Clinicopathological Conference

A Child with Encephalitic Presentation

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Clinical Protocol by Dr. Debabrata Ghosh

S, a 6-year-old boy hailing from Muzaffarnagar of Western Uttar Pradesh presented with a short history of vomiting and fever for 3 days and seizure and altered sensorium with abnormal posturing for 2 days. The illness started 3 days back with vomiting which was nonprojectile, nonbilious and of uncertain frequency [(?) once, (?) 5-6/day]. About 2 hours later he started having mild to moderate grade fever, intermittent with no associated chills or rigors, upper or lower respiratory symptoms, pus discharge from anywhere or urinary symptoms. Next day he threw a seizure, reportedly, generalized tonic clonic lasting for 5 minutes. Since then he became unconscious. In the next few hours there was recurrence of seizure of same description. A lumber puncture was done outside. Thereafter he started having abnormal posturing suggesting decerebration and so he was referred to the Post Graduate Institute of Medical Education and Research (PGIMER). There was no history of ear discharge, skin rash preceding this illness, dogbite or contact with tuberculosis.

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The child was 2nd of the twins, born at term to a 2nd gravida mother. The first twin died after 11 days of birth. This baby cried 2 hours after birth. Thereafter development was normal. On presentation, the boy was a student of class 1. He was unimmunized. On examination, at admission, this child weighed 11 kg and head circumference was 48.5 cm. He had pallor but no cyanosis, jaundice, clubbing, edema, skin rash or lymphadenopathy. The pulse rate was 106/min, respiratory rate 34/min and BP 110/72 mm of Hg. His temperature recorded at admission was 38.5°C. Chest and cardiovascular examinations were essentially normal. Abdominal examination did not reveal any organomegaly. The child was comatose with no eye opening or vocalisation even to deep pain and he was having decorticate posturing on deep pain. Pupils were bilaterally 4 mm in size and normally reacting. Fundus showed doubtful papilledema. There was no cranial nerve palsy; limbs were hypotonic; and deep tendon reflexes were normal with upgoing plantar response. He was not having any meningeal signs. On investigation, he had a Hb of 11.2 g/dl, total leucocyte count of 32000/mm^3 with 72% polymorphs and 2% eosinophils. Platelets were adequate on peripheral smear examination. Serum electrolytes were normal. Blood urea was 70 mg/dl and serum creatinine was 2 mg/dl. On intravenous drip he had a blood sugar of 210 mg/dl. Initial arterial blood gas study revealed normoxia with CO_2 washout followed by development of hypoxia and CO_2 retention preterminally. Chest X-ray, reportedly was normal. Electrocardiography was normal initially with a QoTC interval of 0.44 sec. Preterminally there was wide QRS ventricular

tachycardia. Laryngeal swab did not show any abnormality on Gram's staining, Albert's staining and culture. There was no malarial parasite on peripheral blood film examination. Blood culture was sterile. Lumbar CSF was mildly xanthochromic with RBC 120/mm^3, WBC 5/mm^3 (1 polymorph, 4 lymphocytes). CSF protein and sugar were normal. Detailed microbiological test reports were not available. Before arrival at PGIMER, Chandigarh the child was put on dexamethasone and antitubercular drugs from day 2 of illness. Following lumbar puncture the patient worsened with deepening of coma and frequent decerebration;, so he was referred to PGIMER. Here he was observed to have initial left focal clonic seizure for 5 minutes, thereafter right focal clonic serizure about an hour later. He developed pupillary asymmetry following second seizure with a rise in BP to 140/100 mm of Hg. Mannitol infusion and fluid restriction brought the pupils back to equal and reacting but the sensorium remained same. The child had left sided chest crepitations. The patient was put on controlled hyperventilation. About 6 hours prior to demise he developed hypotension and hypoxia. Ventilator settings were readjusted and dopamine and dobutamine infusion were started. The CVP remained between 8 to 12 cm of saline. Thereafter he was seen to have bilateral chest crepitations with profuse frothy pinkish endotracheal tube aspirates. About 2 hours prior to demise, the heart rate dropped to 58/min from 160/min for about 5 minutes, with sudden spontaneous increase to 178/min. Then he suddenly developed cardiac asystole. Cardiopulmonary resuscitation including injection adrenaline revived the child but he developed wide QRS ventricular tachycardia and very soon repeat arrest from which he could not be resuscitated. The treating unit's final diagnosis was encephalitis (?viral) with raised intracranial pressure and right sided pneumonia.

