Case Report

Hypertensive encephalopathy after successful treatment of eclampsia

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ABSTRACT

The association between acute rise of blood pressure and encephalopathy with early recognition, and therapy reversibility has been reported. We reported a case of a young lady in postnatal period, presented with acute rise of blood pressure, encephalopathy, quadriparesis, and apraxia. Magnetic resonance imaging of the brain showed hyperintense lesions in occipital, parietal and right temporal areas. Cerebral angio showed multiple segmental vasoconstriction and narrowing of intracerebral vessels. Immediate control of blood pressure enhanced recovery but it is incomplete.

Keywords: Hypertension, encephalopathy, posterior leucoencephalopathy, pregnancy.

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Hypertensive encephalopathy is frequently caused by high blood pressure associated with nephritis and toxacemia of pregnancy (Wilson 1963). A few cases have been reported in association with phaechromocytoma (Millez 1963, Graham 1951), and lead poisoning. Jellinech et al 1964 reported a case of renal artery occlusion, Meyer 1961 also mentioned porphyria as a rare cause.[1] Hypertensive encephalopathy is a rare condition and the prognosis is usually good. Similar conditions may be encountered in toxemia of pregnancy, in immunosuppressive drugs, cyclosporin toxicity, or use of erythropoietin.[2] These conditions can occur with only minimal or no elevation in blood pressure or with normal cyclosporin levels. We described one case with high blood pressure, seizure, visual changes and quadriparesis. Magnetic resonance imaging and cerebral angiography were compatible with the diagnosis of hypertensive encephalopathy.

Case report. A 22-year-old woman, who was diagnosed at her 4th month of pregnancy to have high blood pressure. She was started on antihypertensive medication (not known). At 32 weeks of gestational age she stopped her treatment. Two weeks later she experienced severe headache, generalized odema, seizure associated with very high blood pressure. She was admitted to a local hospital, where she delivered by caesarian section and treated with phenytoin and antihypertensive medications, with full recovery except, the blood pressure remained high.

On the 4th day post partum she became agitated and aggressive. Her clinical examination showed dysarthria, left upper motor neuron (UMN) facial palsy, and spastic quadriparesis more on the left side (2/5). She was intubated and received atenolol 100 mg/od, lazix 20 mg/bid, nifedipine CR 20 mg/bid, alpha methyl dopa 250ms/tds, dexamethasone 4 mg/IV/8 hrly and aspirin 100 mg/od. Computerized tomography scan of the head showed diffuse odema (in the report). Then four days later she was extubated. On the 3rd week post partum, she was transferred to Riyadh Armed Forces Hospital. Clinical examination on arrival, she was afebrile, blood pressure 139/83. Conscious, oriented, with visual activity 20/200 bilaterally, oculopraxia normal visual fields and normal fundi. She had dysarthria, left UMN facial nerve palsy, spastic quadreparesis, left (2/5), right (4/5) and motorapraxia. Her blood

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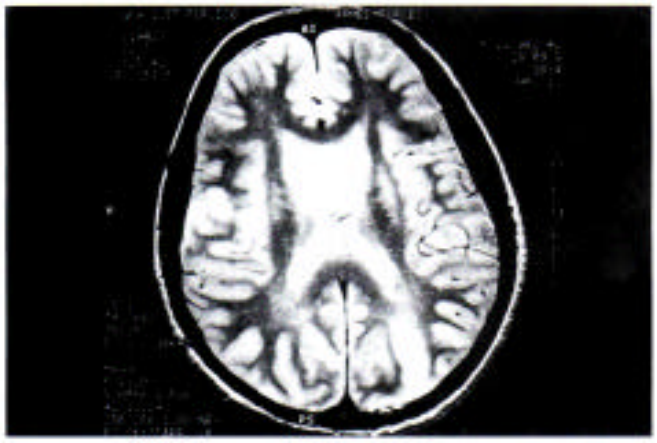

Figure 1a - Axial T2 MR (TR 3000, TE 45) at the level of the lateral ventricles multifocal cortically located hyperintensities in the right temporal and left posterior parietal cortex.

