

# Chlamydia Pneumoniae: Can It Cause Atherosclerosis?

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The Chlamydia species that infect<sup>1-3</sup> humans (*C. trachomatis*, *C. psittaci* and *C. pneumoniae*) are known to cause myocarditis and endocarditis. *C. pneumoniae* (also labelled strain TWAR) can cause coronary artery disease<sup>4-11</sup>. Infections and atherosclerotic heart disease are both common in Pakistan, therefore, this opens new avenues for researchers. Factors which favour the infectious etiology of coronary atherosclerosis are elevated serologic titers and the presence of Chlamydia pneumoniae in mature and immature<sup>12-15</sup> plaques in various age groups. Chlamydia species have been detected in the coronary artery wall of patients with atherosclerosis versus those with other forms of cardiovascular disease. *C. pneumoniae* is also seen in association with symptomatic atherosclerotic disease (79%), but not in plaques formed by other processes like rejection (4%).

Factors that cast doubt are that *C. pneumoniae* is prevalent in about 50%<sup>16</sup> of middle aged subjects without symptomatic coronary artery disease, so the issue of causality has to be ruled out. *C.*

*pneumoniae*<sup>14</sup> has also been detected in the plaques removed from carotid arteries but this does not prove that this is the causative organism.

At the physiologic level, abnormal interaction among endothelial cells, platelets, macrophages and lymphocytes may lead to acute endothelial damage and atheroma formation in blood vessels. It is known that tissue factor is a major inducer of thrombus formation. The blood vessels which are injured, exhibit increased tissue factor procoagulant activity that promotes<sup>17</sup> thrombosis and platelet adhesion at site of injury. This causes development of atherosclerosis. Infection by Chlamydia pneumoniae has been shown to have a similar effect. Detection of *C. pneumoniae* is being done by Immunocytochemistry<sup>18-20</sup> as (ICC) PCR and electron microscopy. Double immunocytochemical staining (ICC) has shown that *C. pneumoniae* antigen is associated with macrophages and smooth muscle cells<sup>21</sup>. Since only 4 urn of the atheroma is represented on a microscopic slide, it is likely that the ICC results are an underestimation rather than an over estimation of the frequency of *C. pneumoniae* organism in atheroma. Treatment of *C. pneumoniae* can be done by tetracycline<sup>14</sup> and macrolide drugs, it is not known whether the organism in atheromata in which there is a low level of replication, could be eradicated by treatment or whether eradication of the organism could have a favourable effect on the lesions. This trial may be considered for patients eligible for surgery. So far only the first of Koch's postulates has been satisfied, the remaining three yet to be proved. There is a strong need to carry out research in this area in the local population, to see whether an infectious agent is also a causative factor for atherosclerotic disease.

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