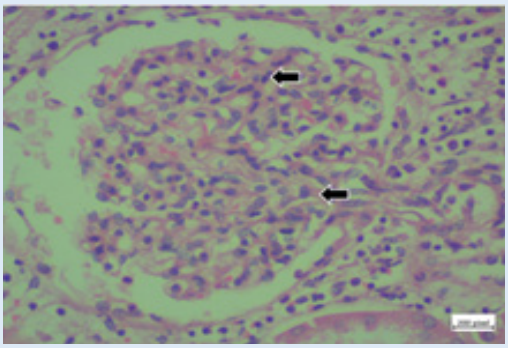

Figure 22: Glomerular tuft with mild mesangial proliferation (H&E, 10x).

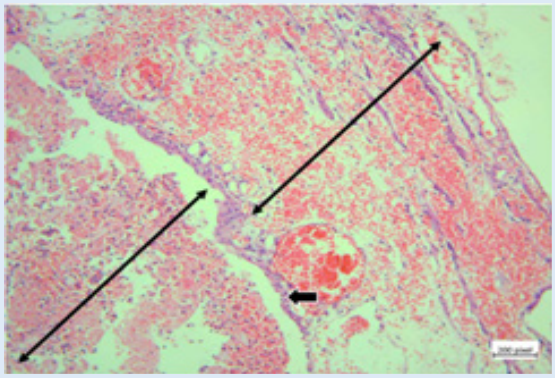

Figure 23: Blunting of urothelium in the ureter with massive intraluminal, intramural hemorrhages and ischemic injury.

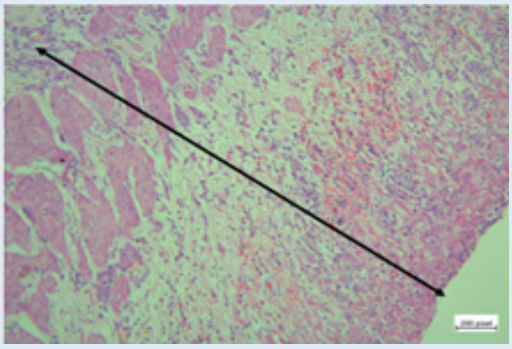

Figure 24: Moderate degree of inflammatory infiltrates in all layers of vascular wall (More pronounced in tunica intima and media) (H&E, 10x).

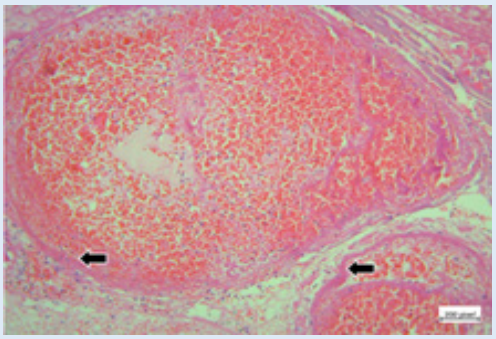

Figure 25: Vascular dilatation with congestion and wall necrosis (H&E, 10x).

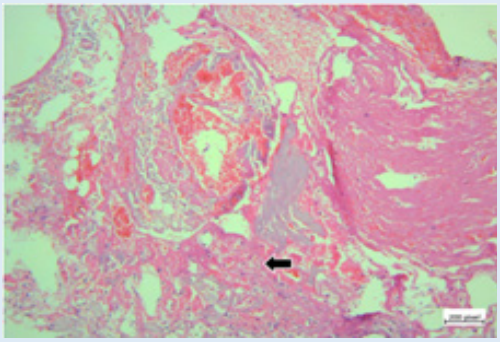

Figure 26: Fibrinoid necrosis in the vascular wall in the hilar area (H&E, 10x).