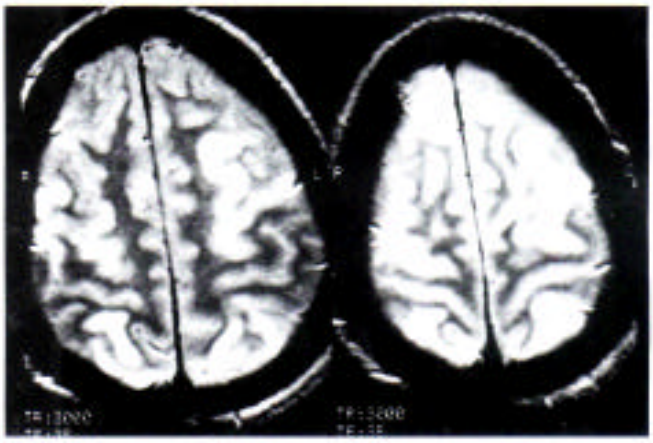

Figure 1b - Axial T2 MR (TR 3000, TE 45) at the level of the centrum semiovali showed bilateral multifocal cortical hyperintensities in the posterior frontal, the occipital and parietal corticies. Note the sparing of the underlying white matter.

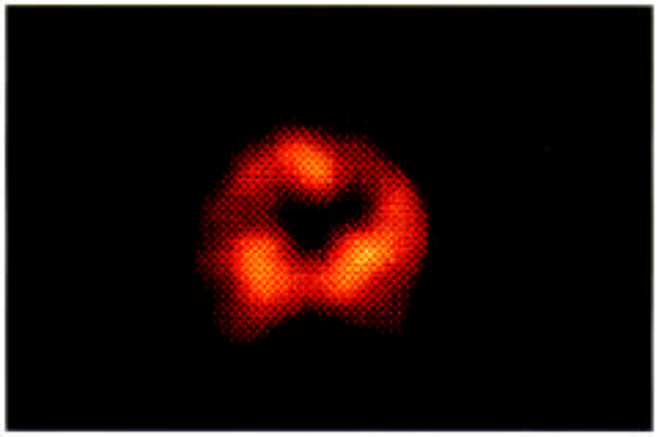

Figure 2a - Brain SPECT scan anterior coronal view shows hypoperfusion in the right frontal cortex.

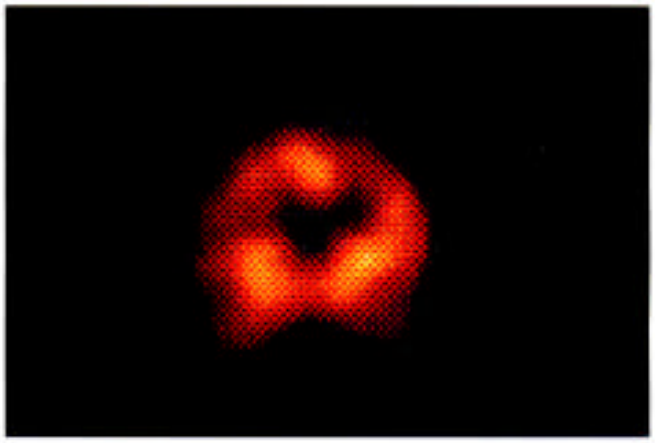

Figure 2b - Brain SPECT scan posterior coronal view shows multifocal cortical hypoperfusion in the left and right parietal cortex.

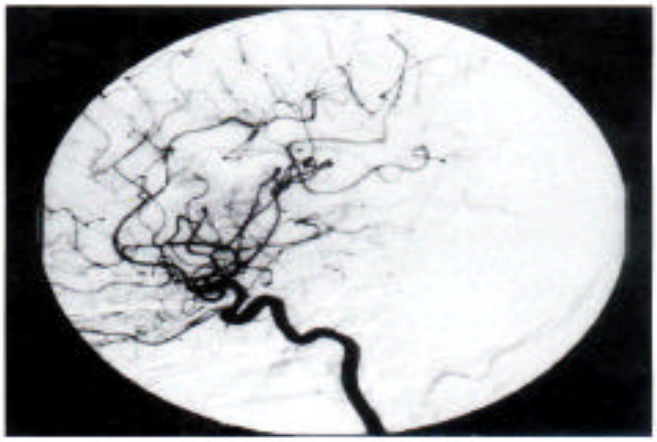

Figure 3 - Cerebral angiography demonstrating segmental narrowing.