Clinical Discussion

To analyze the case clinically, firstly each of the major clinical features will be dissected further. Presence of seizure suggests cortical involvement. As the observed seizures were initially left focal and later right focal, the disease affected both hemispheres in the form of bilateral focal cortical involvement. He also had fever without meningeal signs. So the child probably had focal encephalitis with rapid progression to other side or else inflammatory granuloma or abscess.

Altered sensorium suggests either diffuse bilateral hemispheric disease or brain stem reticular activating system (RAS) affection. This RAS involvement may be primarily due to lesion in brain stem only or secondary to transtentorial herniation because of hemispheric disease.

Abnormal posturing suggested decortication initially, worsened to decerebration following lumbar puncture outside. This may suggest a lesion progressing from above midbrain level downwards even below transcollicular level. Asymmetric pupil and hypertension were seen at an early phase which may indicate early transtentorial herniation secondary to raised intracranial pressure.

Sudden bradycardia to 58/min from 160/min and again reverting to 170/min without any other intervention may suggest autonomic seizure, the site may be temporal lobe or this may rarely be found with raised intracranial pressure.

The triad of fever, seizure and coma comprise the entity described by some as acute febrile encephalopathy. I personally prefer using the terminology of acute febrile encephalopathy or encephalitis

(AFEE) as clinically it may be due to inflammatory cause, *i.e.,* encephalitis or noninflammatory cause, *i.e.,* encephalopathy. The specific diagnosis underlying this clinical condition is based again upon a tripod comprising of clinical, epidemiological and investigative tools. Investigative limb is again based on a tripod of imaging (CT/MRI Radionuclide scan), electrophysiology (EEG) and CSF study.

If we approach through clinical angle, this child had early onset transtentorial herniation which may be due to encephalopathy, infective meningitis/meningoencephalitis or large intracranial hemorrhage. Early and prominent seizure as the clinical feature is odd for ence-phalopathy. Among the common causes of encephalopathy at this age, important ones are Reye's syndrome; enteric, hepatic, uremic and lead encephalopathyies; and diabetic ketoacidosis; and aflatoxin, valproate and salicylate poisoning. Because of lack of proper setting I will not consider causes of encephalopathy other than Reye's syndrome. For Reye's syndrome also odd points are absence of preceding viral illness, salicylate intake, hepatomegaly and hypoglycemia (can be found in upto 40%).

Infective causes can be divided into bacterial, like pyogenic meningitis and brain abscess which are odd considering the CSF report and spread of lesion to other side. Other causes may be protozoal like cerebral malaria or primary amebic meningoencephalitis or viral encephalitis. Cerebral malaria does not stand high on the list as fever was low grade in this case; clinical raised intracranial pressure is rare; early and prominent seizure are uncommon and above all peripheral blood film for malarial parasite was negative. In absence of history of swimming and because of rarity and absence of meningeal signs, primary amebic meningoencephalitis is hot being considered.

The common causes of acute viral encephalitis are summarized in *Table I.* Among the causes in the Table it is unlikely that this child had enteroviral encephalitis or other encephalitides characterized by primary non-CNS involvement. So arboviral and herpes simplex virus encephalitis will be considered. Among arboviral diseases, Japanese B is the one commonly encountered in India.

