CASE REPORT

ABNORMAL UTERINE BLEEDING: AN UNUSUAL MANIFESTATION

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HOW TO CITE THIS ARTICLE:
S. Aruna, R. Padmaja. "Abnormal Uterine Bleeding: An Unusual Manifestation". Journal of Evidence based Medicine and Healthcare; Volume 2, Issue 15, April 13, 2015; Page: 2352-2356.

ABSTRACT: Women presenting with abnormal uterine bleeding are not uncommon in any Gynaec OPD. However we came across a young woman who had an unusual cause for it and who perhaps had deliberately concealed important Family and Personal history. She had Familial hypercholesterolemia and a very early attack of cardiovascular disease needing a Coronary bypass surgery. Later we came to know that hers was a classic case. She stabilized with conservative management and later absconded. This highlights the fact that any person might have inhibitions about revealing important history and that a careful clinical examination and history taking are vital armaments for any clinician.

KEYWORDS: Detailed history, young women with MI, Hypercholestrolemia.

CASE REPORT: A 26 year old female presented with menorrhagic cycles of six months duration and breathlessness from one week.

Relevant history – age of menarche was 12 years, marital life was 6 years and she had 2 children. Her last childbirth was 4 yrs.' back.

There were no significant features in her family and her personal history.

On examination – she was anaemic, thin in stature. Spine, breast and thyroid seemed normal. General condition and vital data were normal.

Knob like projections were present on her elbows, ankles and wrists (these were later identified to be tendon xanthomas) and there was also a longitudinal scar on her chest and a corneal arcus. Her JVP was elevated, a Parasternal heave was present and auscultation revealed a Pansystolic and an early diastolic murmur.

On persistent questioning she revealed that had a Coronary bypass surgery 3 yrs back and that she had 2 episodes of Myocardial infarction for which Coronary Angiography was done. This revealed Triple vessel disease which necessitated a CABG.

Consultation of a Cardiologist was taken and history revealed Familial Hypercholestrolemia and recurrent episodes of Myocardial infarction in all family members. It was revealed that she was taking anticoagulant Warfarin without any supervision or getting PT INR report.This led to her menorrhagia.

The salient features of her 2 D Echocardiogram were as follows – Post CABG status, AO 2.4% Ejection fraction 56%.

Severe atherosclerotic Aortic stenosis with moderate Aortic regurgitation.

Severe Mitral regurgitation, no regional wall motion abnormality of left ventricle.

Good left ventricle and Right ventricle systolic function.

No pericardial effusion and no Left ventricle clot.

Her investigations were as follows - Haemoglobin 7.4gm% (For which 1 unit compatible o+ve blood was transfused),Total count 7200,DC -Polymorphs 69% lymphocytes 27%, eosinophils 4%, ESR 25 mm/ 1st hr.

RBS 80 mg/dl, Serum creatinine 1.2 mg/dl, Serum Cholesterol 200 mg, HDL – 50, LDL-132, VLDL – 18.

Triglycerides 94, BGT O+ve, after blood transfusion repeat Hb 8.7%, PCV 27.

She was kept on Tab.Clopidogrel 75 mg daily, Tab Atorvastatin daily, Tab Aldactone 25 mg daily. Tab Enalapril 2.5 mg twice a day and Tab Iron and Folic acid one per day.

Pelvic examination and ultrasound were normal. Menorrhagia subsided with a combination of Tranexamic and Mefenamic acid.

After transfusion she absconded from the hospital and was lost to follow up. It was surmised that she had come to Gyn OPD only for a blood transfusion.

DISCUSSION: Predominant hypercholesterolemia has an increase in LDL more than the 95th percentile for age. LDL levels are twice as high as those of unaffected subjects. The Familial form may be Homozygous or Heterozygous.[1,2,3,4,5]

There is a basic deficiency of normal LDL receptors on cell membrane. (Or defects of APO B 100 or increased function of PCSK 9).[1,2,3,4,5]

The heterozygous form is inherited as Autosomal dominant with a high prevalence and the prevalence is 0.2% with a co dominant high penetrance. (1 mutant LDL receptor allele is inherited).[1,2,3]

Family history reveals that approximately 50% of each generation suffers from hypercholesterolemia often with very premature CVS disease.[1]

Where there is a founder zone effect or consanguineous marriage the homozygous form occasionally occurs resulting in more extensive xanthomas and precocious CVS disease in childhood. A Recessive form of this condition has been described recently.[1]

