A case of Castleman disease with pulmonary hypertension

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Introduction
Castleman disease, also known as Giant lymph node hyperplasia or Angiofollicular lymph node hyperplasia, is a disease of lymph nodes and related tissues. It is not considered as a malignancy but one variety of it acts like lymphoma.

Case report
A 52 year old male was admitted to Teaching Hospital Karapitiya with loss of weight for 1 year, abdominal distension for 6 months and loss of appetite for 1 month. There was no history of fever. He had stable angina and was on anti-anginals, aspirin and verapamil.

On examination he was emaciated, pale and had large, discrete, firm and non-tender lymphadenopathy in the cervical, axillary and inguinal regions. Clinically he had cardiomegaly, a grade 4 pansystolic murmur best heard in the lower sternum and radiating to the axilla and a loud pulmonary second heart sound. Respiratory system was normal except for few bilateral fine basal crepitations. Abdominal examination showed a 5 cm hepatomegaly and splenomegaly.

His haemoglobin was 9.9 g/dL while white cell and platelet counts were normal. Blood picture showed hypochromic red cells while bone marrow examination showed normal active marrow. ESR was elevated at 80 mm/1st hour. Liver enzymes were normal and Mantoux test was negative. Ultrasound scan of the abdomen showed hepatomegaly with normal echo texture and no focal lesions and splenomegaly. There was no intra-abdominal lymphadenopathy or ascites. ECG showed right axis deviation and evidence of right atrial enlargement. Chest radiograph showed gross cardiomegaly and oligaemic lung fields. 2D Echo found severe tricuspid regurgitation and dilated right ventricle and atrium indicative of severe pulmonary hypertension. There was also a small pericardial effusion. Lung function tests were compatible with restrictive lung disease. Biopsy from an axillary lymph node showed features suggestive of Castleman disease. HIV antibody was negative.

Discussion
Castleman disease usually presents with fever, weight loss, fatigue, night sweats, infections and anaemia. Pulmonary hypertension has been rarely reported in association with multicentric Castleman disease. Only three such reported cases were found during the literature search. Of these, two had tested negative for HIV while the other had tested positive. The proposed mechanism for pulmonary hypertension has been promotion of angiogenesis by Interleukin-6 produced in the germinal centres of hyperplastic lymph nodes.

Castleman disease may present in a localized form or a multicentric form. The localized form only affects a single lymph node group, most often in the chest and abdomen. The multicentric form affects more than one group of lymph nodes and also other organs containing...
lymphoid tissue. The multicentric form sometimes occur in HIV positive patients.

Histologically it is divided into a Hyaline vascular type and a Plasma cell type. The Hyaline vascular type is usually clinically localized while the Plasma cell type is usually multicentric.

Treatment decisions depend on the clinical subtypes and not on microscopic subtypes. Localized Castleman disease is treated with surgery and external beam radiation for cases not amenable to surgery. Chemotherapy either alone or in combination with radiotherapy or steroids is used in multicentric Castleman disease.

Monoclonal antibodies like rituximab or tocilizumab can neutralize the targets for IL-6 on cell surfaces.

References

Prolonged haloperidol induced Parkinsonism in a patient with cirrhosis

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Introduction
Haloperidol is a high potency typical antipsychotic agent having an increased propensity to cause drug induced extrapyramidal symptoms (EPS). Prolonged drug induced Parkinsonism with haloperidol use has been reported a few times in world literature 1,2. We describe here a patient who had haloperidol induced Parkinsonism for about seven weeks after discontinuation of the drug.

Case report
A 56 year old lady was admitted to Teaching Hospital Kandy with an acute confusional state which was retrospectively diagnosed as an episode of steroid induced psychosis. On physical examination, patient did not have rigidity, bradykinesia or any other features of Parkinsonism. Haloperidol lactate 10 mg was given intramuscularly to calm the patient and it was followed by oral haloperidol 1.5 mg and benzhexol 2 mg twice a day. Investigations done while she was taking in-ward treatment revealed that she is having cirrhosis (Macronodular cirrhosis in liver biopsy). She was not on any other medication which was likely to give rise to EPS.

She was reviewed in the clinic one month afterwards. Neurological examination revealed symmetrical rigidity of all four limbs. Both leadpipe and cogwheel rigidity was clearly seen. Facial expression and gait were normal. No resting tremors were seen. A mild bradykinesia