Clinically our patient was having either focal encephalitis or diffuse encephalitis with focal cortical involvement. What can be the site of focality? Clinically fluctuating heart rate as mentioned earlier may indicate autonomic seizure-which may have temporal lobe origin. If a focal cortical lesion produces early transtentorial herniation, the commonest site will be temporal lobe rather than far off frontal or parietal

TABLE 1-*Common Causes of Acute Viral Encephalitis*

*	Arbovirus	- Japanese B Kyasunur Forest Disease Dengue
*	Enterovirus	- Polio Coxsackie Echo
*	Herpes	- *Herpes simplex*
*	Associated with primary involvement of other system	- Measles - Mumps - Rubella - Varicella
*	Associated with respiratory illness	- Adeno - Influenza - Reo
*	Indeterminate (etiology not found)	

cortex. This is because of early pressure cone formation across the tentorial hiatus if lesion involves structures adjacent or nearer to tentorial hiatus. When these two features are considered, the most probable localization remains in the temporal lobe. Clinically, the commonest cause of encephalitis involving temporal cortex is *Herpes simplex* virus. The fulminant course and rapid spread to other hemisphere may support the diagnosis.

When approached from epidemiological angle, the commonest cause of sporadic encephalitis all over world, irrespective of age is *Herpes simplex* virus (l). Secondly, the commonest cause of focal encephalitis is herpes simplex virus. Absence of any prodrome is supportive for this diagnosis. In India, though Japanese B encephalitis is the commonest encephalitis, this is strictly endemic, localized in certain pockets (2,3). Western UP does not fall in the endemic area. So the child is less likely to be suffering from Japanese B encephalitis virus infection. Such fulminant course is also uncommon in Japanese B encephalitis.

The only investigative tool at our disposal is the incomplete CSF study which shows xanthochromia with RBC 120/mm^3. The presence of xanthochromia with RBCs rules out a traumatic CSF and suggests hem-orrhagic CSF. Among the causes of hem-orrhagic CSF, subarachnoid hemorrhage, Herpes simplex virus (HSV) encephalitis and acute hemorrhagic leukoencephalitis are common. In absence of proper setting, subarachnoid hemorrhage is not considered. There are many points against acute hemorrhagic leukoencephalitis. Almost 100% of such patients have preceding viral respiratory illness or varicella. Meningeal signs are common. Sudden onset coma is less likely to be seen in such cases. Presence of xanthochromia with RBC in CSF without pleocytosis is another relative odd point. Thus *Herpes simplex* virus encephalitis remains the diagnosis from the investigative angle.

When seen from all the three angles of clinical, epidemiological and investigative aspects, the common diagnosis is *Herpes simplex* virus encephalitis.

This child also had right infra-mammary crepitations with polymorphonuclear leukocytosis suggesting pneumonia. Preterminally he developed bilateral chest crepitations and pink frothy profuse endotracheal tube aspirate suggesting neurogenic pulmonary edema. Preterminally this patient had hypoxia [may be due to adult respiratory distress syndrome (ARDS)], acidosis, hypotension and cardiac arrest. After revival with cardiopulmonary resuscitation including adrenaline he was found to have wide QRS ventricular tachycardia, which may be a preterminal event. So the final clinical diagnosis is: *(a) Herpes simplex* virus encephalitis with transtentorial herniation; and *(b)?* Right sided pneumonia, neurogenic pulmonary edema, ?ARDS. Cause of death: transtentorial herniation and neurogenic shock.

Comments From the Audience Before Pathology Protocol

Dr. P.C. Reddy: As the senior resident of the admitting unit, I agree with the diagnostic possibility. This child also had right sided pneumonia and ultimate rapid deterioration may be explained by development of ARDS.

Prof. R.f. Dash: Why the primary treating unit did not consider *Herpes simplex* virus during life?

Dr. P.C. Reddy: We thought of some acute encephalitis. However, detailed investigations like EEG or CT head could not be done because of short stay in the hospital. The major thrust in the initial part was in

controlling raised intracranial pressure and refractory shock later.

Dr. S. Prabhakar: I agree primarily with the diagnosis of *Herpes simplex* encephalitis. The heart rate fluctuations may very well be due to temporal lobe seizure. However, considering hemorrhagic CSF with altered sensorium, I will keep an alternative diagnosis of subarachnoid hemorrhage, may be due to vein of Galen aneurysm rupture.

Prof. R.J. Dash: How frequent is *Herpes simplex* virus encephalitis in children:

Dr. S. Prabhakar: No age group is immune to Herpes simplex encephalitis.

