Thrombolytic Therapy For Prosthetic Heart Valve Thrombosis May Cause Silent Cerebrovascular Ischemic Injury

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**Objectives**

Thrombolysis is considered to be the first line therapy for thrombosis of prosthetic heart valves (PHV). Although major stroke burden related to thrombolysis seems to result from hemorrhage, microembolizations due to fragmentation of thrombus during lyzis may result in silent cerebrovascular ischemic injury (SCII) which is known to be associated with dementia. We aimed to investigate whether thrombolytic therapy causes SCII in pts with PHV thrombosis by detecting changes in serum neuron specific enolase (NSE) levels, a well known marker for cerebrovascular injury.

**Method**

Between 2008 and 2010, 32 consecutive pts (male:8, female:24, mean age: 45.7±12.1 years) who had been detected to have a thrombus on their PHV (mitral:27 and aortic:5) by transesophageal echocardiography (TEE) were included in the study. Venous blood sampling for NSE were obtained before and after the thrombolytic therapy, at the 48th and 72nd hours. Neurologic examinations and TEE evaluations were repeated within 24 hours after the completion of thrombolytic therapy with tPA. Scanning of the cerebrovascular tissue by cranial computerized tomography (CT) was performed just before and 10 days after the therapy.

**Results**

After the thrombolytic therapy, lyzis of thrombus was detected in 30 pts (93.8%). In 2 pts (6.2%) thrombus size did not changed. TIA or stroke were not observed. In 14 pts (%28.6), the NSE levels exceeded the cut-off value of 16.3 ng/ml and were significantly elevated at the 48th (mean:22.0±8.3 ng/ml, p<0.001) and 72nd (mean:22.1±9.2 ng/ml, p<0.001) hours as compared to prethrombolysis (mean:11.3±1.7 ng/ml). In 8 (57.1%) of those pts, regions of microinfarction in the cerebrovascular tissue was detected by cranial CT.

**Conclusion**

Our study suggests that asymptomatic microembolizations to the central nervous system during the thrombolytic therapy are not infrequent. Hence, SCII may be considered as a potential complication of thrombolysis although clinically silent.