



LAB #: B\$\$\$\$\$!\$\$\$\$!\$
PATIENT: GUa d`YDUjYbh
ID: D5 H-9 BH!G!\$\$\$\$%
SEX: Female
AGE: 35

CLIENT #: %& ()
DOCTOR:
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' +)) `]bc]g'5 j Y"
GH"7 \ U`YgZ=@* \$%+(

Oxidized LDL; plasma

RESULTS

| | Within | Outside | Reference Range | |
|--------------|---|---|-----------------|-----|
| Oxidized LDL | | 46 | < 45 | U/L |

INFORMATION

This test measures plasma levels of oxidized low density lipoproteins (Ox-LDL) using a highly sensitive and specific immunoassay. Oxidized low density lipoproteins are directly involved in the initiation and progression of atherosclerotic lesions in coronary arteries that can result in atherosclerotic coronary artery disease (CAD). Plasma levels of Ox-LDL are a sensitive biomarker of atherosclerosis. Elevated levels of Ox-LDL are associated with accelerated atherogenesis, CAD, acute myocardial infarction and stable and unstable angina. Importantly, the levels of total cholesterol are not necessarily higher than normal in patients with unstable CAD. Elevated Ox-LDL has also been associated with metabolic syndrome, impaired glucose tolerance/insulin resistance and untreated overt hypothyroidism.

Low density lipoproteins (LDL), the major carrier of circulating cholesterol, are very susceptible to oxidation of the constituent apoB-100 protein moiety by prooxidants such as metal ions, reactive oxygen radicals, oxidized macrophages, lipoxygenase and peroxynitrite. When the LDL protein is oxidized it becomes antigenic and the Ox-LDL are taken up excessively by the unregulated "scavenger" or "Ox-LDL receptors" on monocyte-derived macrophages. Native LDL (un-oxidized) are not involved in the unregulated uptake process and Ox-LDL is present in macrophages in atherosclerotic lesions but not in normal arteries. Once macrophages breach the arterial endothelial barrier (damaged) the excessive uptake of lipids form Ox-LDL contributes to their entrapment in the sub-endothelial space. The trapped lipid-laden "foam" cells elicit biosynthesis and release of factors by the arterial wall that are pro-inflammatory and chemotactic for other monocytes, perpetuating the atherosclerotic process with injury to the arteries. Injury to the sub-endothelial vessel walls results in decreased production of nitric oxide and elasticity of the arteries and, the damaged lipid-laden arteries eventually narrow restricting the flow of blood.

Increased antioxidant protection and amelioration of oxidative stress would be expected to decrease levels of atherogenic Ox-LDL. These test results should be interpreted in context with the constellation and severity of symptoms/findings and family history. Direct testing for CAD may be warranted if the level of Ox-LDL is undesirable.

References:

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