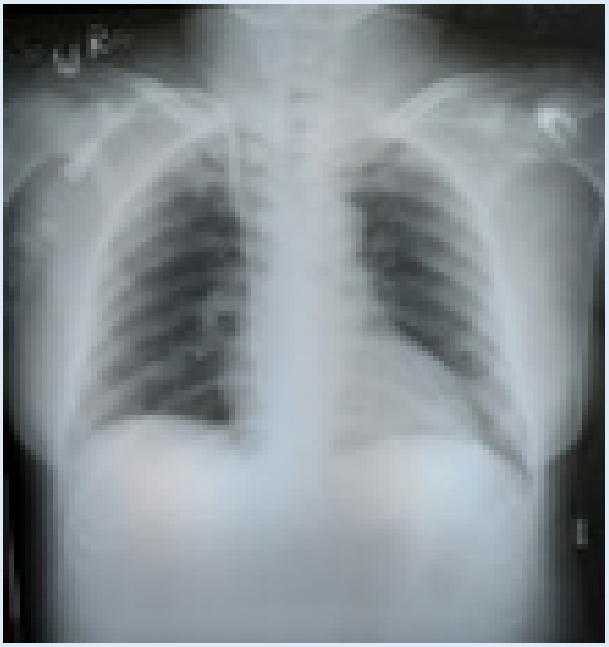

Figure 27: Immediate post-op day CXR (AP view) on 22.4.2022.

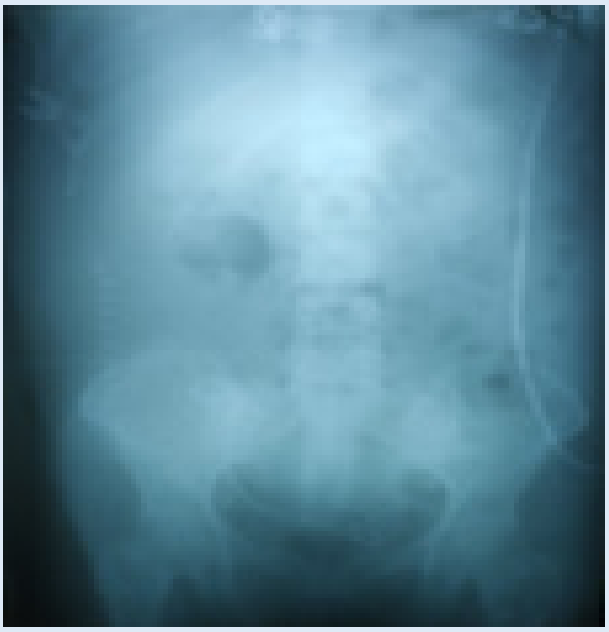

Figure 28: 25.4.2022, Day 3 (after CECT abdomen) No nephrogram or excretory phase in plain Xray 4 hours after intravenous contrast.

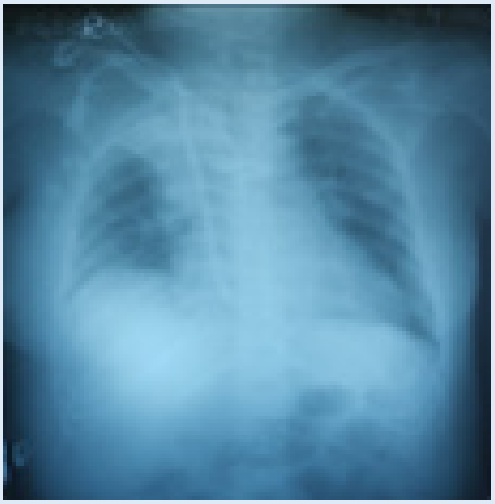

Figure 29: 28.6.2022 Morning before bronchoscopy.

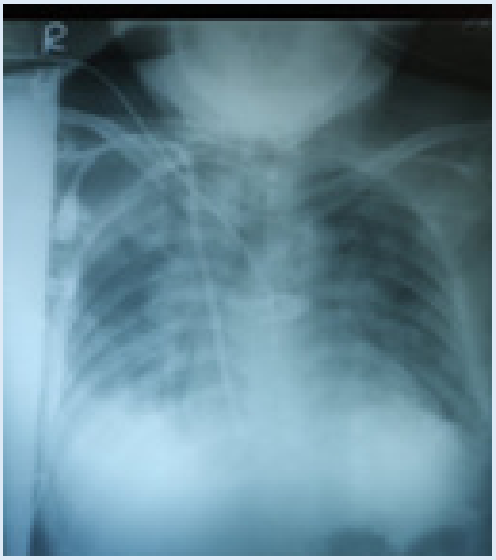

Figure 30: 28.6.2022 After bronchoscopy.

