Table 1: Pesticide residues in river waters in India. OC=Organochlorine pesticides, OP=Organophosphorus pesticides, Sr.=River sediment. Other abbreviations used are standard ones for pesticides

<table>
<thead>
<tr>
<th>No</th>
<th>Pesticides</th>
<th>Sample</th>
<th>Level</th>
<th>References No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>OC: DDT, HCH, Aldrin, Endosulfan</td>
<td>River Ganges/Patna</td>
<td>DDT=HCH</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>OC: HCH isomers, DDT and metabolites</td>
<td>River Cauvery</td>
<td>HCH4.35-15.84ng/g SâHCH&gt;âHCH&gt;âHDC</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>OC: DDT, âHBC, Aldrin, Endrin, Dieldrin: OP=Dimethoate, Methyl Parathion</td>
<td>Tamliland</td>
<td>HDDTo.69-4.58ng/g Sp’-DDE= in all sites</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>OC: DDT, DDD, âHBC, Aldrin, Dieldrin</td>
<td>Ganges, UP</td>
<td>Levels (ppb) respectively- 3.3-5.3 3.1.73-3.</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>DDT, HCH</td>
<td>Drinking water</td>
<td>47.4, 256.9ng/l, respectively</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>DDT, HCH</td>
<td>River Yamuna</td>
<td>0.24Ig/l, water 0.24mg/kg S</td>
<td>6</td>
</tr>
</tbody>
</table>

Ganges water, which are mainly pesticides and other industrial discharges.

One such episode is the vehemently increasing frequency of cancers and birth defects among the villagers in a remote hamlet called Swarga (it is a Kannada word meaning- heaven!) in Kerala state of south India, possibly due to high levels of the pesticide- Endosulfan being sprayed for years to protect the cashew plantation. A few scientists believe that water reservoirs (rivulets) around the site might show very high levels of said pesticide (although not conclusively known), responsible for hazardous clinical conditions.

With minor differences, other countries too present similar scenarios. These studies simply indicate that we are not safe in a world of increasing industrialization, lack of sanitary engineering and abysmal insipidity of law-enforcing authorities. Increasing teratogenesis, cancer-rate and other associated health problems (for example-infertility), therefore have correlation with the quality of drinking water. In this context, the study by Siddique and Ahmad has come as another warning to the public as well as government.

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REFERENCES

CT scan of brain was normal. CSF examination showed 70 cells with 60% lymphocytes with normal sugar and proteins. Pure tone audiomerty revealed bilateral sensorineural deafness, more severe on the right side. Electrophysiological studies revealed generalized sensory neuropathy.

MENINGEAL CARCINOMATOSIS—AN UNUSUAL CAUSE OF MULTIPLE CRANIAL NERVE PALSYES AND SENSORY NEUROPATHY

Sir,

Neurological manifestations of a distant primary malignancy can occur due to intraparenchymal metastasis, leptomeningeal carcinomatosis and paraneoplastic syndromes.1 Leptomeningeal carcinomatosis is diffuse involvement of meninges by the tumor cells and involves the cerebral hemispheres, cranial nerves & spinal cord and roots.2 We present a patient with rare combination of all the three forms of extensive neurological involvement due to dissemination from a silent primary site.

A 60-year-old man presented with progressive weakness in right upper and lower limbs, impaired hearing in right ear, and diminished vision in right eye over past one month. Later he developed inability to close right eye and deviation of angle of mouth to left side. There was no headache, fever, convulsions, or altered sensorium.

On neurological examination, visual acuity was diminished in the right eye, but well preserved in the left eye (6/18). A sensorineural deafness and lower motor neuron facial palsy on the right side were noted. There was hypotonia on right side with normal power on both sides. He had decreased pain, joint position and vibration sense on right side. All reflexes were diminished and plantars were extensor on right side.

MRI with gadolinium contrast revealed multiple intraparenchymal lesions with enhancement (Figure 1) in both cerebral hemispheres. A repeat CSF cytology detected adenocarcinoma cells and confirmed the diagnosis of Meningeal carcinomatosis.

CT scan of chest revealed a solitary pulmonary nodule (Figure 2) in the posterior segment of right upper lobe. Transpleural CT guided biopsy of the lesion confirmed an adenocarcinoma. The patient was discharged
First described by Eberth in 1870, meningeal carcinomatosis has an incidence of 1-5% in all solid tumors and is a presenting manifestation in 6-38% of unknown primary malignancy. Though meningeal carcinomatosis is uncommon, this case emphasizes the need for detailed neuroradiological and oncological investigations with repeated CSF in patients with progressive cranial nerve palsies of apparently unknown cause.

CT scans are normal in about 60% of the cases while gadolinium enhanced MRI has a sensitivity of about 70%. MRI may demonstrate thickening of nerve roots, subependymal or pachymeningeal enhancement, or multiple enhancing nodular deposits.

The gold standard for diagnosis is CSF cytology; though positive in only 60% cases, its sensitivity increases to 85% after 3 lumbar punctures. Lymphocytic pleocytosis as in this case is strongly supportive.

This case had a rare presentation with involvement of unusual cranial nerves (II, VII, & VIII), intraparenchymal metastasis, and paraneoplastic sensory neuronopathy.

The treatment of meningeal carcinomatosis consists of radiotherapy (2400 rads) with chemotherapy using methotrexate, cyto-A, and thio-tepa. The prognosis is usually gloomy, median survival being 4-6 weeks after diagnosis.