



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.¹ 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).^{1,2} 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

	High Pressure Pulmonary Edema	ARDS
Cardiac size	Enlarged	Normal
Upper lobe vessels	Dilated	Normal
Kerley lines	Present	Absent
Lung shadowing	Central hazy	Peripheral patchy
Air bronchogram	Unusual	Frequent

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,³ ARDS as a result of single dose Aspirin is very unusual.

MK Jain*, M Indurkar, S Malviya***, V Kastwar*****

*Professor and Head, **Associate Professor, ***Postgraduate Student, Department of Medicine, SS Medical College, Rewa (M.P.).

Received : 17.6.2005; Revised : 15.1.2007; Accepted : 19.2.2007

REFERENCES

1. Ashbough LDB, Bigelow DB, Pelty TC, Levine BC. Acute respiratory distress syndrome in adults. *Lancet* 1967;2:319.
2. Pelty TC, Ashbough LDB. The adult respiratory syndrome: clinical features, factors influencing prognosis and principles of management. *Chest* 1971;60:233.
3. Jackson Roberts II, Hason D. Marrow-Analgesic Antipyretic, Antiinflammatory agents and drugs employed in the treatment of gout, salicylates. Cited in Goodman and Gilman's- The Pharmacological basis of Therapeutics, 20th Edition, 2001;696-704.

Ramsay Hunt Syndrome Presenting as Cranial Polyneuropathy

Sir,

Ramsay Hunt syndrome (RMS) is the herpes zoster infection of the geniculate ganglion of seventh cranial nerve. This is the common form. We saw a case who presented with cranial polyneuropathy. A forty four-year-old male named AK presented with pain and vesicular eruption in the left mastoid region and over the left pinna. Two days after this he felt difficulty in swallowing food and water and simultaneously noticed facial asymmetry. He also felt vertigo and tinnitus. There was no diplopia, ptosis or facial pain, headache, vomiting, convulsions, or loss of consciousness. There is no history of weakness of limbs or bladder and bowel involvement. He was not a diabetic or a known case of tuberculosis. There was no family history of similar problems. His general examination did not reveal any abnormality. His higher functions were normal. However he had lower motor neurone (LMN) type of facial nerve lesion on the left side. There were also LMN type of ninth and tenth cranial nerve on the same side. He had neural type of deafness on the left side. No other cranial nerves were affected and he had no motor or sensory deficit. There were no cerebellar signs or signs of meningeal irritation. Skull and spine were normal. CSF study did not reveal any abnormality. CT scan of the brain was also normal. Routine haematological tests, blood glucose and X-ray chest were normal.

As there were no evidences of any other cause of multiple cranial nerve lesions in this case like basal meningitis, infiltration of the base of the brain by malignancy or evidence of brain stem vascular or neoplastic lesion; it was diagnosed as a case of multiple cranial nerve involvement (polyneuritis cranialis) due to Varicella zoster, variant of R H Syndrome. The case was treated with Acyclovir 750mg every 8hourly by intravenous route for 10days. The patient improved remarkably but continued to have impaired hearing after three months of follow up.

Our case was a case of Varicella zoster involvement of facial nerve as the illness started with facial weakness with vesicular eruption over pinna and mastoid area. This case fits into the diagnosis of Ramsay Hunt syndrome. This syndrome is more commonly seen in 5th and 6th decade¹ as happened to our case. However, this case had added features to the classical description of RMS. There were ipsilateral other cranial nerve lesions like 8th, 9th and 10th cranial nerves. Few others have also noticed other cranial nerve involvement. The other cranial nerves that are affected are VIII, IX, V, and X in that order.² Facial numbness, tongue numbness, corneal anaesthesia may be associated with R H Syndrome. But other cranial nerve involvement is quite rare.³ Most of the patients recover to some extent, but it may be incomplete.¹ Our case had almost complete recovery except some hearing impairment on the affected ear after three months of follow up.

KN Padhiary*, A Mishra, P Routray****

*Associate Professor, **Postgraduate students, Department of Medicine, SCB Medical College, Cuttack - 753 007.

Received : 28.5.2005; Revised : 6.3.2007; 13.3.2007

REFERENCES

1. Devriese PP, Mosker WH. The natural history of facial paralysis in herpes zoster. *Pin Otolarygo* 1988;13:289-98.
2. Dickins JRE, Smith JJ, Graham SS. Herpes zoster oticus diagnosis and treatment. *Irish Med J* 1992;85(3):1156.
3. Mehta J, Mahajan V, Khanna S. Disseminated zoster with polyneuritis cranialis. *Neurol India* 2002;50:228-9.

Hypoglycemia and Catatonia

Sir,

Middle aged male with history of diabetes mellitus on oral hypoglycemic presented with acute onset of catatonia. Investigation revealed low blood glucose level. He responded promptly to intravenous dextrose.

Catatonia is a state of apparent unresponsiveness to external stimuli in a person who is apparently awake. Other than primary psychiatric disorder (e.g. Schizophrenia, substance abuse or mood disorders), various metabolic and neurological disorder also may present with catatonic symptoms.

Fourty two years defence civilian was brought by his friends to the causality of Naval Hospital at Port Blair on 22 June 2006, with history of not talking and not performing his routine works since morning. There was no history of convulsion, loss of consciousness or fall. The individual is staying with his friends in a rented house leaving his family behind at Kerala. According to one of his friends he was taking some Ayurvedic or allopathic medicines prescribed by a medical practitioner at Kerala for diabetes. They denied any history of abnormal behavior in the past or history substance abuse.

On examination, he was awake but mute, sitting on the bed. Temp. 98.4 F. Pulse – 84/min, regular. BP – 116/84 mmHg. There was no pallor, icterus or lymphadenopathy. He was spontaneously moving all 4 limbs but not following any verbal command. Resistance to passive movements was present in all muscle groups. DTJ – Normal. Plantars were flexor. There was no abnormal posture or movement. Meningeal signs were negative. Examination of cardiovascular system, respiratory system and abdomen were essentially normal.

Random capillary blood glucose was 36mg/dl by glucometer. Venous blood glucose – 40 mg/dl. Hemogram/Urine analysis/LFT – WNL.ECG – WNL. 50 ml of 25% Dextrose was administered. He showed prompt response, started talking and following command. The individual revealed that he was on Glibenclamide 5 mg BD. He was not feeling well in the previous day and did not take his meal properly.

He was admitted in the ward for hypoglycemia