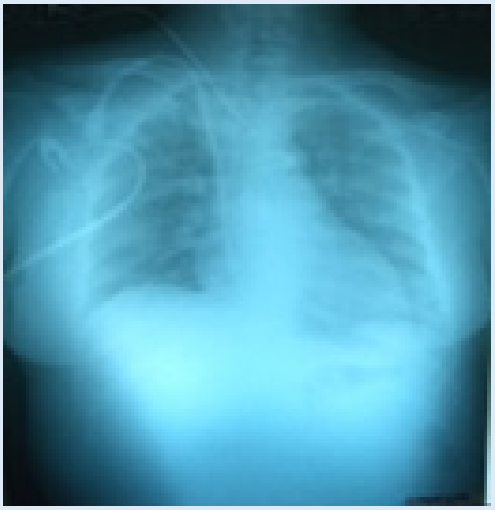

Figure 31: Re CXR on Recheck CXR (AP View) on 3.5.2022.

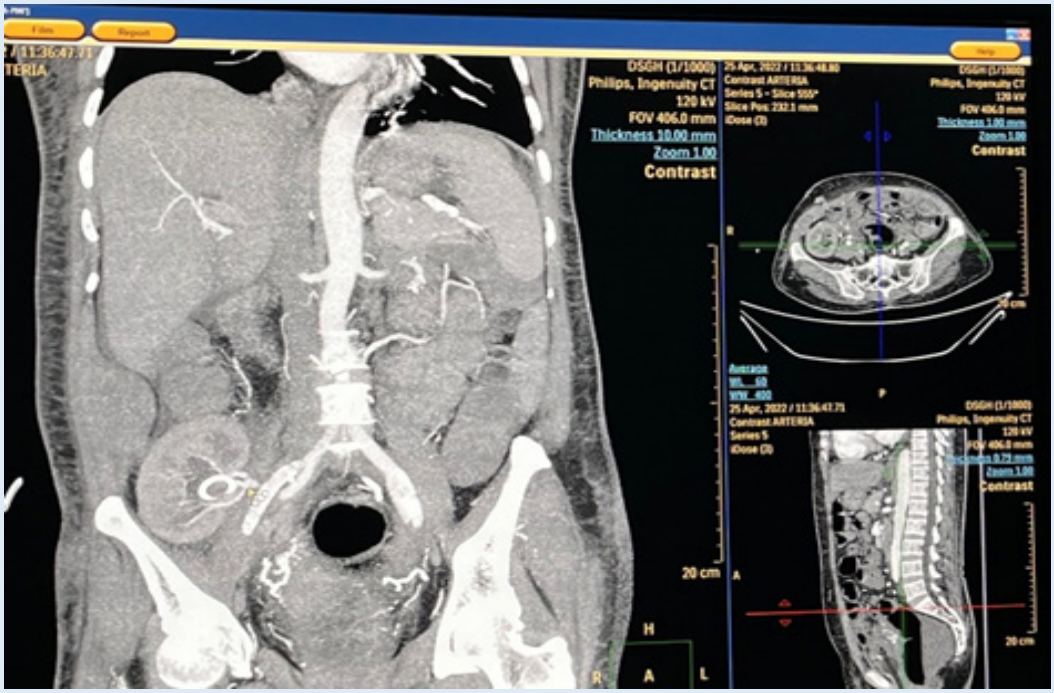

Figure 32: CT renal angiogram showing normal renal artery and vein.

Compliance with Ethical Standards

Acknowledgments

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Disclosure of conflict of interest

The authors declared no potential conflicts of interests with respect to authorship and publication of this article.

Statement of informed consent

This study was approved by Hospital Research and Ethic Committee from Defence Services General Hospital (1,000 Bedded) Mingaladon, Myanmar. Informed consent was also taken from the patient.

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