Prof. R.J. Dash: Now, I request Dr. Ritmabha to present the pathology protocol.

Pathology Discussion (PM 15174)

A complete autopsy was performed through midline thoraco-abdominal and biparietal incision. The peritoneal cavity contained one liter of hemorrhagic fluid. This child had systemic vasculitis involving various visceral vessels. Infarction and hemorrhages were present in various organs. However, visceral vessels were not available for gross examination.

The kidneys weighed 150 g and showed irregular pale grey infarcts in the cortex along with medullary congestion *(Fig. 1)*. On microscopic examination, the infarcts were infiltrated by neutrophils at the border and were of less than 6 hours of duration *(Fig. 2)*. Intrarenal arteries showed necrotizing vasculitis in varying stages of healing. There was fibrinoid necrosis along with infiltration by acute inflammatory cells involving all the layers of arteries (acute phase) *(Fig. 3)*. Proliferating fibroblasts and lymphomononuclear infiltrate had replaced this fibrinoid material in some of the arteries (healing phase)(Fjg. *4)*.

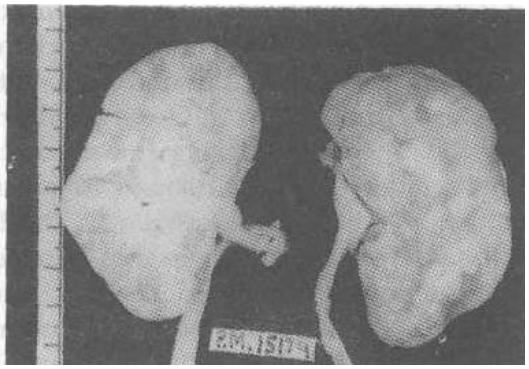

Fig. 1 *Irregular geographic infarcts with sharp borders seen both on the outer (right) and cut (left) surface*

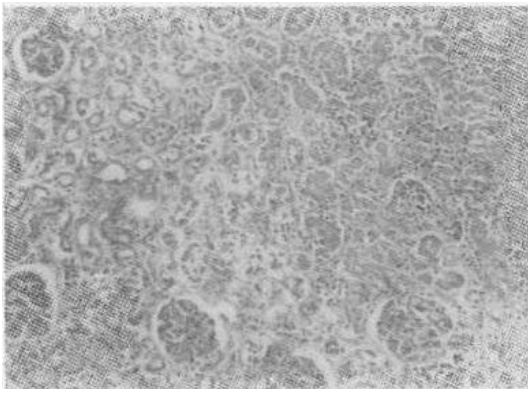

Fig. 2. Photomicrograph of a recent renal infarct with polymorphonuclear leucocyte infiltration (H&E x 150)

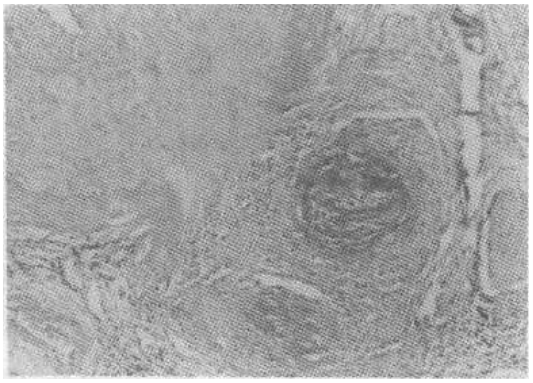

Fig. 3. Aneurysmal bulge of the artery shows fibrinoid necrosis of the wall (acute vasculitis) (H&E x 550)

Elastic von Grieson stain revealed break in internal elastic lamina, through which vessel wall was bulging out and represented aneurysmal dilatation in the larger arteries *(Fig. 5)*. There was varying amount of intimal proliferation and thrombi with organization in many of these vessels (healed phase). These vasculitic changes were segmental sparing varying lengths of the arteries. Rest of the kidneys showed normal glomeruli, mild interstitial inflammation and acute tubular necrosis.

