Low Dose Methylprednisolone Induced Bradycardia

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Abstract
Methylprednisolone induced arrhythmias, especially bradycardia, are well known. Most of the available reports suggest the occurrence of these arrhythmias with high dose intravenous therapy. We, hereby report a case of low dose methylprednisolone induced bradycardia.

Introduction
High dose intravenous methylprednisolone is an important therapeutic modality for many autoimmune conditions. Adverse effects of this therapy include hypertension, hyperglycaemia, fluid and electrolyte disturbances and cardiac arrhythmias (both tachyarrhythmias as well as bradyarrhythmias). Here we report a 33 year old male patient who developed sinus bradycardia during therapy with low dose intravenous methylprednisolone for clinically and radiologically suspected acute eosinophilic pneumonia. Bradycardia recovered after cessation of steroid therapy.

Case Report
A 33 year old male, was admitted as a case of sepsis with acute lung injury. On fifth day of admission, he developed rapidly worsening dyspnoea and hypoxaemia. Chest X-ray showed bilateral peripheral fluffy opacities with central sparing (photographic negative of pulmonary oedema). He had to be intubated and put on mechanical ventilatory support. On the basis of clinical suspicion and radiological picture, he was suspected to be suffering from acute eosinophilic pneumonia and was initiated on intravenous methylprednisolone 40 mg three times a day as an infusion over 30 minutes each. By next day, his oxygenation status improved, however he developed sinus bradycardia (minimum HR 38/min). Clinically his BP and cardiovascular system examination was normal. His serum potassium was 4.3 meq/dL, serum calcium (corrected) was 9.8 mg/dL and serum magnesium was 1.9 mg/dL. ECG showed sinus bradycardia (Figure 1) and 2D-Echo showed structurally normal heart. Methylprednisolone was suspected to be the culprit and hence was stopped. He recovered from bradycardia 42 hours after the last dose (Figure 2). Thereafter, he was put on low dose oral steroids and was extubated the same day.

Discussion
Intravenous methylprednisolone is an important therapeutic modality for treating many life threatening diseases. Rhythm disorders have been reported in 1% to 82% of patients receiving such therapy and these include sinus bradycardia, atrial fibrillation/flutter, and ventricular tachycardia. The available literature suggests that bradycardia occurs with high dose steroids (intravenous methylprednisolone at a dose of 30 mg/Kg body weight). It has been reported after single and consecutive daily doses, and its onset may occur as early as during administration of the methylprednisolone to as
late as several days afterward. The late development of arrhythmias may make it difficult to identify methylprednisolone as the cause, if the association is not appreciated. The patient in our case developed bradycardia after about 36 hours of the first dose of intravenous methylprednisolone (Figure 3).

Patients may remain asymptomatic or may experience palpitations, altered sensorium and even cardiac arrest. Meticulous cardiac monitoring is necessary and depending upon the clinical status, they might require administration of chronotropic, antiarrhythmic agents or temporary cardiac pacing. The reported duration of arrhythmias has varied from hours to days. Our patient recovered after 42 hours of last methylprednisolone dose (Figure 3).

The exact mechanisms underlying the development of arrhythmias due to steroids is unknown, however the proposed mechanisms include (i) direct action on the myocardial cell membrane and via alterations in cardiovascular sensitivity to catecholamines (ii) alteration of the stimulation threshold of myocardial cells due to rapid electrolyte shifts across myocardial cell membranes (iii) methylprednisolone induced expansion of plasma volume and hypertension, triggering a reflex bradycardia (iv) reaction to excipients in the steroid preparations. Our patient was normotensive, had normal haemoglobin, haematocrit and serum electrolytes, hence it is difficult to postulate a definite cause for bradycardia. Possibly underlying aetiology was multifactorial.

Conflicts of interest
None identified.

Conclusion
Sinus bradycardia, can occur following low dose regimen of methylprednisolone. A high index of suspicion is required to make this diagnosis and stoppage of drug can rapidly revert the bradycardia.

References