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RESEARCH ARTICLE

HIGH FACTOR VIII AND CORONARY ARTERY DISEASE A CASE REPORT.

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Abstract

High factor VIII is the risk factor for venous thrombosis and coronary artery disease. Here we are presenting a case of coronary artery disease with high Factor VIII. Thrombophilia screening does not include screening of factor VIII. By this study we come to the conclusion that high factor VIII is one of the risk factor of thrombosis and should be considered as one of the test for screening patients with thrombosis.

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Introduction:-

Factor VIII is a large glycoprotein cofactor (320 kilodaltons) that is produced mainly in hepatocytes, but also to some extent by liver macrophages, megakaryocytes, and endothelial cells.^{6,10} Factor VIII circulates in the plasma bound to von Willebrand factor (vWF) at a concentration of approximately 0.1 mg/mL.¹ The plasma half-life of factor VIII is short at about 8 to 10 hours.¹ Factor VIII deficiency should be suspected when a patient with excessive bleeding has a normal prothrombin time (PT) and an extended activated partial thromboplastin time (aPTT).

Factor VIII levels are elevated at birth and increase during pregnancy.² Factor VIII is an acute phase reactant with levels that rise during periods of acute stress, following surgery, and in inflammatory conditions.² Levels can also increase as the result of strenuous exercise or the administration of several drugs including epinephrine, DDAVP, or estrogen (for birth control or hormone replacement therapy). Factor VIII levels can be elevated in a number of clinical conditions including carcinoma, leukemia, liver disease, renal disease, hemolytic anemia, diabetes mellitus, deep vein thrombosis, and myocardial infarction.²

Persistent elevation of factor VIII above 150% is associated with an increased risk for venous thrombosis of more than fivefold.^{1,3} Elevated factor VIII is also associated with an increased risk for recurrence of venous thromboembolism. Risk is graded such that the higher the factor VIII activity, the higher the risk.⁴ The basis for this increased risk is not well understood as genetic studies of the factor VIII and von Willebrand factor genes failed to identify a genetic basis for this increased risk.¹ Values >150% are observed in 20% to 25% of individuals with venous thrombosis or thromboembolism in the absence of other known causes of factor VIII elevation.³

Case History

Here is the 78year old saudi male patient, non smoker presented with left sided chest pain in the early morning while he was doing some physical activity. The chest pain was stabbing in nature associated with sweating and numbness in both forearms. Minimal shortness of breath. Initial work up was done in ER and diagnosed as acute anterior ST elevation MI and he was shifted to Cath lab, where he developed ventricular tachycardia which was treated by DC shock. He is the known case of DM and HTN. He also complains of bleeding tendency. There is history of GI

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malignancy in family. On examination he had muffled heart sounds. And was diagnosed as Acute Coronary Syndrome. Coronary angio done shows total ostial LAD thrombotic occlusion.

His lab investigations showed Normal PT and APTT with high Factor VIII levels.

The results are as follows:

| Tests | Results | Reference value |
|-------------|----------|-----------------|
| PT | 16.4secs | 11.0-15.0secs |
| APTT | 29.5secs | 26.0-40.0secs |
| INR | 1.21 | |
| Factor VIII | 424% | 60-150% |
| Factor VII | 61% | 55-170% |
| Factor IX | 123% | 60-150% |

Renal and liver function test

| Tests | Results | Reference value |
|---------------------|---------|------------------|
| A. BUN | B. 7.7 | C. 2.5-6.4Mmol/l |
| D. S.Creatinine | E. 104 | F. 62-115umol/l |
| G. Total bilirubin | H. 19 | I. 3-17umol/l |
| J. Direct bilirubin | K. 5.5 | L. 0-3umol/l |

Cardiac markers

| Tests | Results | Reference value |
|----------|---------|-----------------|
| M. LDH | N. 542 | O. 85-227 U/L |
| P. CK | Q. 119 | R. 39-308 U/L |
| S. CK-MB | T. 32 | U. 7-27 U/L |

The patient was treated by thrombus aspiration and percutaneous insertion of 1 transluminal stent into single coronary artery.

Conclusion:-

By this study we can conclude that, elevated FVIII levels do not simply reflect a post-thrombotic acute phase response; these rather play an important role in the pathogenesis of the thrombosis. And high factor VIII levels are an independent risk factor for thrombosis, having a greater impact on venous than on arterial thrombosis. Various genetic and environmental factors have been described known to alter FVIII levels, most noteworthy being the ABO blood group and vWF levels from the first category while old age, blacks, and women in the second category.

Studies have shown that FVIII levels appear to be associated with both occurrence as well as outcome of ischemic stroke such that asymptomatic adults with elevated FVIII levels have twice the risk of ischemic stroke as compared to those with normal or low FVIII⁵. However, despite not yet universally recommended as part of routine thrombophilia screening, its potential role in prognostication and association with arterial thrombotic events is undeniable.

By this study we come to the conclusion that, it is worth examining the coagulation system, including FVIII concentration, the abnormality of which may play a significant part in arterial thrombosis. More research is needed to determine the relationship between abnormal FVIII activity and coronary artery disease.

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