Founder populations like Lebanese Christians, French Canadians, Afrikaaners have a higher prevalence. The normal prevalence is 1 in 500.[1,2,3,4]

There are high plasma levels of LDL C (200-400) with normal triglycerides,seen at birth itself in the cord blood. One parent and 50% of the siblings also have hypercholestrolemia, so manifestation in the family is a supporting feature.[1,2,3,4,5]

They present in childhood or early adulthood with Xanthelesma, Corneal Arcus (before age 40), extensor digitorum xanthomas, prepatellar and Achilles tendon xanthomas and premature cardiovascular disease in the family.[1,2,3,4]

This premature atherosclerosis involves the aortic root and can cause aortic, valvular or supravalvular stenosis and typically extends into the ostia which become stenotic.[1,2]

Untreated, the morbidity and mortality is high and sudden death is not uncommon, especially in the homozygous form which also has tuberous, cutaneous xanthomas as a distinguishing feature.

One has to exclude Nephrotic syndrome, Hypothyroidism, obstructive liver disease, Familial defective APO B 100 and polygenic hypercholesterolemia even though diagnosis is essentially clinical.[1,2,3]

- Management involves.
- Lipid lowering therapies for primary and secondary prevention of CVS disease.
- Assessment of absolute risk.
- Treating all modifiable risk factors and.
- Optimization of lifestyle especially diet and exercise.[1,2,3,4,5]
- Those with greatest absolute risk of CVS disease will derive greatest benefit from treatment.[1,2]
- Dietary counseling and medical advice.[1,2,3,4,5]

Public health organizations recommend thresholds for the introduction of Lipid lowering therapy based on identification of patients in very high risk categories – for eg Joint British societies coronary risk prediction charts. These tables should be calibrated for the local population Recommended target levels for those on drug treatment – High risk patients should aim for HDL C >38mg/dl and fasting triglycerides, < 2 mmol/litre

Reduce intake of saturated and Trans saturated fat to less than 7-10% of total energy.

Reduce intake of cholesterol to less than 250 mg/ day.

Take more of low glycaemic index carbohydrates, vegetables, fish, pulses, nuts, legumes, dietary fibre, low fat dairy products.

Reduce energy dense foods such as fats and soft drinks, adjust alcohol consumption, while increasing activity and exercise to maintain or lose weight.

Pharmacotherapy is centered on Lipid Lowering drugs like HMG CO a reductase inhibitors (Statins)

Bile acid sequestering resins like Colestyramine, Colestipol.These prevent resorption of bile acids thus increasing de novo bile acid synthesis from cholesterol. The resultant Cholesterol depletion up regulates the LDL receptor activity.[1,2,3,4,5]

Cholesterol absorption inhibitors like EZETIMIBE. They inhibit intestinal mucosal transporter NPC 1L 1 that absorbs dietary and biliary Cholesterol. This depletion of Hepatic Cholesterol UP regulates LDL receptor activity and the standard dose is 10mg/day.[1,2,3,4,5]

Nicotinic acid reduces peripheral fatty acid release with Cholesterol and Triglycerides.[1,2,3,4]

Statins inhibit cholesterol synthesis and so up regulate the LDL receptor activity. They show a clear evidence of protection against total and coronary mortality, stroke and Cardiovascular events in high risk patients. They are generally well tolerated and serious side effects are rare though they may cause LFT abnormalities, Myalgias and Asymptomatic increase in Creatine Kinase.[1]

Where drug therapy and lifestyle does not work Liver transplant may decrease plasma LDL but there is the risk of immunosuppression.[2,3]

Selective LDL apheresis works but is out of reach to the common person as of now.[4,5]

Gene therapy experiments are going on to deliver the complete receptor genomic locus to cells in vitro to regulate LDL gene receptor expression.[1,5]

