Clinicopathological Conference Report— PM 25821

Lupus Vasculopathy: Is It all that Benign?

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Chief complaints: C/O pain abdomen with vomiting – 2 days

HISTORY

1997: C/O swelling of both lower limbs with facial puffiness and hypertension (requiring 10 mg once daily). Diagnosed as lupus nephritis (Light microscopy: MPGN pattern with dsDNA positive) induced with pulse cyclophosphamide and steroids. Was started on maintenance with azathioprine and steroids. Probably had achieved some remission.

2006: Diagnosed as diabetes mellitus (? Possibly Steroid induced). Was managed with insulin therapy from then onwards.

August 2011: Had relapse of proteinuria and diagnosed as nephrotic flare of lupus nephritis and renal biopsy done. Biopsy was suggestive of some activity with significant chronicity with chronic thrombotic microangiopathy. Patient was managed with high dose steroids and mycophenolate mofetil (MMF) for 6 months followed by azathioprine and steroids as maintenance therapy.

April 2012: Had history of cough with sputum production and was diagnosed as pulmonary tuberculosis. Received 6 months of anti-tubercular treatment (ATT).

June 2013: Had an increase in serum creatinine from 1.3 mg/dL to 1.8 to 2 mg/dL and was clinically diagnosed

as flare and started on 1 mg/kg/day prednisolone and MMF.

August 2013: Fever, headache, and swelling of face. Computed tomography (CT) was suggestive of pan sinusitis. Endoscopic PNS scabbing showed growth of Rhizopus. Was managed with conventional amphotericin initially, then shifted to liposomal amphotericin due to worsening renal function (sr creatinine rise from 1.7 mg/dL to 4.3 mg/dL) (MMF). Developed pneumonia during the stay and was diagnosed as pulmonary TB (BAL AFB+). Anti-tubercular treatment started and liposomal amphotericin B (LAMB) converted to conventional amphotericin for financial reasons.

Final admission (12th Dec 2013): 4th Dec nasal scraping negative for mucor and amphotericin was stopped. Patient had presented with complaints of abdomen pain with vomiting of 2 days duration. Pain was mainly in the epigastric region, associated with h/o constipation. No history of decrease in urine output. No H/O melena or blood in vomitus.

O/E: Patient was sick looking. Pallor (+), Pedal edema (+). BP: 130/86 mm Hg, PR: 98/minute, RR: 18/minute. CVP 18 cm of normal saline @ admission. S/E: Tenderness (+) in the epigastrium with guarding present. Chest: B/L NVBS with no added sounds. CNS & CVS: WNL.

Investigations (Tables 1 and 2). 16.12.13: PTi=68%. APTT: 66 (25–36). Fibrinogen: 2.89 (2–4). D-Dimer: Positive
Radiology: NCCT PNS (5th Dec 2013): Thickening of mucosa of sinus present.

CECT abd: S/O Pancreatitis with collection present anterior to the pancreas. Consolidation (+) left lower zone with pleural effusion.

Nasal scraping: Negative for mucor. iPTH: 15 pg/mL, Vit-D: < 3 IU, Sr cortisol (8 AM):

1140 ug/dL (on hydrocortisone at 7 AM). C3=41 mg/dL (73–135) C4=36 (9–36).

T3/T4: 0.73/3.90 TSH: 9.13. Blood C/S: sterile.

Urine R/E: Alb ++ to +++ with no sediments. CRP: 1070 mg/l. Blood C/S: Sterile.

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Table 1: Investigations

	10.08	13.08	26.08	03.09	12.12	14.12	16.12	18.12	19.12
Hb	7.4	7.8	6.1	9.0	10.2	9.4		7.5	8.7
TC	3300	4900	12200	5100	12600	6700		21700	20800
DC	70/24/3/2		70/25/3/2	78/18/2/2				80/15/2/3	
PC	136000	172000	84000	93000	349000	234000		156000	149000
Sodium	138	137	130	140	139	145	146	143	
Pottasium	2.8	5.5	3.8	3.8	3.9	5.2	4.8	3.5	
Chloride	112	114	105	111	96	113.6	116	112	
S Cr	2.0	1.89	3.90	2.89	5.2	5.3	6.7	4.8	
B Urea	72	70.6	127	166	217	205	223	132	
TP	3.8	4.3	—	—		3.9	3.4	3.2	
ALB	2.1	2.5	—	—		2.0	1.5	1.6	
Ca	8.1	8.6	9.5	—		10.8	9.2	9.6	
PO	4.2	4.91	—	—		7.3	12.9	4.9	
AKLP	728	567	—	—		121	247	270	
Amylase	—	—	—	—	1719	1483			
Lipase	—	—	—	—		1981			
OT/PT	15/03	20/10	—	—		121/39	41/17	33/09	
TB	0.1	0.3	—	—		1.4	291/2.1	5.5/4.2	
TC/TG	—	—	—	—		154/133			
pH	7.37	—	—	—	7.24		7.22	7.31	
HCO ₃ ⁻	19	—	—	—	11.6		6.2	13	
CO ₂	39	—	—	—	27		15	26	
O ₂	97	—	—	—	55		105	85	
Mg	1.98	—	1.65	1.65	2.3	2.2	—	—	