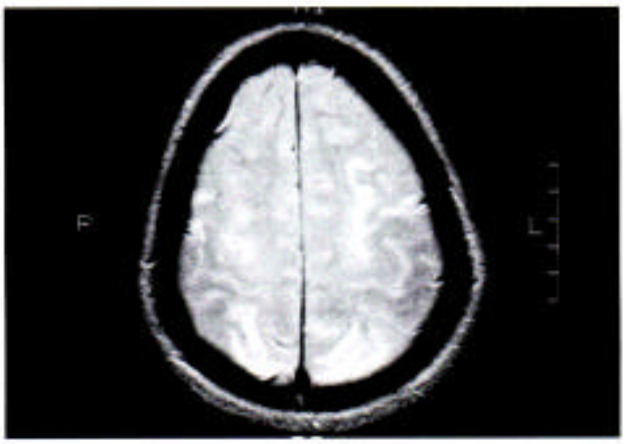

Figure 4a - Follow-up MR after 6 weeks T2 W1 (TR 3000, TE 45) showed persistance of the cortical hyperintensities in the right partietal, left posterior frontal, the occipital and left parietal corticies.

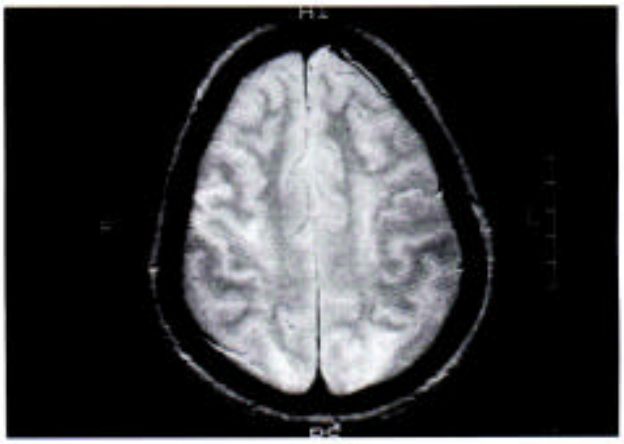

Figure 4b - Three month follow-up MR T2 W1 (TR 3000, TE 45) the hyperintensities affecting the cortex are essentially unchanged.

counts, renal function, hepatic profile thyroid function and coagulation profiles were all within normal. Her cholesterol was 8.04 mmol/L, LDL 5.03 mmol/L and ESR 30. Autoimmune screen, anti-neutrophil cytoplasmic antibodies, antiphospholipids, anti cardiolipin, complements, protein C, protein S, corticol and vinyl mandelic acid were normal. Brucella and hepatic screens were negative. Urine analysis showed no proteinuria. Ultrasound, electrocardiogram, chest x-ray, and echo were all normal. Ultrasound abdomen and doppler of renal arteries were normal. Normal cerebrospinal fluid analysis. Transcranial doppler showed increased velocity in left anterior cerebral artery, which indicates vasospasm or hyperemia. Electroencephalogram showed poorly developed and organized background, slightly depressed on the right with pleomorphic delta waves (1-3/sec) on the right temporo-parietal areas. Evoked Potential: The electroretinogram is normal but the severely abnormal flash visual evoked potential (FVEP) indicates bilateral involvement of the visual pathway. Normal brainstem auditoy evoked responses and abnormal somatosensory evoked potentials suggest a lesion affecting the right thalamoparietal projections.

Magnetic resonance imaging (MRI) of the brain showed bilateral multifocal cortical lesions involving the occipital and parietal lobes as well as the right temporal lobe (Fig. 1a,1b). Single photon electron computer tomography scan was done later it showed decreased perfusion at the areas of high signal intensities in the occipital, parietal and right temporal lobes (Fig. 2a, b). Her cerebral angiography showed multiple segmental vasoconstriction and narrowing of intracerebral vessels (Fig. 3). During her admission, the blood pressure was controlled on nifedipine 20 mg/bd and atenotol 100 mg/d in addition to enalopril 10 mg/bd. After the 4th week post partum she showed great improvement, but remained with minimal left-sided hemiparesis (4/5) and global apraxia. Follow-up MRI done after six weeks, showed no significant changes noted from the previous ones. The patient was transferred to rehabilitation unit, then seen 3 months later in the out-patient clinic. She is still having some apraxia but no paresis. Repeated MRI done 3 months later showed no changes from previous ones with the suspicion that the high signals may represent multiple cortical infarcts (Fig. 4a,b).