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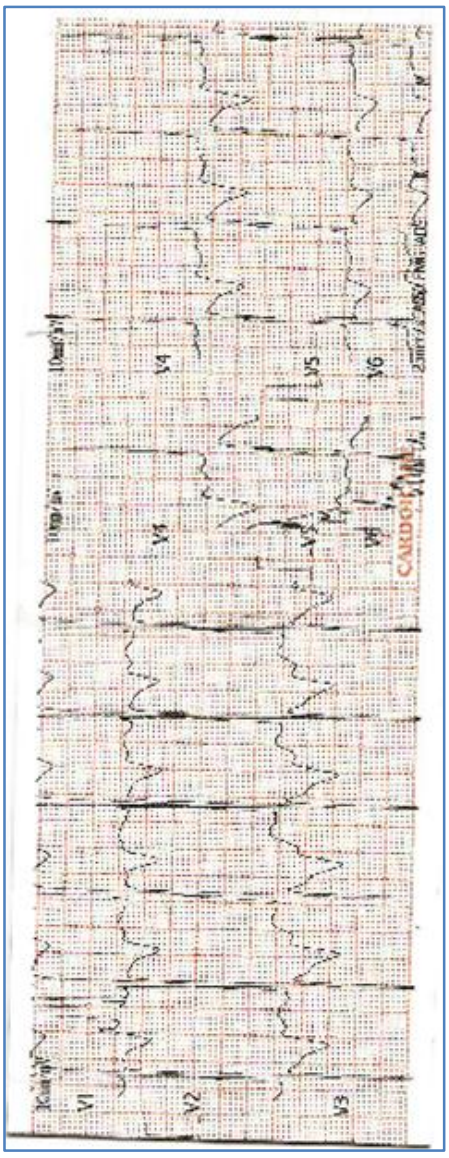
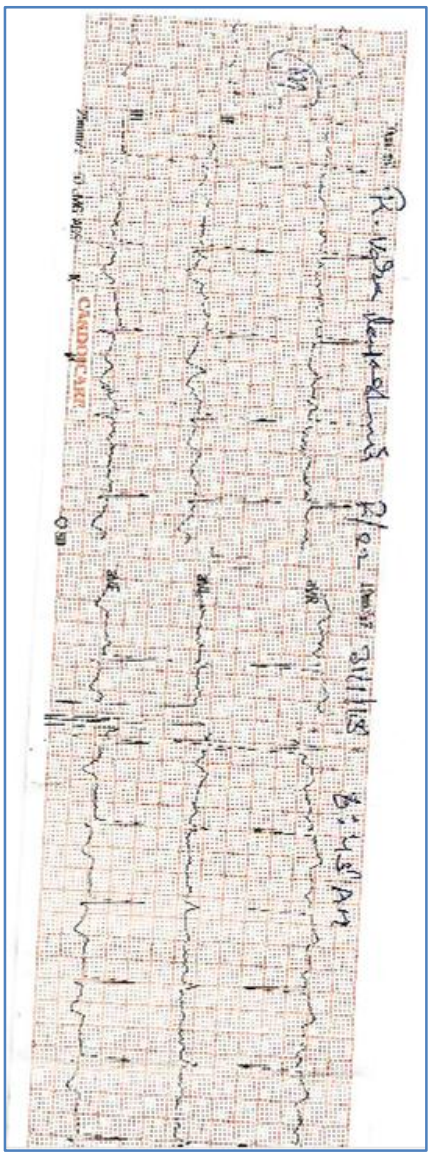


Graph 1



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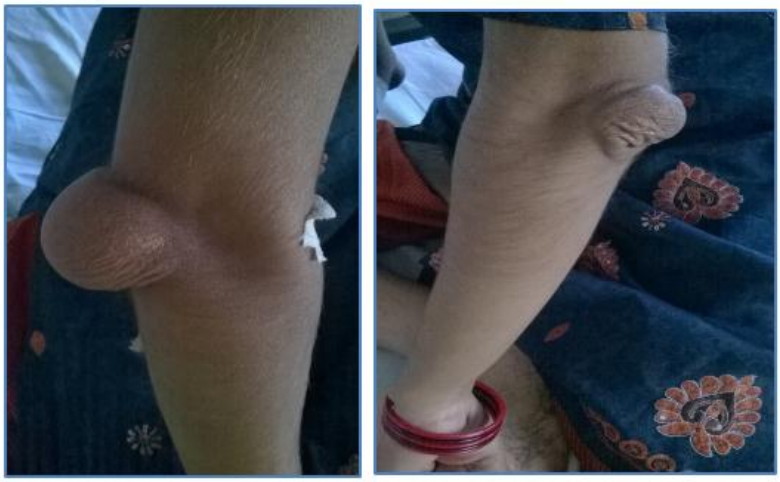

Fig. 1 & 2: Tendon xanthoma

Fig. 3: Tendon xanthoma

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Date of Submission: 27/03/2015.
Date of Peer Review: 28/03/2015.
Date of Acceptance: 07/04/2015.
Date of Publishing: 13/04/2015.