Table 2: There is a significant association of pancreatitis with steroid use

<i>SLE pancreatitis</i>	<i>ATT-induced pancreatitis</i>	<i>Mucormycosis pancreatitis</i>	<i>Tuberculosis pancreatitis</i>	<i>IgG4 pancreatitis</i>
H/O SLE (+++)	H/O drug use (+)	H/O Mucor (+++)	H/O TB (+)	Autoimm Dis (++)
↓C3 (++++)	2 successful use (+++)	Immunosuppressed (+)	Immunosuppressed (+)	Worsening of renal disease (+)
Stoppage of GC (+)	Occurring at 4 months (+++)	Successful Tx (?) (+++)	On ATT (+++)	Late occurrence (+++)
Renal dysfunction (±)	Severe pancreatitis (+)	CT Picture (+++)	Ac pancreatitis (++)	Ac pancreatitis (+++)
dsDNA –ve (+)		Stoppage of GC (+)	CT Picture (+++)	No EHB invt (+++)
No activity				

Course during Stay

Patient presented with C/O abdomen pain with vomiting and diagnosed as a case of pancreatitis (? Gallstone related *vs* drug-induced pancreatitis). Managed by nil orally and analgesics. Hypocalcemia was managed with calcium gluconate. Patient had episode of hypoglycemia and insulin was put on sliding scale. Patient received levofloxacin for 2 days. On 17th Dec onwards patient had developed drowsiness and then antibiotics changed to Pip+Taz. Contrast-enhanced CT was suggestive of severe pancreatitis with left lower zone consolidation. Patient was started on dialysis on the suspicion of uremia. During hemodialysis patient had respiratory distress and hypotension. Patient was intubated and was shifted to

MICU. Was started on inotropes and gradually increased. Vancomycin, metronidazole, and I/V hydrocortisone were added on 18th Dec 2013. Patient condition deteriorated and had cardiac arrest and CPR was carried out. However, patient could not be revived and was declared dead on 19th Dec 2013.

UNIT'S DIAGNOSIS

K/C/O Systemic lupus erythematosus (SLE) with lupus nephritis with CKD-stage III/IV

K/C/O Invasive mucormycosis received amphotericin (7 gm).

Acute pancreatitis (cause-?Gallstone *vs* drug induced) with sepsis with refractory septic shock.

CLINICAL DISCUSSION (DR. RAJA RAMACHANDRAN)

In a known case of lupus nephritis and diabetes mellitus (poor control) with H/O invasive mucormycosis treated with amphotericin B (7 gm), the patient presented with pain in abdomen, vomiting, hyperamylasemia, renal failure, and leukocytosis.

Causes of Hyperamylasemia could be

- Acute pancreatitis
- Mesenteric ischemia
- Intestinal obstruction
- Peritonitis.

In view of site of pain and high amylase and lipase (>500) are highly suggestive of lupus-related acute pancreatitis. However, in this setting of SLE with H/O mucor, the patient was treated with AmB and tuberculosis on ATT. Other possibilities which should be considered are:

- ATT/Steroid/AmB induced pancreatitis
- Pancreatic mucormycosis
- Tuberculosis pancreatitis
- Pancreatitis unrelated to SLE/Mucor/Drugs
- IgG4-related disease – Lupus-like disease.

On investigation points favoring lupus pancreatitis are:

- H/O SLE with nephritis
- Low C3 level
- Presence of hyperbilirubinemia
- Hypoalbuminemia
- Leukocytosis
- Disease occurring after GC withdrawal
- Pleural effusion and ascites.

And points against it are dsDNA negative and no other clinical activity elsewhere.

