Correspondence

Reversible Atrioventricular Block in Thyroid Storm

Sir,

The case report “Reversible atrioventricular blocks in thyroid storm”1 was interesting and we would like to offer these comments.

a. Clinical picture of thyroid storm is one of severe hypermetabolism and fever is almost invariable.2 In this case patient was a febrile.
b. Renal function of the patient was not mentioned.
c. Treatment given to the patient is sketchy - not mentioned about beta blocker / iodide given or withheld.
d. Adrenal insufficiency, hypermagnesemia, other infectious / inflammatory conditions were not excluded before diagnosing thyroid storm as a cause of AV block.
e. Patients presenting with heart block on auscultation should have soft S1 with variable intensity, loud S1 can not be explained.3
f. Index patient has clinical features of increased JVP and features suggestive of CCF however has normal 2D Echocardiogram.
g. ECG showing His bundle rhythm as in Figures 1 and 2, patients should be more stable with rare symptoms.

References

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Reply from Author

Sir,

Reference to our article entitled “Reversible atrioventricular blocks in thyroid storm” published in JAPI, March 2011, Vol. 59. The authors welcome and appreciate the questions raised by Dr. Jha, Graded Medical Specialist, our comments are as follows:

1. Thyroid storm is characterized by severe metabolism and fever is invariable. There are no set criteria for diagnosis of thyroid storm. In apathetic form of thyrotoxicosis, all features of hypermetabolism may not be present; hence, thyroid storm is likely to be missed by the physicians in practice. The same thing happened in this case. First of all, she was treated as a case of gastroenteritis where antibiotics and fluids were given with a dose of antithyroid drug and was referred to us in fluid overload state.
2. Renal functions of the patient were normal.
3. Treatment in this case is not sketchy. In the presence of heart blocks, patient was neither given digoxin nor beta blockers or calcium channel blockers, iodine was also not given. Only antithyroid drugs were continued and steroids were added to control the storm. After two days, patient felt slightly better and left the hospital as pacing was advised. As we lost the follow up of this case, we cannot comment anything what happened outside and when the blocks disappeared. At 4 weeks, when patient came to us in good condition, we were surprised to note the pulse which was normal in rate and ECG reverted to normal. Therefore we cannot comment on treatment received outside.
4. All other causes like infections, electrolyte disturbances and myocardiitis were excluded. There was no evidence of potassium disturbance; hence, magnesium disturbance which is concomitant to potassium disturbance was not estimated.
5. Patient with heart blocks or patients with thyrotoxicosis with high output state and patients with fluid overload usually have loud heart sounds including first sound and even may have systolic murmur at outflow tract. The patient had sign of congestive heart failure due to fluid overload which cleared with diuretics. Echocardiogram was normal which was done after diuretic therapy with relief of CHF.
6. The patient had complete heart block with pacemaker below the AV node, may be in bundle of HIS. His bundle electrocardiography was not done. The patient was stable; hence, immediate pacing was not done. It was considered as patient had frequent syncopal attack.

References

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Hypomagnesemia- An Under Recognized Metabolic Disorder

Sir,

We read an interesting article entitled “hypomagnesemia in critically ill medical patients” by Limaye et al published in January 2011 issue of our esteemed journal JAPI (Vol 59, page 19-22). The article is worth praise and a milestone in Indian perspective. Here we would like to add our experience, views and reviews-

A- Isolated hypomagnesemia is unusual but it usually occurs with deficiency of other electrolytes.1 The neurological complication of hypomagnesemia are similar to that of hypocalcaemia and often two coexist and so this aspect should be taken into account while correcting either of them.2 Same way the hypomagnesemia an hypokalaemia have similar clinical manifestations and so this fact has also to be kept in mind while correcting hypokalaemia for example as in differential diagnosis of Guillian-Barre Syndrome (GBS). Like wise we must also see for magnesium levels besides other electrolytes in patients of status epilepticus which will helps in reducing morbidity and mortality associated with it.

B- Authors have found hypomagnesemia with poisoning like organophosphorous compounds but, along with other electrolyte abnormalities, it is quite common in acute aluminium phosphide poisoning which is widely prevalent in northern part of India and often is fatal unless treated promptly.3 Hypomagnesemia is also fatal, of course authors did not find statistically poor outcome with hypomagnesemia. Hypocalcaemia potentiates the effect of excess magnesium which has to be taken care of when dealing a patient of ecclampsia while giving magnesium to such patients.

References

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