Abras Precatorius Poisoning

Sir,

This is with reference to Vaidya et al’s correspondence on the case report “Poisoning due to white seed variety of Abrus precatorius” by Pillay et al. Vaidya et al have noted that the use of broad spectrum antibiotics like meropenem and ceftriaxone by Pillay et al has no evidence based rationale. However, they themselves have mentioned the antitodal therapy for Abrus poisoning which has no evidence based rationale. They state that the eminent ayurvedic physician V.M. Gogte has mentioned in his textbook that the antitodes for Abrus precatorius poisoning are the juice of Amaranthus (Tandulja) and sugar. In our opinion, this data seems to have no evidence based rationale. Moreover, they themselves mention that the Amaranthus juice and sugar used as antitodes in the treatment of Abrus poisoning needs to be investigated in an experimental toxicity study. ‘Ellenhorn’s Medical Toxicology: Diagnosis and Treatment of Human Poisoning’, the classic treatise on clinical toxicology states that there is no antitode for treating Abrus poisoning.

The mention of use of broad-spectrum antibiotics in the patient reported by Pillay et al suggests that the authors have disclosed all the facts in relation to the treatment of such a patient, and haven’t reported information needed only to the liking of the case report. We do agree with the reasoning provided by V.V. Pillay in this respect, in his ‘reply to the author’, that antibiotics were tried as an empiric measure before it could be conclusively established that the underlying cause for gastrointestinal clinical features was plant toxin related and not microbial in nature.

Regarding the toxic profile in their case, Pillay et al wondered whether the white seed variety of Abrus precatorius had a different toxic profile as compared to the usual reported red seed variety of Abrus precatorius. The chemical constituents of both the white and red varieties of Abrus precatorius seeds are the same, and therefore, in our opinion there wouldn’t be any difference in their toxicity profile.

Severe Hypoglycemia in a Patient with Type 2 Diabetes Mellitus on Metformin Monotherapy

Sir,

Mr. R. G. was a 56 year old having type 2 diabetes mellitus for 19 years and essential hypertension for 5 years and was on regular treatment with Metformin 250 mg, after lunch and 500mg after dinner and Tab. Enalapril 5mg, after breakfast. He was admitted as an emergency case in agitated, rowdy and confused state.

On examination, besides the above-mentioned findings, he was totally disoriented in time, place and person. There were no lateralizing signs in CNS. Examination of other systems did not reveal any abnormality. He had not missed any meals and was regular as regards exercise and medications. His random plasma glucose on admission was 49 mg%. S.creatinine was 0.9 mg%., Liver function tests and TSH were within normal limits. He was treated with 50 ml of 25% glucose bolus followed by one pint of 5% glucose over the next 4hrs. He made an uneventful recovery following administration of intravenous glucose and was discharged on 1500 calories, high complex carbohydrate, low fat, salt restricted diet and exercise in the form of 30 min brisk walk. Presence of classical neuroglycopenic symptoms, documentation of hypoglycemia during the symptoms; and prompt recovery following administration of intravenous glucose clearly established the diagnosis of hypoglycemia in this patient on Metformin monotherapy. After improvement in his neurological status he was interrogated in details. He was a senior computer professional. He did regular self-glucose monitoring and systemically recorded his data in a tabular form. On inspection of the data, it was observed that he had quite a few readings of capillary blood glucose values between 60-70 mg%, and many of these coincided with symptoms such as confusion and irritability. However, he did not consult a doctor and made self-adjustments in metformin dosage.
There are very few case reports of hypoglycemia in patients on Metformin monotherapy. UK Prospective Diabetes Study has reported fewer hypoglycemic episodes with Metformin monotherapy as compared to Sulphonylurea monotherapy, but more than patients on diet alone. One or more hypoglycemic episodes were reported in 6% of patients on Metformin monotherapy and one of these episodes was severe.  

Severe hypoglycemia in an elderly patient on Metformin monotherapy was also reported by Zitzmann. The patient was also on ACE inhibitor and NSAID. ACE inhibitors increase insulin sensitivity and could cause hypoglycemia in patients on anti diabetic medications.  

Our patient was taking enalapril for 5 years and was also on metformin for a long time. Thus drug interaction between Metformin and Enalapril possibly contributed towards hypoglycemia. Other possible contributing cause could be natural improvement in insulin sensitivity due to aggressive lifestyle changes and thus reduction in need for Metformin therapy. 

A case of metformin-induced severe hypoglycemia presenting with neuroglycopenic symptoms is presented to bring forward a point, that possibility of hypoglycemia should not be ignored in patients on Metformin monotherapy presenting with symptoms suggestive of hypoglycemia. ACE inhibitors are commonly co prescribed with Metformin. Since these agents increase insulin sensitivity, the chances of hypoglycemia are increased. 

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Received : 28.12.2006; Revised : 17.4.2007; Accepted : 10.5.2007

REFERENCES

Macrophage Activation Syndrome: Experience from a Tertiary Referral Centre

Sir,

We read with pleasure the study ‘Macrophage activation syndrome: Experience from a tertiary referral centre’ by Dr. L Pinto et al and the accompanying editorial ‘Macrophage activation syndrome’ by Dr. VR Joshi in the March 2007 issue.

Being from rheumatology practice this data includes patients with rheumatic diseases and the emphasis in the editorial also reflects the same.

While Macrophage activation syndrome (MAS) is the term applied to Haemophagocytic Lymphohistiocytosis (HLH) associated with rheumatic diseases, probably the majority of cases of Haemophagocytic Lymphohistiocytosis seen by the general physician are related to infectious agents and deserves emphasis. The diseases we have commonly seen being associated with HLH are dengue and thyroid. The relative frequency of association between Mycobacterium tuberculosis, Salmonella typhi, Leishmania, Brucella, viruses and HLH suggests that the syndrome results from a poorly regulated or inappropriate Th1 response to intracellular pathogens.

The presence of HLH in association with an infectious agents may obscure the diagnosis of the infectious disease itself. Therefore all patients meeting the criteria for diagnosis of HLH should undergo diagnostic tests for the above mentioned infections. The extensive testing associated with this disorder should be guided by epidemiological data including factors like the patient’s travel history, medical history and clinical characteristics including immunocompromise. Since there are associated with this syndrome it becomes all the more necessary for the physician to work in close associated with the pathologist and the microbiologist to clearly define the underlying cause.

Recognition of the triggering infectious agent is important since most of these infections are treatable. While the prognosis of HLH associated with rheumatic diseases is grave, if the cause is an infectious agent, antimicrobial treatment and supportive care alone leads to complete recovery in majority of the cases.

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Received : 11.3.2007; Accepted : 13.4.2007

REFERENCE

Reply from the Author

Sir,

We thank Dr. Rajeev Soman for the pertinent remarks that he has made. Macrophage activation/ Hemaophagocytic syndrome is a feature of many infectious diseases, some of which have been listed by Dr. Soman.

It is evident that the population we studied comprised mainly of rheumatic diseases and malignancies and in that respect it was biased. However, during the period of our study the bone marrow registry did not contain cases of infectious disease induced macrophage