Pancreatitis in SLE seen in 0.7 to 4% J Lariño Noia et al (2009), Ruchika Goel et al (2012) cases and dsDNA is positive in 82 to 88%, Low C3 is universal in LAP J Lariño Noia et al 2009. Hypoalbuminemia, hyperbilirubinemia, renal involvement (85%), and leukocytosis are universal in patients with mortality.

Anti-tubercular treatment-induced pancreatitis was considered. There are at least 25 and 11 cases of AP induced by rifampicin and isoniazid respectively (Sarah Mattioni, JOP 2012). Acute pancreatitis due to isoniazid and rifampicin develops 0.5 to 35 days after starting (Sarah Mattioni, JOP 2012). Always occurs with the first course of therapy. INH pancreatitis is mild-moderate (Briongos-Figuero LS 2007). Patients with diabetes mellitus are at high risk of developing rifampicin AP. On re-challenge ATT-induced AP is diagnosed.

Use of ATT suggests possibility of ATT-induced pancreatitis but one previous successful use of ATT and occurring after 4 months of commencing ATT are point against this possibility.

Usually seen in middle-aged to elderly group with co-morbidity like alcoholic, ischemic heart disease, consumption of other drugs, gallstone disease, and hyperlipidemia (Omid Sadr-Azodi, JAMA 2013).

The risk is highest during 4 to 14 days of starting steroids, then gets attenuated.

Prednisolone and betamethasone are associated with highest risk. With other steroids, no significant association was found (Omid Sadr-Azodi, JAMA 2013).

Use of steroids with the presence of amylasemia would favor steroid-induced pancreatitis but points against were:

- Age
- Absence of co-morbidity
- Occurring after many years of GC therapy
- Stoppage of GC 6 to 8 days before pancreatitis
- Hydrocortisone preparation used.

Another possibility for pancreatitis could have been amphotericin induced, however, points against it were

- Occurring after 4 months of AmB use
- Occurring after stopping AmB.

Gastrointestinal (GI) mucormycosis could also have resulted in similar features; however, it is extremely rare, reported in few cases of AIDS, renal transplant, and SLE with stomach, colon, and ileum are the most commonly involved sites. Pancreatic mucormycosis is very rare, and isolated pancreatic mucor would be extremely uncommon. Usually GI mucor is a part of disseminated disease and often diagnosed post-mortem.

Points in favor could be that the present status was suggestive of activity on CT scan of PNS done. Negative nasal smear does not discount mucor infection.

Another possibility could be part of IgG4-related autoimmune pancreatitis, keeping in mind confirmed pancreatitis in setting of autoimmune disease with renal involvement, acute pancreatitis, however, presence of disease in presence of chronic immunosuppressive therapy.

And absence of extra hepatic biliary involvement, occurrence after 16 years of SLE onset, patient was on ATT with improvement in general condition. Steroids were off, presenting only as acute pancreatitis and CT picture of the patient.

Analysis renal part seemed most likely to be SLE class VI. Else, it could be any other cause of acute or chronic disease, disease activity related, amphotericin induced, acute pancreatitis related, and combination.

Causes of acute kidney injury could be as follows (Flow Chart 1):

Flow Chart 1: Acute factor

Cause of Terminal Deterioration can be

- Sepsis with septic shock (1° ? Respiratory tract infection)
- Sudden cardiac arrest
- Sepsis related end organ failure
- CAD: Long term GC use, Menopause 3 years ago, SLE
- Combination.

Analysis of 17 SLE patients analyzed from 1998 to 2007 revealed the causes of death were primarily due to SLE, or due to infection or those that were multifactorial.

- 7 (41%) died of SLE-related complications
 - 3 (16%) died of infections
 - 7 (41%) died of infections + SLE complications
- Hence, in a known case of SLE with lupus nephritis:
- Iatrogenic Cushing's syndrome and GC-induced diabetes mellitus
 - H/O Mucormycosis of the nasal and paranasal sinus
 - H/O Pulmonary TB on ATT
 - Acute pancreatitis with significant EPN
 - (?) SLE related/(?) ATT induced/(?) Mucor pancreatitis
 - Ac on CKD (Ac-Drug/Pancreatitis/Sepsis)
 - Sepsis with septic shock with DIC (?) - HAI/LRTI - Bacterial, followed by Mucor/Aspergillus/TB/CMV/Combination of these (?)
 - Fatty liver.