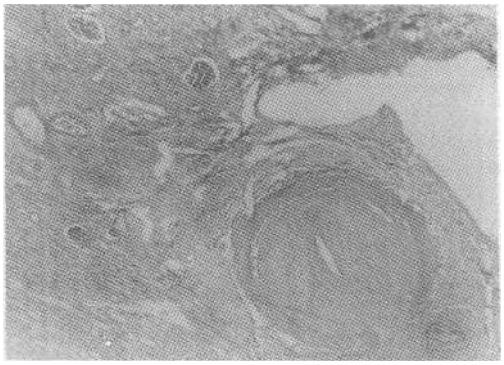

Fig. 4 *Interlobar artery showing ancurysmal dilatation with focal break in internal elastic lamina and marked intimal proliferation (H&E x 150)*

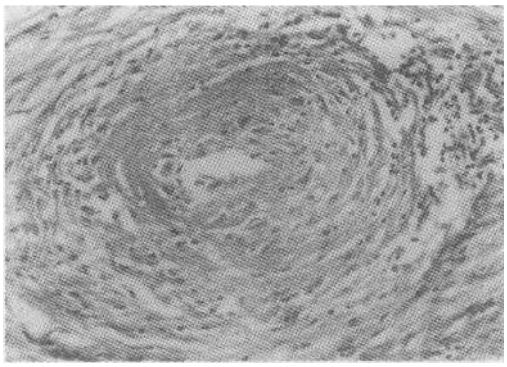

Fig. 5. Lymphomononuclear infiltrate and fibroblast proliferation replacing fibrinoid necrosis of a small artery (healing vasculitis) (H&E x 550)

The liver weighed 560 g and showed pale infarcts of varying sizes and shapes involving both the lobes *(Fig. 6)*. These infarcts were of varying extent depending upon ischemic damage to that particular segment. Confluent hepatic necrosis along with that of portal tracts were seen in severely damaged areas, whereas periportal hepatocytes were preserved in the lesser affected areas *(Fig.*7). Portoportal bridging fibrosis was present in preserved areas. Hepatic arteries and its branches in portal tracts showed necrotizing vasculitis as seen in the kidneys. Portal veins were normal.

The heart was enlarged, weighing 180 g. There was left ventricular hypertrophy. Serial slicing of coronary arteries showed luminal narrowing (25-50%). Aneurysmal bulging of vessel wall through broken elastic lamina and thrombus was seen on microscopic examination of these arteries *(Fig.*8*)*. There were healed myocardial infarcts. Valves were normal.

Stomach showed two 0.8 cm ulcers which were confirmed to be hemorrhagic acute ulceration of the mucosa. Submucosal and serosal arteries showed necrotizing and healed vasculitis *(Fig.*9*)*. Similar vasculitic changes were present in the serosal of small intestine and had resulted in the hemorrhagic ascites. Small intestine showed patches of mucosal hemorrhages. Mesenteric artery showed healed vasculitis with a thrombus.

Similar vasculitis was also present in adrenals *(Fig.*10*),* pancreas, testis and spleen. Left adrenal showed medullary hemorrhage. The rest of the organs were normal.

The lungs weighed 300 g and were heavy because of presence of pulmonary edema. Bronchial arteries showed vasculitic changes; however, pulmonary arteries were normal.

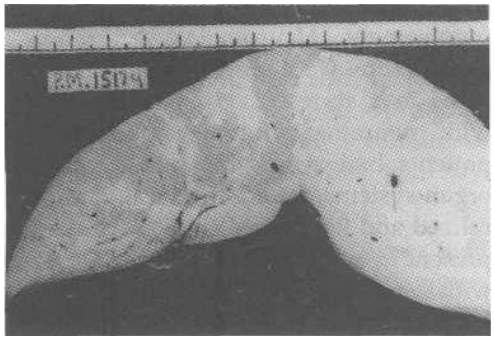

Fig. 6 *Irregular pale infarcts of liver*

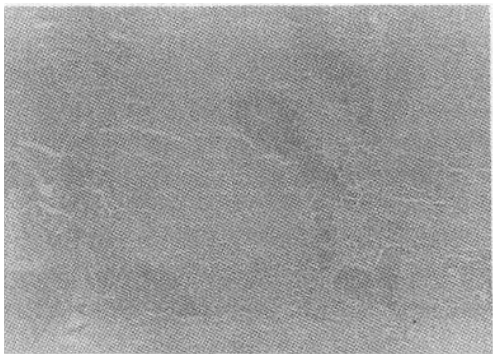

Fig. 7. Photomicrograph shows confluent hepatic necrosis with preservation of a small rim of hepatocytes (H&E x 150)

