Aspirin-induced Non-cardiogenic Pulmonary Edema

Sir,

Aspirin is a non-selective inhibitor of cyclooxygenase (Cox) enzyme, which inhibits production of prostaglandins. Several anaphylactic reactions have been reported in response to aspirin therapy, viz asthma, angioneurotic swelling, urticaria, fixed drug eruptions etc. We are reporting an unusual case of acute respiratory distress syndrome (non-cardiogenic pulmonary edema) caused due to hypersensitivity to aspirin.

A 25 year old chronic smoker male was prescribed Aspirin (Dispirin) 325 mg for headache. There was no history of prior ingestion of aspirin. Half an hour after taking the drug he developed severe breathlessness and cough, which was associated with pink coloured frothy sputum. Patient was hospitalized immediately. There was no history of fever, thoracic trauma, head injury, toxic gas inhalation, chest pain, aspiration of gastric contents, radiation injury, intake of any other drug (Opioids, Phenothiazines, TCA’s, Amiodarone, Chemotherapeutic agents, Paraquat), tuberculosis, drowning or heavy smoke inhalation.

Physical examination revealed a young average built cyanosed individual with no pyrexia, jaundice, clubbing or lymphadenopathy. Pulse rate was 140/min. Respiratory rate was 42/minute. JVP was normal and blood pressure was 90/70 mmHg. Oxygen saturation (SpO₂) was 54%. There were bilateral extensive crepitations. Cardiac auscultation was normal. There was no hepatosplenomegaly. Laboratory investigations done were-Hb 10 gm%, total leucocyte count 12000/cu mm, differential count N60L30E8M2, platelet count 2.5 lacs/cu mm, serum bilirubin (0.7 mg/dl), urea (20 mg/dl), creatinine (1 mg/dl) and sugar (random) was 90 mg/dl. The rapid test for HIV was non-reactive. ECG and 2D echocardiography was within normal limits. Skiagram chest on admission showed multiple, irregular small and medium-sized fluffy opacities bilaterally in the lung.

Patient was put on pressurized oxygen, parenteral methylprednisolone, antibiotics and other supportive therapy. The oxygen saturation and respiration gradually improved in the next 48 hours. By 5th day of hospitalization there were only minimal crepitations in the chest. Total radiological clearance occurred by 6th day.

Clinical and radiological features suggested diagnosis of pulmonary edema which improved with therapy. History suggested that the disease had occurred due to the ingestion of single tablet of aspirin.

Pulmonary edema is defined as excess of extravascular water within the lungs because of either increased permeability of small vessels and alveolar walls causing ARDS or increased hydrostatic pressure in the small pulmonary vessels causing high pressure pulmonary edema as seen in LVF. Causes of ARDS include infections, lung contusion, toxic gas inhalation, pulmonary embolization, aspiration of gastric contents (Mendelson’s syndrome), near drowning, radiation injury or drug overdose (salicylate, opioid, bleomycin and other cytotoxic drugs, paraquat). Table 1 depicts radiological differences of high pressure and increased permeability pulmonary edema.

Table 1: Radiological differences of high pressure and increased permeability pulmonary edema

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<thead>
<tr>
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<th>High Pressure Pulmonary Edema</th>
<th>ARDS</th>
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<tbody>
<tr>
<td>Cardiac size</td>
<td>Enlarged</td>
<td>Normal</td>
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<tr>
<td>Upper lobe vessels</td>
<td>Dilated</td>
<td>Normal</td>
</tr>
<tr>
<td>Kerley lines</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Lung shadowing</td>
<td>Central hazy</td>
<td>Peripheral patchy</td>
</tr>
<tr>
<td>Air bronchogram</td>
<td>Unusual</td>
<td>Frequent</td>
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The radiological features in this patient were suggestive of non-cardiogenic pulmonary edema. Exclusion of other causes by history, clinical findings, ECG and 2D echocardiography point towards the single dose Aspirin as the probable etiology. Through salicylate overdose has been reported earlier as a cause of such an event, ARDS as a result of single dose Aspirin is very unusual.

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REFERENCES

Ramsay Hunt Syndrome Presenting as Cranial Polyneuropathy

Sir,