CLINICAL DISCUSSION

Dr. KL Gupta: Raja has discussed the long course of the patient and particularly stressed on pancreatitis. She came to us when she was not married, disease came down, she got married, and had two children. She had stress-related diabetes mellitus. Patient had waxing and waning of the disease. Financially she was poor. I started her on MMF. The main point I would like to stress on is nasal mucormycosis which was persistent. Despite two doses I expect to see disseminated mucor beside nasal region, in the lungs. And pancreatitis may also be related to that.

In addition, tuberculosis is definitely there; her sputum was positive for tuberculosis.

Speaker 1: Good morning, although Dr. Raja has mentioned that most common cause for demise was disease activity and infection, I do not disagree with that, but in this patient evidence is tilting more toward infection rather than disease activity because dsDNA was negative, only C3 was low, C4 was normal, there was active sediments in the urine, CRP was highly activated. I expect to see multiorganism infection with combination of TB and fungal infection. If we see disease activity histologically on autopsy, this is not contributing to the clinical presentation of the patient.

Speaker 2: As far as pancreatitis is concerned autoimmune pancreatitis still will come into the picture. Types I and II are two varieties. Type I is usually related to IgG4-related disease. Type II can present with normal IgG4. As far as early menopause is concerned, she had received cyclophosphamide that can cause early menopause.

Speaker 3: This patient also had significant hypoglobulinemia, the globulin ranged from 1.7 to 1.8 g/dL. This patient was severely immunocompromised probably because of medication. With previous kidney report I will support infection as the cause of demise rather than activity.

Dr. Sinha: It is rightly pointed out that type II autoimmune pancreatitis is not associated with IgG4-related disease but almost 95% of these patients will have biliary obstructive disease. Peripancreatic collection is extremely rare. Both type I and II to my mind is not a possibility.

Speaker 4: Though the patient appears to have pancreatitis, lipase and amylase levels were low because of renal failure, it is excreted by kidney. In such cases estimation of trypsin and chymotrypsin from stool will be more confirmative. We do not have such facility; in that case putting a small amount of stool over the X-ray film and clearing of that area provide the clue for pancreatitis.

Chairperson: Any more comments.

Dr. Sanjay Jain: I think diabetes is the only point which is remaining and I do not believe that this is steroid-induced diabetes, it is type I diabetes which is the cause of demise.

Speaker 5: Possibly type I diabetes cannot be ruled out and is less likely in this case. The patient is on high dose of steroid, not requiring insulin for long time. In addition to this, vitamin D level is very low and PTH is raised. We do not have explanation for that.

Dr. Chakrabarti: With lung shadow I do not think it is rhinocerebral fungus. It does not go for dissemination except for orbital extension.

Speaker 6: With one BAL positive and patient being immune suppressed, I would still think TB should be considered. If a colleague says that pancreatitis is not there, then it could all be intra-abdominal TB.

Figs 1A to D: Biopsy from nose showing: (A) inflammation, (B and C) necrotising inflammation with presence of (D) aseptic wide angle branching hyphae consistent with Mucormycosis (H&E a-c, Grocott's-d, 10×-a-c, 40×-d, Original magnification)

Chairperson: We are told that it is unlikely to be pancreatitis.

Dr. SC Varma: I personally feel that radiological picture and clinical story is very much consistent with acute pancreatitis and issue would be etiology. Dr. Raja talked about DIC, what I want to tell is that the mere presence of D-dimer does not mean DIC. Raised D-dimer occurs in any type of organ injury and can be associated with pancreatitis.

PATHOLOGY PROTOCOL (DR. RITAMBHRA)

S-6580/1997 – Non representative, S-8171/1997 – MPGN, S-12301/2011 – Lupus Nephritis IV, chronic TMA, ANS, S-16280/2013TBLB – Non specific changes, 16870 – Nasal cavity – Allergic fungal rhinosinusitis – Mucormycosis (Figs 1A to D).

Partial Autopsy (PM 26821) was performed. Kidneys (wt 290 g): showed patchy cortical discoloration (Fig. 2). There were tiny infarcts. Histomorphology showed multiple areas of small necrosis in the background of fibrosis. Glomeruli (50%) show mesangiocapillary pattern with endocapillary proliferation. Arterioles and arteries show chronic thrombotic microangiopathy

Fig. 2: Gross of kidneys shows blotchy cut surface with evidence of discolored infarcts

with vasculopathy. IF on paraffin: IgG C1q, IgM kappa, lambda 2+ in capillary loops + mesangium, Arteries also show C3C1q, kappa, lambda. Lupus nephritis IV C/a, lupus vasculopathy and thrombotic microangiopathy (Figs 3 to 6).

