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 associ-
ated 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 mi-
crobleeds [2]. Hence, the lack of data on WML is surpris-
ing. 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 understand-
ing 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 dis-

desa (SVD): neuropathological findings corresponding to
SBI are thickening and hyaline deposition of the small per-
forating 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 dys-
function and lipohyalinosis also play an important role in
progressive renal disease [5].

There are haemodynamic similarities between the vascu-
lar beds of the kidney and the brain [6]. Thus, we speculated
that information about vascular disease in one organ in-
formed 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 mat-
ter that were visible on both T2 and fluid-attenuated inver-
sion recovery and not visible on axial T1-weighted images.
WML were graded according to the Fazekas scale into ab-
sent (grade 0), punctuate (grade 1), early confluent (grade
2) and confluent (grade 3) abnormalities [4].