profiles (total cholesterol, low density lipoprotein cholesterol, and high density lipoprotein cholesterol), vWF activity, TM, thrombin-antithrombin complex (TAT), soluble intercellular adhesion molecule-1 (sICAM-1), soluble vascular cell adhesion molecule-1 (sVCAM-1), and P-selectin were measured on days 0 and 56. Fisher's exact test was used for statistical analysis. Values of $p < 0.05$ were regarded as significant.

Data before pravastatin treatment were compared between the patient groups and the healthy control group. vWF, TM, TAT, sICAM-1, and sVCAM-1 were significantly increased in the patient groups compared with the healthy controls (fig 1). Before pravastatin administration, no significant difference was found in any measures between the pravastatin group and non-pravastatin group. In the pravastatin group, plasma activity of vWF and TAT on day 56 were significantly reduced compared with those before treatment (table 1). vWF activity in the pravastatin group was significantly lower than in the non-treatment group ($p = 0.024$) on day 56. During the follow up period, low dose pravastatin did not have significant effect on the other measures, including lipid profiles.

vWF is synthesised by endothelial cells and its secretion is triggered by inflammatory/thrombotic mediators, thus establishing it as a marker of endothelial activation/injury. Although TAT is not a generally accepted marker of endothelial injury, it reflects thrombin generation induced by endothelial perturbation. Statins induce an increment in plasminogen activator synthesis and release, a decrease in plasminogen activator inhibitor-1 activity and endothelin-1 expression, and an up regulation of nitric oxide synthase, thus diminishing procoagulant activity and vasoconstriction.^{7,8}

In this pilot study, we have shown that low dose pravastatin reduces plasma vWF activity and TAT without

affecting lipid profiles and has a protective effect against perturbation of endothelial cells, leading to some beneficial effects in the affected patients.

Authors' affiliations

S Furukawa, S Yasuda, O Amengual, T Horita, T Atsumi, T Koike, Department of Medicine II, Hokkaido University Graduate School of Medicine, Sapporo, Japan

Correspondence to: Dr S Furukawa, sinfuru19680804@hotmail.com

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CORRECTION

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Early menopause, low body mass index, and smoking are independent risk factors for developing giant cell arteritis (Larsson K, Mellström D, Nordborg C, Odén A, Nordberg E. *Ann Rheum Dis* 2006;65:529–32.)

We regret that the last author's name was incorrect. It should have been **Nordborg E**.

Additionally, owing to a production problem, which has now been identified, a number of question marks appeared in the abstract between production of the final proof and the printed journal. This has been rectified and readers can now download a corrected copy of the article at <http://ard.bmjournals.com/content/vol65/issue4/>

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