Pancreas: showed areas of necrosis as whitish specs extending onto mesentery and omentum (Fig. 7), which

Fig. 3: Photomicrographs confirm presence of cortical infarcts. (H&E, 10× original magnification)

on microscopy showed duct obstructive changes with chronic interlobular and, fat necrosis, intralobar, and interlobular inflammation and fibrosis. Arteries show vasculopathy and vasculitis which had deposition of immunoglobulins (Figs 8 and 9).

Liver (wt 1620 gm) was pale and firm. There were no gross lesion. Microscopy showed only fatty changes.

Spleen (wt 120 gm) was grossly normal and microscopically showed white pulp depletion.

Lungs (wt 910 gm) were subcrepitant with brownish discoloration and had bronchocentric whitish nodules 0.5 to 1.5 cm, especially in both upper/lower lobes (Fig. 10). Microscopic evaluation showed bronchocentric chronic invasive granulomatous aspergillosis (Fig. 11) with foci

Fig. 4: Photomicrograph shows MPGN Pattern with hyaline thrombi and wire loops which were positive for all immunoglobulins, light chains (IgG, IgA, IgM, Kappa, Lambda) and fibrinogen in crescents along with hyaline occlusion of arteries which were also immunoglobulin positive. Extraglomerular positivity in tubules was seen (H&E, Pas, Massons trichrome, Immunofluorescence FITC, $\times 4-40$)

of old fibrocaceous tuberculosis (AFB positive) (Fig. 12). *Heart* (wt 334 gm) chambers and valves were normal. There was focal discoloration of myocardium. There were old healed infarct in the myocardium with organized thrombus and endotheilitis (Figs 13 and 14).

GIT showed superficial congestion and serosal fat necrosis.

FINAL AUTOPSY DIAGNOSIS (PM NO. 25821)

Known Case of Lupus Nephritis on Treatment

- Lupus nephritis class IV chronic/active, acute cortical necrosis, lupus vasculopathy and chronic thrombotic microangiopathy.
- Acute or chronic pancreatitis with lupus vasculopathy and vasculitis.
- Chronic invasive granulomatous aspergillosis, old fibrocaceous tuberculosis.
- Steatosis liver.
- Old healed infarct in myocardium with organized thrombus.

Fig. 5: Presence of vasculopathy in the form of hyaline subendothelial deposits in the arteries of varying sizes (H&E-a&c, PAS-b, Massons Trichrome-d, $\times 10$ -a, $\times 20$ b-&d-40, $\times 20$ -c, Original magnification)

Fig. 6: (Lt side) presence of IgG in walls of normal arteries and (Rt Side) IgA and IgM in subendothelial hyaline deposits in lumen of arteries of vasculopathy (Immunofluorescence-IgG, IgA ,IgM, $\times 20$, Original magnification)

Fig. 7: Gross shows enlarged liver and fat necrosis as white specs on the pancreas

Fig. 8: Areas of fat necrosis and intralobular fibrosis with one artery showing ectasia with necrosis (H&E, $4\times$ original magnification)

FINAL AUTOPSY DISCUSSION

Dr. V Sakhuja, Chairperson: The main problem was with the basic disease which has spread causing vasculitis, and pancreatitis looks like related to the main disease itself. Any comments please...

Dr. Manish Rathi: Although Mam you have said that it is 4A and C, both chronic and active or c or a, but what you have shown here is all chronicity, you have not shown any activity. Why are you putting A also, why not just 4C ? That was my first question. My second

Figs 9A to D: Segmental necrosis with ectasia in medium sized intrapancreatic artery (H&E, PAS, MSB, Massons trichrome, 40× Original magnification)

Fig. 10: Gross of lungs shows bronchocentric necrotic nodules

Fig. 11: Necrotizing granulomas with suppurative centres which had (inset) septate fungi consistent with Aspergillosis in giant cells (H&E, inset- 4× inset 40×, Original magnification)

question is on lupus vasculopathy. You have shown lupus vasculopathy and there is no doubt that there is lupus vasculopathy but whether this lupus vasculopathy is acute or chronic? Because whatever classification

system we have they are silent on the vascular involvement in lupus ... they do not put either as chronic or active. Presence of acute cortical necrosis can put toward the acute nature of the disease, otherwise

Fig. 12: Necrotizing granulomas of tuberculosis with AFB positive bacilli (H&E, inset-Zeil Nelson stain, 40 $\times$, inset 100 $\times$ original magnification)

Fig. 13: Section of heart shows old infarct and artery (inset shows arteries and luminal narrowing. (H&E, 10 $\times$, inset 19 $\times$ original magnification)

Fig. 14: Area of myocardial infarct showing fibrosis and artery with luminal narrowing. (H&E, 40 $\times$, inset 20 $\times$ original magnification)

immunoglobulin deposition and other manifestations can be in chronic also.

