study, we have not investigated this aspect. Yet we have data on body mass index, a main component of the metabolic syndrome together with insulin resistance. A BMI of 30 or more was present in 20.3% of kidney stone formers with hypertension and in 9.4% of subjects without hypertension. This difference is statistically significant ($P < 0.01$), but does not exclude the presence of hypertension in subjects with normal BMI. Furthermore, essential hypertension was present in formers of various types of stone, and not only in uric acid stone formers, that are the object of the hypothesis of Afsar et al. In conclusion, the hypothesis put forward by the authors is interesting but deserves a properly designed study to be confirmed.

Conflict of interest statement. None declared.

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**Relationship between silent brain infarction and chronic kidney disease**

Sir,

We read the article by Kobayashi and colleagues [1] with great interest. The authors report that in addition to age and hypertension, renal function is also independently associated with silent brain infarcts (SBI). However, we wish to request a couple of clarifications.

The first point concerns the relationship between SBI and white matter lesions (WML). SBI and WML are highly correlated, and recent findings suggest that most lacunar strokes are due to widespread abnormalities of the small cerebral arterioles that are responsible for WML and microbleeds [2]. Hence, the lack of data on WML is surprising. Would the authors be willing to add these data (which are usually readily available in the MRI examination)?

The second point relates to the number of SBI. Kobayashi and colleagues showed that a decline in the estimated glomerular filtration rate was associated with not only the prevalence of SBI but also their number. However, no clear data were given. Reporting the number of SBI (mean and range) would improve the reader’s understanding of Kobayashi and colleagues’ results, given that the pathophysiology of lacunar strokes remains heterogeneous [3].

Conflict of interest statement. None declared.

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**Reply**

Sir,

We thank Dr Bugnicourt for his comments and for the interest shown in our study [1].

Silent brain infarction (SBI) and white matter lesions (WML) are different forms of cerebral small vessel diseases (SVD): neuropathological findings corresponding to SBI are thickening and hyaline deposition of the small perforating end arterioles supplying the white matter [2]; on the other hand, those of WML are neuronal loss, ischaemic demyelination and gliosis [3,4]. These cerebral SVD are risk factors for stroke and dementia. For the kidney, the renal SVD characterized by glomerular endothelium dysfunction and lipohyalinosis also play an important role in progressive renal disease [5].

There are haemodynamic similarities between the vascular beds of the kidney and the brain [6]. Thus, we speculated that information about vascular disease in one organ informed us about vascular disease in the other, and reported that there was an independent association between SBI and chronic kidney disease (CKD) in our previous study [1].

As the author of the letter mentioned, we also think that it is interesting to clear the association between WML and CKD to confirm the relationship between cerebral SVD and CKD. Therefore, we re-analysed the WML findings in the same study population.

We defined WML as focal lesions in cerebral white matter that were visible on both T2 and fluid-attenuated inversion recovery and not visible on axial T1-weighted images. WML were graded according to the Fazekas scale into absent (grade 0), punctuate (grade 1), early confluent (grade 2) and confluent (grade 3) abnormalities [4].