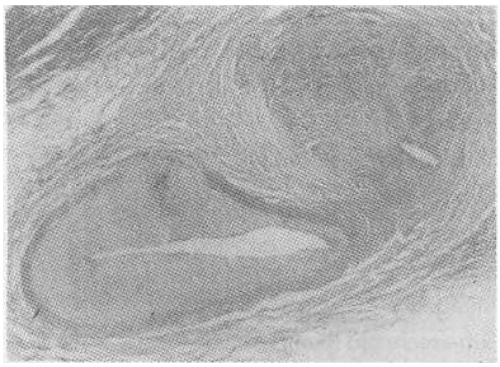

Fig. 8. Healing phase vasculitis and aneurismal bulge through broken internal elastic lamina in coronary arteries (H&E x 550).

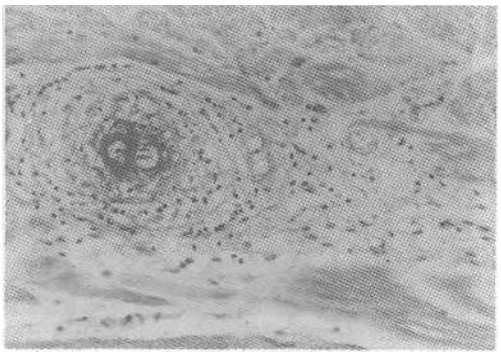

Fig. 9. Necrotizing vasculitis in a small artery in muscularis propria of stomach (H&E x 550)

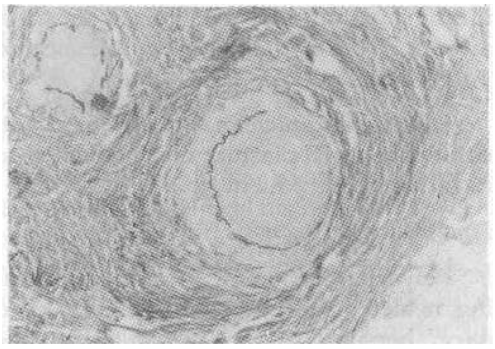

Fig. 10. Proliferated intima with break in internal elastic lamina of adrenal artery (Elastic von Grieson x 550)

The brain weighed 1380 g and showed diffuse edema with mild uncal herniation. On slicing, petechial hemorrhages at grey white junction were present in fronto-parietal and temporal lobes. Small infarcts were seen grossly *(Fig.*11*)*. Microscopically, early infarcts (24 h), hemorrhages, fibrinoid necrosis of arterioles were present along with edema. Meningeal vessels were normal. There was no encephalitis.

Summarizing, this patient had systemic necrotizing vasciilitis in varying stages of healing, involving circumferentially or

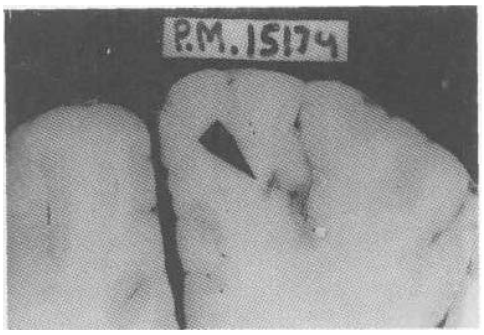

Fig. 11. *Petechial hemorrhages at grey white function with recent infarcts in frontal cortex.*

focally medium and small sized muscular arteries. These features are classical of polyarteritis nodosa.

HBsAg, antinuclear antibody, and anti-neutrophil cytoplasmic antibodies were negative in this patient.

Final Autopsy Diagnosis

Polyarteritis nodosa (necrotizing, healing, healed stages) involving cerebral, coronary, hepatic, renal, splenic, pancreatic, mesenteric, gastric, adrenal and testicular arteries.