Dr. Ritambhara Nada: I thought, when we saw the first biopsy, arteries were absolutely normal; in second biopsy we have chronic TMA changes, there were no hyaline deposition. When we see on autopsy along with chronic TMA changes there was hyaline deposition which showed some immune deposition. So I thought it has evolved over time rather than was present since beginning...

Dr. Inder: I would like some comment from Dr. Ritambhara and Dr. Chakrabarti about use of the term chronic granulomatous aspergillosis, you mean it is bronchocentric granulomatosis. Otherwise, is it CNPA...at least I am not aware of CGA term.

Dr. Chakrabarti: I was also thinking this term which have been used...Is it in the Pathology? Did you see this chronic invasive? Because in paranasal sinuses, of course, chronic invasive granulomatous aspergillosis is there and in case of the lung earlier it was the term chronic

necrotizing pulmonary aspergillosis. But now as David Daning has shown many of these cases, especially from the post-tubercular cavity, there is colonization ultimately invasion. He tried to coin all these terms into chronic aspergillosis, tried to look much more into details, but I am not clear about this.

Dr. Sanjay Jain: I need more clarification on so-called lupus vasculopathy which you have shown. Naturally there is immunoglobulin deposition which could be immune complexes. We do not have anti dsDNA positivity; most of these complexes are if I am correct are complex of anti dsDNA and ANA. So in a presence of a negative antibody what are these deposits....number 1...question number 2 is if you have deposits you have the active disease because it has lot of significance as far as treatment is concerned. Because has it been a part of TMA, the treatment would be antiplatelets drugs, on the other hand, if you have deposits like this, treatment would be immunosuppression. I'm pretty sure this person is on 16 years follow-up, you will have lupus anticoagulants status, anti cardiolipin, beta 2 glycoprotein. What was the situation on PT, APTT status?

Second about pancreas. You have ductal obstruction. I just want Dr. Sinha to mention what are you going to label it as? Tropical pancreatitis, calcific pancreatitis, or steroid induced pancreatitis, what is it...?

Dr. SC Varma: With demonstration of acid fast bacilli would you call it healed pulmonary TB. Do you think they are dead bacilli or are there morphological characteristic that can say that TB was there treated or not completely gone? My second question is what happens to Aspergillus when you treat these patient with antifungal for long time. This patient had been on amphotericin for long time the reaction....the histological manifestation would be somewhat different between treated *vs* an active infection.

Dr. V Sakhuja, Chairperson: Last comment by Dr. Sinha.

Dr. Sinha: Dr. Ritambhra very clearly put that this is not auto-immune pancreatitis. There are 2 forms, one is form of duct centric chronic pancreatitis which is not there and second is gel granulocytic epithelial lesion that is also not there.

Vasculitis is listed as important and reasonably common cause for acute pancreatitis. To my mind chronic pancreatitis has not been described unless you say that there was repeated episodes of acute pancreatitis, this is sequelae of the same.

Histology is of usual pancreatitis and you can label it as tropical pancreatitis but is not fitting into anywhere.

Dr. V Sakhuja, Chairperson: Dr. Ritambhra kindly make a comment.

Dr. Ritambhara Nada: Autopsy description in patient with pancreatitis describe as presence of lymphocytes and inflammatory cells in the pancreas. But in this case there was vasculitis in addition. As far as vasculopathy was concerned, there are massive hyaline deposits and C3 levels were low which is taken as part of it being active lupus vasculopathy, and not as just being inactive exudation and trapping of these immunoglobulins. Clinically there is no good description of pathology for pancreatitis for fatal cases. Features of low C3 is considered as active lupus.

Dr. V Sakhuja, Chairperson: Thank you very much. Session is closed.

Summarizing in a case of SLE with lupus nephritis there was acute deterioration of renal function due to vasculopathy resulting in cortical infarcts, and acute pancreatitis was also due to vascular inclusion resulting from occlusive vasculopathy.

SUGGESTED READINGS

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