

PREMATURE CORONARY HEART DISEASE IN A YOUNG ADULT- PERNICIOUS ANEMIA THE UNDERLYING CULPRIT: A CASE REPORT

Shamaila Burney, Saqib Qayyum Ahmed*, Saerah Iffat Zafar

Pakistan Air Force Hospital Islamabad, *Army Medical College Rawalpindi

INTRODUCTION

Megaloblastic anemia is frequently seen in Pakistan with vitamin B12 deficiency as a leading cause. However prevalence of pernicious anemia is low in our population. Vitamin B12 deficiency can result in elevated homocysteine levels which is associated with atherothrombotic diseases and pernicious anemia can be a cause as reported in recent studies¹⁻².

CASE REPORT

A 24 year old young male was referred to the Medical Department of PAF Hospital Islamabad for workup of severe anemia. Recently the patient had survived myocardial infarction and had undergone percutaneous intervention with stent placement to left anterior descending coronary artery. No risk factors for early coronary heart disease (CHD) could be identified. General examination revealed a pale looking young man with glossitis and oral ulcers. Systemic examination was unremarkable. Investigations revealed a megaloblastic anemia (hemoglobin 5.6 g/dl, mean corpuscular volume 117 fl). Lactate dehydrogenase levels were high (2388 U/L). Reticulocyte count was 2.0%. Bone marrow aspirate showed hypercellular marrow with megaloblastic changes. Serum vitamin B12 levels were low (113 pg/ml) while serum ferritin and folate levels were normal. Malabsorption work up revealed no abnormality. Upper GI endoscopy was negative for giardia or helicobacter pylori. Barium meal and follow through studies showed normal mucosal pattern. Anti gliadin antibodies were negative. However, anti intrinsic factor antibodies were significantly raised (3.8) and anti gastric parietal cell antibodies were also

positive. Keeping in view the past history of CHD in our patient, homocysteine levels were checked and found to be significantly elevated (>15 micromoles/liter). The patient was treated with parenteral vitamin B12 therapy with excellent clinical and hematological response.

DISCUSSION

This young patient presented with features of megaloblastic anemia and a significant past history of premature CHD. Since pernicious anemia is generally perceived to be a disease of elderly, an effort was made to first rule out the commoner causes of vitamin B12 deficiency in young age. Pernicious anemia was ultimately diagnosed as the cause of B12 deficiency in our patient. Vitamin B12 deficiency is often associated with hyperhomocysteinemia. A large number of studies have demonstrated the presence of strong association between hyperhomocysteinemia and atherothrombotic diseases including CHD²⁻⁴. Melhalm et al recently reported the case of a young adult with pernicious anemia and hyperhomocysteinemia presenting with an acute myocardial infarction⁵. To date, no such case has been reported in Pakistan. A recent study in Pakistan however reported that 50% of CHD patients in our population had high homocysteine levels as compared to 40% in West⁶. The premature onset of CHD in our patient without a family history or significant risk factors other than a co existent finding of pernicious anemia led us to determination of homocysteine levels which were found to be significantly raised. Long standing pernicious anemia resulting in vitamin B12 deficiency and hyperhomocysteinemia is the most logical explanation of premature CHD in our patient.

CONCLUSION

Owing to the rarity of this condition in the young, its profound consequences and lifelong substitution therapy required, it is important to recognize and treat pernicious anemia. Finally, we conclude that hyperhomocysteinemia is an

Correspondence: Dr Shamaila Burney, D-53/1, Officers Mess, PAF Complex Sector E-9, Islamabad
Email: drshamailaburney@hotmail.com

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easily modifiable risk factor for thromboembolism and needs to be considered in the workup of those individuals who manifest with atherothrombotic disease such as premature CHD without the usual risk factors.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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