Research Paper

Volume : 2 | Issue : 6 | June 2013 • ISSN No 2277 - 8179

Fatal Anaphylactic Reaction to Intramuscular Diclofenac Sodium

Medical Science

KEYWORDS : Fatal Anaphylaxis, Hypoxic brain damage, diclofenac sodium, intramuscular

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ABSTRACT

Diclofenac sodium is a non-steroidal anti-inflammatory drug widely used as an analgesic and anti inflammatory drug on OPD basis as well as self medication. We present a case of hypoxic brain damage that occurred after intramuscular injection of diclofenac due to a severe anaphylactic reaction. A 23 year old male patient treated for bodyache with 50 mg diclofenac intramuscularly. Twenty minutes later, he collapsed and developed coma and cardio respiratory arrest. After cardiopulmonary resuscitation he was reverted and put on ventilator support .Throughout his admission period he was comatose and received controlled ventilation of the lungs. Magnetic resonance imaging of brain showed signs of hypoxic brain injury and the patient died from central cardiopulmonary failure 16 days later. Intramuscular treatment with non-steroidal anti-inflammatory drugs such as diclofenac has rare but potentially severe side-effects. Therefore, intramuscular injections are inappropriate and should be replaced with oral or rectal treatment, which has similar absorption profiles. The rare and fatal adverse reaction of most commonly used drug prompted us to report this case.

INTRODUCTION

Diclofenac sodium is a non-steroidal anti-inflammatory drug (NSAID) widely used as an analgesic and anti inflammatory drug on OPD basis as well as self medication. Commonly used routes of diclofenac administration are oral, intramuscular, intravenous, transdermal and rectal. We report a case of severe fatal hypersensitivity reaction to IM diclofenac given on OPD basis for bodyache resulted to hypoxic brain damage and then death of a 23 yrs male patient. The rarity of the case prompted us to report this case.

CASE REPORT

- A 23 years young male patient, working as laborer came to medical emergency ward at around 4 a.m. for generalized bodyache and mild epigastric and generalized chest discomfort since last 3-4 hours. When patient arrived, he came walking and able to give history himself almost comfortably. He had taken his dinner at 8p.m. last evening. No history of fever or breathlessness. Past history of having polio affecting left leg since age of 3 years. Family history, personal history was not significant. No history of any drug allergy previously. He was fully conscious and oriented. Vitals, respiratory and CVS examination was clinically normal, spo2 was 98% at room air. ECG was done which was normal. Random blood sugar was done by glucometer which was 118 mg/dl.
- After all this examination, considering non specific bodyache patient was given inj. Diclofenac sodium 2cc (50mg) IM stat.
- After 20 minutes of this injectable drugs patient became restless, developed difficulty in breathing and speech, neck discomfort with choking sensation .
- On examination, patient was conscious and somewhat anxious. Stridor and chest indrawing was present. At that time, Temperature was normal, pulse rate 92/min regular, blood pressure 112/70 mmhg in sitting position, respiratory rate was 20/min SpO2 on air was 98%. Respiratory auscultation suggestive of bilateral generalized mild bronchospasm. CVS examination was normal.
- Considering allergic reaction , Patient was immediately given inj. Chlorpheniramine IV, inj. Hydrocort 100mg IV and inj. Deriphylline 200mg IV stat.
- Patient not responded, and within five minutes patient suddenly collapse and became unconscious. On examination, patient cyanosed, pulse was not palpable, blood pressure was not recordable and cardiac sounds(s1s2) was absent. Immediately cardiopulmonary resuscitation has started. Adrenaline (0.5 ml of 1:10,000) and atropine 2cc(1.2mg) was injected intravenously and both repeated till cardiac activity returns. Simultaneously Patient's trachea was intubated and IPPV given with AMBU. Patient was reverted within 5 minutes and he was put on a ventilator. Fluids were infused rapidly and vasopressor support (inj. dopamine 10 µg/kg/minute) was started to maintain hemodynamic stability.
- After that Patient became unconscious, not responding to even deep painful stimuli. Temperature normal, pulse 124/min, BP was 100/70 mmhg, SpO2 97% with ACMV ventilator support with FiO2 60%.
- CNS examination suggestive of—decreased tone in all four limbs, not moving any of four limbs. All superficial and deep tendon reflexes are absent. Corneal, conjunctival, Oculocephalic and gag reflex was absent. Both pupils are semidilated and not reactive to light. But spontaneous respiratory efforts was present.
- Complete blood count was normal except rise in total count upto 14,600/cumm. CRP was 10 mg/l. Liver and renal function tests, Serum electrolytes level were normal. Repeat ECG s/o sinus tachycardia, bedside 2D echo was normal, bedside CXR was normal. USG Abdomen was normal. Central fundus examination was normal.
- Patient was treated with IV antibiotics, IV deriphylline, IV hydrocortisone, and IV fluids with sos nebulisation with salbutamol respiratory solution.
- MRI brain with MR angiography was done on 5th day of admission which suggests "changes of global hypoxic ischemic encephalopathy with cerebellar tonsilar herniation."
- Tracheostomy was done on 11th day of admission.
- Patient remained same without any improvement during his admission period, and died on 16th day of admission due to cardio respiratory arrest.

DISCUSSION:

- NSAIDs are amongst the most frequently used drugs that may cause hypersensitivity reaction. Anaphylaxis to diclofenac is an idiosyncratic reaction and is a rare event. [1] Safety data from clinical trials in the United States have shown that diclofenac sodium has lower rates of adverse reactions than any of the other comparative NSAIDs[2].
- Clinical features of anaphylaxis include increased vascular permeability, vasodilation, hypotension, tachycardia, bronchospasm, interstitial pneumonitis, urticaria, angioedema, and even shock can occur.[3] In the lung, bronchospasm, tissue edema and hypotension all contribute to impaired gas

exchange resulting in hypoxia. Anaphylactic reactions have been reported to trigger cardiovascular events, including myocardial infarction and acute coronary syndromes, even in patients with normal coronary vasculature.[4]

- The reported case of anaphylactic reaction was due to IM administration of diclofenac. This view is supported by the sequential association between the administration of diclofenac and the patient's symptoms and abrupt onset of illness with lack of previous symptoms.
- Causal relationship between the diclofenac and event assessed using Naranjo probability scale[5] revealed a possible relationship (score +3).
- Careful clinical assessment is important so that appropriate treatment can be instituted promptly to avoid further morbidity and mortality. Epinephrine is the first and most important treatment for anaphylaxis; considering its relative safety, when in doubt, epinephrine should be administered. [6]
- To our knowledge, this is a rare case of anaphylactic reaction developing due to IV diclofenac. This communication is to bring awareness that although IM diclofenac sodium is a safe and widely used drug, severe and potentially fatal anaphylactic reactions may occur with its use. Prompt recognition and treatment of serious complications can help avert such untoward outcomes.

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