Discussion. Hypertensive encephalopathy (HTE) is a rare disease nowadays. The full crisis of hypertensive encephalopathy is often heralded by weakness, apathy, headache, drowsiness and vomiting, concomitant with a severe rise in blood pressure. Convulsions are common, either generalized or focal and usually recurrent.[3,4] Loss of vision is a common feature, while papilloedema and hypertensive changes in the fundi are frequently lacking.[1] Drowsiness may lead to coma and various focal cerebral disturbances. Stiffness of the neck is frequent (Millcz 1943).[3]

In our patient the most likely cause is the sudden rise in blood pressure precipitated by toxemia of pregnancy.[5] Subarachnoid hemorrhage, intracerebral hemorrhage, cerebral infarction, cerebral abscess or tumour were ruled out by computerized tomography (CT) of the brain.[1,3,5] Isolated vasculitis cannot be ruled out by CT brain without biopsy, but with this clinical course of the disease it is unlikely.[4] The etiology of HTE is due to sudden severe rise in blood pressure, but it can occur with moderate hypertension.[3,7] Two theories have been proposed to account for the clinical and radiological abnormalities. The first hypophysis postulates that HTE is due to spasm of cerebral arterioles (i.e. over regulation), resulting in ischaemia and cytotoxic edema involving mainly the borderzone arterial regions. Another theory suggests that HTE results

from a breakthrough of autoregulation with passive overdistension of cerebral arterioles. Results in interstitial extravasation of protein and fluids, producing focal vasogenic (hydrostatic) edema in vascular distribution of the involved vessel. Also disruption of blood brain brain (BBB) with resultant edema had been found.[1,7-9]

The susceptibility of the posterior circulation is well known but poorly understood.[8,9,10] The only explanation is due to regional heterogenecity of the sympathetic vascular innervation. The neuroimaging supports the 2nd hypotheses. Computerized tomography scans demonstrate symmetrical hypodensitis in white matter. Often superimposed on diffuse edema.[7] The MRI showed high signal intensity lesions in cortical and subcortical regions with consistent involvement of parietooccipital lobes. Best seen in T2 weighted images. Symmetrical confluent lesions and patchy enhancement in subcortical white matter were identified but asymmetry can be encountered. Also, multiple areas of symmetric edema can be seen in basal ganglia, cerebellum and brain stem. in severe cases infarction with or without hemorrhages can be seen. All these changes are reversible.[7,8,11] Magnetic resonance imaging of the brain is more sensitive than CT and better in defining anatomy.[7] Single photon electron computer tomography (SPECT) showed increased perfusion in the areas of abnormal signal intensities on MRI, especially during crisis.[9] When blood pressure normalizes, the images may show mildly abnormal or normal perfusion.[9] Cerebral angiograpy shows segmental narrowing.[9] However, the term benign acute cerebral angiopathy is reserved for postnatal angiopath. (Michel et al 1985, Call et al 1988, Garner et al 1990). All these changes have been seen in our patient and remained persistent even after three months follow-up. Transient, severe, uncontrolled hypertension may be antecedent to the development of angiopathy, but not chronic hypertension (Bougouss lausky 1989, Chester et al 1978).[3,6] Sometimes acute and transient high blood pressure may be missed. All investigations done in our patient ruled out evidence of end organ damage as complication of long standing hypertension.

The EEG in Jellinek et al 1964 cases showed in acute stage bilaterally synchronous often-rhythmic occipital sharp and slow activity. The alpha activity actually was lost or impaired during the period of blindness.[3] Cerebrospinal fluid pressure is usually raised but not constant. Also protein content may be high or normal. Cell count is normal but neutrophilic[5,12] pleocytosis may be detected in absence of infection.[3,12] Both HTE and eclampsia share similar pathophysiologic mechanism and now appear to be forms of acute process known as reversible posterior leucoencephalopathy syndrome.

Postnatal eclampsia is a rare complication of hypertensive pregnancies and usually develops within 48 hours of delivery, which is not the case in in our patient. However, there are some reports in which eclampsia can occur up to 16 days, but this is exceedingly uncommon and should be considered with caution especially in hypertensive patient. The treatment is essentially based on a prompt lowering of mean arterial pressure.[3,7] The main drugs available are: sodium nitroprusside which is the drug of choice, alternatives labetalol, diazoxide and nifedipine.[13] Avoid vasodilators such as papaverine and hypercapnia. Agents such as methyldopa and clonidine have the effect of depressing central nervous system (CNS), which may be confused with CNS deterioration and it should be avoided.[3,4] The cerebral disorders may disappear very quickly in a matter of minutes or hours and sometimes days to weeks.[3,7] When MRI lesions resolve, the patient became seizure free, without requiring chronic anticonvulsant therapy.[4] Despite impressive lesions on MRI, early recognition and treatment of this disorder carries a favourable prognosis, with complete recovery within 6-8 weeks, but it may be fatal if its unrecognized or treatment is delayed.[7,9] However in few percentage recovery may not be as complete as in our patient.[14]

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