Correspondence

Probable Hypoglycaemia Induced by Single Dose Nimesulide

Sir,

A 44 years non-alcoholic non-smoker healthy Hindu male from rural region was admitted with history of body ache and headache from 2-3 hours after a full vegetarian meal for which he took 2 tabs of Nimesulide (100mg each) and slept. Patient was found unconscious by the attendant after 6-8 hrs approximately and was not arousable. He was sweating profusely. Patient was taken to the hospital immediately. On the way to the hospital he vomited once. There was no history of fever, head injury, prolonged fasting, any other drug intake, trauma, alcohol intake, heat exposure, overexertion, chest pain, palpitation. No past history of Hypertension, Diabetes Mellitus, Tuberculosis, Gastric Surgery, Similar episodes, Seizures.

On examination patient was afebrile, perspiring profusely and extremities were cold. No pallor, icterus, cyanosis, pedal edema, lymphadenopathy, clubbing. JVP was not raised. Pupils were normal size and reactive to light. Pulse rate was 100/min with regular rhythm. Blood Pressure-140/90 mmHg. Respiratory Rate – 18/Min. Cardiovascular, Respiratory system, Abdominal examination was normal. On Central nervous system examination patient was unconscious but moving all four limbs on deep painful stimulus. Eyeball movements were normal. Both nasolabial folds were equal. No other Cranial nerve palsies were detected. The plantar reflex was extensor on right and flexor on left. On motor system examination the tone was increased in all four limbs more in Lower limbs than upper limbs, the Deep tendon reflexes were also exaggerated. The sensory system could not be examined. There were no signs of meningial irritation, no evidence of tongue bite or sign of any external injury.

From emergency room the patient was immediately shifted for CT imaging of head and electrocardiograph which revealed no abnormality. The chest X-ray PA view also showed no abnormality. Laboratory Investigations revealed Blood sugar 54mg/dl on admission. Hb – 12.2 g%, TLC – 13200/cumm, DLC– 85/11/03/01, MP-not seen, Platelets–2.25 lakhs/ cumm Blood Urea-19mg%, VLDL 52.2mg%, TG 261mg%.Urine complete examination was not be done due to unwillingness of the patient’s attendant’s.

With the Blood Sugar report of 54mg% immediately he was infused with 100cc of 25% dextrose intravenously. Soon after which he regained consciousness with normal higher function and no neurological deficit. Repeat Blood sugar level was 130 mg%. Patient was observed for the next 48hours and he remained conscious and alert with no neurological deficit or recurrence of episode. He was discharged in healthy condition with the diagnosis of hypoglycaemic encephalopathy caused by the intake of Nimesulide.

**Discussion**

Non-steroidal anti-inflammatory drugs (NSAIDs) constitute a family of drugs which taken as a group represents one of the most frequently prescribed drug around the world. Nimesulide has analgesic, anti-inflammatory and anti-pyretic activity due to potent inhibitory effects on the COX-2 enzymes. The mechanism of action has been attributed to a unique chemical structure of the sulphonanilides class of NSAIDs. Nimesulide bears a good gastric tolerance compared to other NSAIDs. Hepatotoxicity in form of acute hepatitis and pure cholestasis is noted. The mechanism of hepatic toxicity caused by Nimesulide has been noted to be due to a metabolic cause.

Cases of hypoglycaemia and hypothermia in overdose due to Nimesulide administration have been documented in paediatric age group. That is why use of Nimesulide has been banned in paediatric age group in many countries due to its potential side effects.

Some non-steroidal anti-inflammatory drugs (NSAIDs) incidentally induce hypoglycaemia, which is often seen in diabetic patients receiving sulphonylureas. Possible mechanism for hypoglycaemia caused by NSAIDs could be that they influence various ion channel activities, thus may cause hypoglycaemia by affecting ion channel functions in insulin secreting beta cells. There is a case report of Hypoglycaemia in a diabetic patient with the chronic use of Nimesulide, which is a clue for renal failure. Although toxicity and various side effects due to chronic administration of Nimesulide has been reported, to the best of our knowledge there is no report about hypoglycaemia due to a single dose ingestion of Nimesulide in a non-diabetic patient with normal kidney function.

Adverse effect caused by any drug can be assessed by applying causality analysis using WHO probability scale or Naranjo ADR probability scale for it to be considered as an adverse drug reaction by the offending drug.

The WHO probability scale measures how likely an event is to happen. It has been graded as Certain, Probable, Possible, Unlikely, Conditional/unclassified and Unassessable/unclassifiable. A Probable adverse drug reaction is defined as a clinical event, supported by a laboratory test abnormality, in a reasonable time relation to administration of the drug, unlikely to be attributed to concurrent disease or other drugs or chemicals, and which follows a clinically reasonable response on withdrawal (de-challenge). Our patient fulfills all these criteria.

The Naranjo ADR probability scale consists of a questionnaire designed by Naranjo et al. for determining the likelihood of whether an ADR (adverse drug reaction) is actually due to the drug rather than the result of other factors. The scoring for Naranjo algorithm defines a score ≥9 as a Definite ADR; 5–8 = Probable ADR; 1-4 = Possible ADR; 0 = Doubtful ADR. In our patient the score of 7 on this scale defined hypoglycaemia as a probable ADR caused by single dose of Nimesulide.

**Conclusion**

We conclude that hypoglycaemia caused by single dose of Nimesulide can be considered as a Probable adverse drug reaction.

**References**


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