AN INTERESTING CASE OF SEIZURES-POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

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Abstract : We describe here a case of Posterior Reversible Encephalopathy Syndrome (PRES) in a 15 year old girl who presented with new onset seizures, high blood pressure and hematuria. She was diagnosed to be a case of post streptococcal glomerulonephritis. Initial Imaging was suggestive of edema of bilateral parietal and occipital regions which showed complete reversal on controlling blood pressure with medications.

Keyword : Hypertension, Post streptococcal glomerulonephritis, Posterior Reversible Encephalopathy Syndrome (PRES)

AN INTERESTING CASE OF SEIZURES POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME.

CASE REPORT

A 15 year old female was admitted with complaints of headache and vomiting for two days and new onset seizures of generalized tonic clonic type for one day. There was no history of fever or trauma. There was a history of passing red coloured urine for the past one week. There was no history of preceding skin or upper respiratory infection. On examination she was drowsy, afebrile, pale with a pulse rate of 114 per minute and a blood pressure of 160/112 mm Hg. Central nervous system examination revealed bilateral brisk deep tendon reflexes, bilateral extensor plantar and no focal neurological deficit. Fundus examination was suggestive of pappilledema. Both lowerlimbs showed multiple Impetiginous scars. Laboratory investigations revealed normal complete blood count and electrolytes. Urine analysis showed 3-5 pus cells per high power field, plenty of Red Blood Cells and 1+ proteinuria. Urea was 56mg and serum creatinine was 1.43mg. Ultrasonogram of the abdomen showed normal sized kidneys with increased echoes. Urine culture was sterile and 24 hour urine protein was 402 mg. Throat swab culture was sterile. Anti Streptolysin O titre was elevated – 628 U/L (normal < 333U/L). Complement C3 level was reduced- 12 mg (normal 90-180mg/dl).

Computed Tomogram(CT) Imaging of the brain showed bilateral extensive symmetrical hypodensities involving white matter of high parietal and occipital lobes(Fig 1).

Magnetic Resonance Imaging(MRI) of brain revealed non enhancing, non diffusion restricted hyperintensities over right precentral gyrus, right middle temporal gyrus, bilateral parietal cortices and both occipital lobes suggestive of white matter edema(Fig2).
She was started on furosemide and antiepileptics. Blood pressure was gradually reduced. On 10th day of her illness, she was completely normal without further episode of seizures. The patient was discharged on tablet nifedipine 10mg. On follow up after two weeks, the patient’s blood pressure, complement levels and CT Brain were found to be normal.

DISCUSSION:
Posterior Reversible Encephalopathy Syndrome (PRES) is a unique clinico radiological entity first described in 1996 by Hinchey et al. It manifests with headache, vomiting, visual disturbances, confusion and seizures. It is characterised radiologically by bilateral grey and white matter edema in the posterior regions of cerebral hemispheres.

ETIOLOGY: (a) Hypertensive encephalopathy, (b) Infections, (c) Eclampsia, (d) Electrolyte imbalance, (e) Use of immunosuppressants, etc

PATHOGENESIS: In the setting of new onset acute hypertension, cerebral autoregulation fails leading onto vasogenic cerebral edema. The Posterior circulation namely the Vertebro-basilar system has a poor sympathetic innervation and therefore is frequently involved. But PRES can be found in the absence of hypertension in 20-40% of patients. PRES is less common in people who live with chronic hypertension, because they get adapted to the elevation in their blood pressure over time.

DIFFERENTIAL DIAGNOSIS: (a) Posterior cerebral artery territory infarcts, (b) Venous thrombosis, (c) Demyelinating diseases like Acute Demyelinating Encephalo Myelitis (ADEM), (d) Vasculitis, (e) Encephalitis

TREATMENT:
Control of the blood pressure, withdrawal of the offending drug and correction of the underlying cause usually leads to complete neurological recovery within two weeks accompanied by radiological resolution. Few patients with prolonged seizures or hypertension may not recover completely. They may develop persistent neurological deficits due to cerebral infarction.

CONCLUSION
PRES has important implications because of the reversibility of neurological abnormalities on prompt control of blood pressure. The key to diagnosis in PRES is naturally the image, but timely suspicion should be raised by the clinician in order to avoid persistent neurological deficits.

REFERENCES

<table>
<thead>
<tr>
<th>REGIONS INVOLVED</th>
<th>PERCENTAGE</th>
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<tbody>
<tr>
<td>(1) Parieto Occipital</td>
<td>98.7%</td>
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<tr>
<td>(2) Posterior Frontal</td>
<td>78.9%</td>
</tr>
<tr>
<td>(3) Temporal</td>
<td>68.4%</td>
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<tr>
<td>(4) Thalamus</td>
<td>30.3%</td>
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<tr>
<td>(5) Cerebellum</td>
<td>34.2%</td>
</tr>
<tr>
<td>(6) Brainstem</td>
<td>18.4%</td>
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<tr>
<td>(7) Basal Ganglia</td>
<td>11.8%</td>
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Radiographically, PRES manifests on CT as hypodensities of the posterior white and gray matter. Lesions are generally bilateral and parieto-occipital, but may involve temporal or frontal lobes, brainstem, or cerebellum. T2-weighted MRI shows areas of hyperintense signal, but fluid attenuated inversion recovery sequences (FLAIR) may improve detection of cortical and subcortical areas of injury. Diffusion-weighted imaging may help distinguish vasogenic edema (increased apparent diffusion coefficient) from cytotoxic edema (reduced apparent diffusion coefficient, seen in acute arterial ischemic injury).