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Liver function abnormality & Vestibular symptoms with Azithromycin

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ABSTRACT

SUMMARY: 38 years old female patient having sore throat, malaise feverishness and congestion of posterior pharyngeal wall took azithromycin (zithrox; McLeods) 500mg once daily for three days and started having early morning nausea and aversion for food and water, which progressed to become intractable within 10 days with complete aversion for food and water. Liver function tests were found deranged and USG abdomen revealed evidence of fluid around gall bladder and dilatation of biliary radicles. Patient gradually improved. After 8 months patient again had sore throat with fever and again took Azithromycin (zithromycin; McLeods) 500mg once daily orally for 3 days. Again patient started having early morning nausea and aversion for food and water which again progressed within 10 days to become intractable with severe weakness and malaise. This time LFTs revealed isolated rise in Alkaline phosphatase.

Keywords : ADR, Azithromycin, Hepatotoxicity, Vestibular symptoms

Introduction:

Azithromycin belongs to the azalide subclass of macrolide antimicrobials. Macrolide antibiotics are so named because they contain a many membered lactone ring (14 membered ring for erythromycin and 15 membered ring for azithromycin) to which are attached one or more deoxy sugars. Azithromycin differs from erythromycin only by the addition of a methyl substituted nitrogen atom in to the lactone ring. The structural modification improves acid stability and tissue penetration and broaden the spectrum of activity¹. Present case study is a case of rarely documented and reported adverse effect of azithromycin.

Case study:

38 years old female patient, chest physician by profession, known case of bronchial asthma and hypothyroidism taking low dose inhalational corticosteroids round the clock and levosalbutamol MDI as and when required for bronchial asthma and thyroxin (Eltroxin,) 75 µg daily for hypothyroidism. Patient had sore throat, malaise and feverishness, on examination found to be having congestion of posterior pharyngeal wall. Patient took azithromycin (zithrox; McLeods) 500mg once daily for three days from 18th to 20th May 2010. Patient started getting early morning nausea and aversion for food and water, which was progressive and within 10 days patient had intractable nausea and vomiting after eating with complete aversion for food and water. On 4th June 2010 Liver function tests revealed Bilirubin- Total 3.0mg/dl (Direct 1.9mg/dl and Indirect 1.1mg/dl), SGOT - 2390 IU/L, SGPT- 3500 IU/L, Alkaline phosphatase, total protein, serum albumin, serum globulin and A:G ratio were found normal. USG abdomen revealed evidence of fluid around gall bladder and dilatation of biliary radicles. Patient was kept NPO for 48 hours and Ciprofloxacin 100mg IV 12 hourly and Metronidazole 500mg IV 8 hourly was administered along with IV Ondansetron (Periset; Cipla) 40mg as and when required. After 48 hours USG was repeated in which there was no evidence of fluid but slight dilatation of biliary radicles was present. LFTs were repeated on 8th June 2010 with following readings; Bilirubin- Total 1.3mg/dl (Direct 0.6mg/dl and Indirect 0.7mg/dl), SGOT- 230 IU/L, SGPT- 860 IU/L and Alkaline phosphatase- 267IU/L.

Patient gradually improved without any body suspecting any kind of adverse event. After 8 months patient again had sore throat with fever (1010 F) and again took Azithromycin (zithromycin; McLeods) 500mg once daily orally for 3 days from 21st to 23rd January 2011. Again patient started getting early morning nausea and aversion for food and water which progressed within 10 days to intractable nausea and vomiting after eating with severe weakness and malaise. LFTs done on 5th Feb 2011 revealed isolated rise in Alkaline phosphatase which was 576- IU/L. USG done on 3rd and 6th of Feb 2011 was found normal. LFTs repeated on 10th Feb 2011 were normal along with other serum biochemistry, Hb, TLC, ESR and C reactive protein. Abdominal symptoms improved gradually with vestibular nausea persisting which was relieved with Flunarizine (Sibelium; Jhonson and Jhonson) 5mg given orally twice a day for 3 days.

Discussion:

Cholestatic hepatitis is the most striking side effect of erythromycin. It is primarily caused by erythromycin estolate and only rarely by the ethylsuccinate or the stearate². Even rarely it is documented to be caused by azithromycin which is considered safer compared to erythromycin or clarithromycin since it has fewer drug interactions. Authors could not find any study establishing relationship between azithromycin and full blown cholestatic hepatitis even though increase in liver enzymes particularly serum transaminase is documented at few places³. In the present case on both the occasions there was some injury to liver since there was severe nausea and aversion for food and water with vomiting after eating that increased gradually over a period of 10-15 days after stopping therapy along with raised liver enzymes though the pattern was different on both the occasions. In the first episode SGOT, SGPT and bilirubin and in second episode alkaline phosphatase were raised. In first incidence though fluid was present around the gall bladder but it can not be considered cholestatic hepatitis or cholecystitis since there were no findings suggestive of hepatitis on USG and alkaline phosphatase was normal and on repeat USG there were no residual changes of inflammation though biliary radicles were dilated. On second exposure the pattern of nausea and

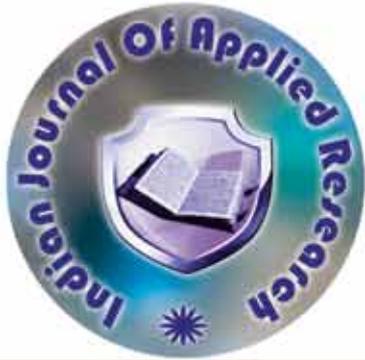
aversion for food and water and vomiting was similar but only isolated increase in alkaline phosphatase was present suggestive of cholestasis.

Another rare adverse effect of macrolide antibiotics is sensorineural injury, both peripheral and auditory in the form of tinnitus and hearing impairment and vestibular involvement in the form of poor balance or dizziness^{3,4}. Authors could not find any clear documentation of vestibular symptoms in the form of nausea but in present study though it can not be es-

tablished, in second episode after abdominal symptoms subsided the patient described the pattern of nausea changing to more like motion sickness and it was relieved with flunarizine. It suggests that in such cases it may be difficult to diagnose vestibular side effects since the patient is already suffering from nausea and so there is a need to suspect these events in more cases than is being done at present. The most striking feature in the present study is the severity of nausea which may be because of the involvement of both hepatic and vestibular regions.

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