Infarction

Early: Brain, liver, kidneys.

Healed: Heart.

Hemorrhage: Adrenal, intestine, retroperitoneum

Ulceration: Stomach

Pulmonary edema

Discussion

Polyarteritis nodosa (PAN) is uncommon in children. The central nervous system is less frequently involved and seizures occur in only 7% of these cases(4,5). Neurological symptoms occur late in the course of this disease and patients usually have manifestation of other organ systems involvement. Neurological symptoms at presentation were present only in 5% of the cases in a series of 114 patients of PAN with neurological symptoms. All these patients had symptoms relating to other organ systems, unlike this case. Both generalized and focal tonic-clonic seizures were present in only 3% of the total 11% patients with seizures in this series. Cerebral infarcts, hemorrhages or both were present in 13% patients. Our case is unusual as he had central nervous system symptoms at presentation with no symptoms relating to other organs. Childhood PAN is similar to adult PAN(6). In this study of 7 children with PAN, coronary arteritis was present in all, along with myocardial fibrosis in five of them. Central nervous system involvement was the cause of death in two out of 7 children. The reported neurological manifestations of PAN are summarized in *Table II.*

Infarction of liver occurs in 15% patients of PAN and when infarctions of liver were reviewed (total 54), 42% of them had polyarteritis nodosa(7).

TABLE II--*Neurological Manifestations of PAN*

Manifestation	Per cent
Peripheral neuropathy	67
Headache	30
Retinopathy	29
Diffuse encephalopathy	16
Focal stroke	14
Cranial nerves	9
Seizure	7
Myopathy	6
Sub-arachnoid hemmorhage	< 1

Table is based on an earlier review of total 215 patients which includes both adults and children(4).

Post Pathological Discussion

Dr. R..J. Dash: Dr. Ritambhra, this was really an interesting case that you have shown today. From the series of cases, mentioned by you, were they diagnosed during life or at autopsy? Could you elaborate on the clinical details of the data you have shown?

Dr. Ritambhra: Only two cases were diagnosed during life and five were diagnosed after autopsy. Testicular biopsy and angiography were done to establish the diagnosis during life.

Out of seven cases that I have shown none had such a short duration of illness. Usually they had symptoms- for 5 weeks-15 months. Only one case of infantile PAN had symptoms for a few hours only. These patients had fever, leukocytosis and were clinically diagnosed to have infection. Other investigations which clinched the diagnosis were undertaken when they failed to respond to conventional treatment for infections.

Dr. R..J. Dash: Are there any clues thinking retrospectively which could have helped in diagnosis?

Dr. S. Verma: With this short illness and fever, infection comes first to anybody's mind. However, on retrospective analysis he had deranged renal function tests (S, creatinine-2 mg/dl; blood urea-70 mg/dl). In the absence of routine examination of urine (which is likely to have shown RBCs as there are renal infarcts in PAN), this mild rise in urea and creatinine can easily be passed off as pre-renal azotemia.

Dr. Lata Kumar: We have reported series of PAN in children. None of the patients had such a short history. Patients who had neurological involvement had severe degree of hypertension. Rarely an acute presentation like acute abdomen is mentioned. If the history has been recorded correctly, then this is most unusual presentation and it is impossible to suspect PAN with the clinical protocol presented. Most of the children in our series are alive and doing well on follow up. The ones who died, did not show involvement of coronaries, which is usually seen in infantile type of PAN which can be referred to as Kawasaki Disease.

Dr. R..J. Dash: Focal seizures, were present in 3 out of 7 children which could be a very high percentage. Do you think this can be taken as clue?

Dr. L. Kumar. Seven-eight of our patients had seizures, focal/generalized. AH of them had multiple organ involvement and diagnoses were possible except in one who had only hypertension. This child had a normal BP recording. So truly speaking there was no clue in this case.

Dr. Surjit Singh: There seems to be something wrong in clinical history. In the world literature, history less than 1 week is unheard of. Hypertension is present in 80% of children with PAN. So on admission BP of 110/72mm of Hg is an odd point for the clinical diagnosis of PAN.

Dr. R.J. Dash: Do you think this blood-pressure recording is dependable with those blood-pressure cuffs used in a thin built child?

Dr. Surjit Singh: It's difficult to say, but we use pediatric size cuffs.

Dr. A. Grover: It is common in hypertensive adults that blood pressure falls and anti-hypertensive dose requirement decreases when myocardial infarction occurs. Retrospectively it can be speculated that he had painless myocardial infarct as a result of mononeuritis and autonomic system involvement because of vasculitis. This could be responsible for normalization of blood pressure.

Dr. Ritambhra: This patient had old myocardial infarct. No fresh infarct was seen. So that possibility is less likely.

Dr. S. Prabhakar: In CPCs, everything odd is possible. Going by this protocol, it is more of a pathological data presentation rather than a clinico-pathological correlation or dis-correlation. I think we have missed something in the history. With such short history of fever, leukocytosis, I think there is no way one can go away from infections. In PAN, the neurological symptoms are always accompanied by renal involvement. The blood pressure recorded was 110/72 mm Hg. Only once high blood pressure was recorded. I do not think we can consider hypertension in this patient.

Dr. A.K. Banerjee: With our limited resources and diagnostic capability, *Herpes simplex* encephalitis is the commonest cause of sporadic encephalitis. This is based on cases in which diagnosis is established. There are many more cases of encephalitis in which we have no idea about the etiplogical agent except a few cases of rabies and one case of Japanese encephalitis.

Involvement of brain by PAN is very rare. Cranial and peripheral neuritis are more common neurological manifestations. In this case also, unlike in other organs, no vasculitis was seen in cerebral arteries. Fibrinoid necrosis of capillaries with infarction could be embolic. There is no direct relation between CNS disease and vasculitis as no vaculitis of similar sized vessels was seen in brain. But it has to be lumped into cerebral lesions of PAN as we cannot get away from the fact that he has PAN with neurological symptoms and small hemorrhagic cerebral lesions.

Editor's Comment

Similar vasculitis of medium sized intracranial vessels were also found focally on careful examination of these vessels after CPC.

Dr. R.J. Dash: Can you tell us about frequency of *Herpes simplex* virus (HSV) in children?

Dr. A.K. Banerji: No. I can't tell frequency in adults or children. It occurs in all ages. Other than rabies this is the most frequently diagnosed encephalitis out of various viruses we can diagnose at autopsy.

Dr. S. Singhi: I have been working in the Intensive Care Unit. We see 2-5 cases of HSV per year. This is the most often proven encephalitis but we see many otherwise. This blood pressure of 110/72 is not normal for 6 years of age. Blood pressure of 110 is 90th percentile but it could be due to raised intracranial tension. We cannot think of vasculitis especially PAN, with this high blood pressure, also. If at all, SLE can be thought of because of seizures and hypertension. During subsequent course, one could think of hypertensive encephalopathy with hypertension (140/100 mm Hg) and cardiac arrhythmia. Such high blood pressure is not seen due to raised intracranial tension.

Dr. D. Ghosh: I could not find any series in Indian literature on HSV in children. In world literature HSV in children is well reported. Series on HSV in adult have been reported from National Institute of Mental Health and Neurological Sciences, Bangalore.

I have two questions to the pathologist: *(i)* How can we explain so much of edema, herniation of brain and RBCs in CSF based on the diagnosis of PAN? and *(ii)* is pulmonary edema cardiogenic or neurogenic?

Dr. Ritambhra: Early infarcts at grey white junction were seen in frontal, temporal, parietal lobes along with accompanying edema. Pulmonary edema can be due to

acute left ventricular failure as a result of arrhythmia. Neurogenic edema cannot be excluded.

Dr. D. Ghosh: These infarcts are fresh (12 h) and edema usually occurs in 3-4 days. So how can you explain so much edema so early? How can RBCs come in CSF?

Dr. A.K. Banerjee: The fact is that edema is present which is resulting in herniation. RBCs in CSF can be explained due to hemorrhagic lesions being present at the depth of sulci from where RBCs can find way to CSF.

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