day. This is a main topic, which deserves all of our thoughts. In this context, attempting to develop a quality clinical study can be a heroic task. I also commented that in many hospitals, we now have the ideal tools for supporting research, such as research institutes and foundations, but it is necessary to instil in them the spirit of intellectual curiosity that is the basis of research. On this topic, it is necessary that doctors struggle in order for those bodies to truly be effective at facilitating and promoting quality investigation, and for them to not be contaminated by the unfortunate schemas that are so common in hospital management. I know that there are still hospital research foundations that develop a model activity by diagnosing problems within the centre and providing real assistance to research groups. And these foundations and institutes should also serve to fuse basic and clinical research: both Rodríguez-Puyol and Cruzado insist on the need for including both types of research together. I agree completely, and I believe that nowhere in my editorial did I state the contrary. But we must take into account, as I stated above, the particular problems that prospective clinical treatment trials suffer from, which require a specific solution.

And lastly, referring to the dejection that my friend José María Cruzado detects in me, this is not the case; the fact that I launch diatribes like this editorial is proof to the contrary. Nevertheless, although the situation is somewhat better than it was a few years ago, we must go on fighting. Furthermore, as I stated in the editorial, one of the purposes of the same was to stimulate debate on hospital research. I feel that my letter has indeed sparked debate, and therefore, I am satisfied.

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Comment on "a discussion on quality"

Nefrología 2009;29**(3)**:272-273.

Dear Editor:

In a recent letter titled "A discussion on quality"[1] the author states that "in order to demonstrate the virtues of the quality indicators, some of the articles use very weak baseline data".[2,3] We feel that this hypothesis could easily be refuted with objective data. We will compare variables from the clinical results of the observational study titled *Dialysis Outcomes and Practice Pattern Study* (DOPPS),[4] which included 575 patients from 20 different centres in Spain, with the baseline results of our study (313 patients from four centres)[2]: mean haemoglobin 10.8 *vs*. 11.7 ± 1.4g/dl, phosphorus 5.5 *vs* 5.3 ± 1.6mg/dl, Kt/Vsp 1.31 *vs*. 1.37 ± 0.29, ferritin 288 *vs*. 370 ± 290mg/ml and percentage of autologous arteriovenous fistulas 81 *vs*. 79.9 (DOPPS *vs*. our own study)[2] (the standard deviation for the DOPPS study is not mentioned because it does not appear in the publication). After seeing the results from both studies, we can state that variables from the clinical results of the DOPPS study could be considered worse than, or at best similar to, those presented by the patients in our study. The conclusion that we reach is not different when we analyse the European population (excluding Spain), which is also represented in the DOPPS study. The comparison with the study carried out by Plantinga et al. is more complex due to the form in which the results are expressed, but in general, although these results are worse than the Spanish and European results, they are similar to those from the rest of the population of the United States. Comparisons of variables from clinical results in centres should be carried out with representative samples from the general population, and not with samples representing select centres. The author does not mention what studies the cited studies are compared with. As Fink et al. describe, the variability of results from centre to centre is well-demonstrated (they call this phenomenon the "centre effect").[5] We heartily agree with the other statements expressed in the letter. Meanwhile, we confirm the limitations of our study (which were not mentioned by the writer of the letter) which were listed in the original publication.

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Acute renal failure after intake of mushrooms: the orellanus syndrome

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Dear Editor:
It was with great interest that we read the article by Gallego et al.[1] describing a patient who presented a profile of severe gastroenteritis 12 hours after having consumed wild mushrooms, which was followed by acute renal failure and severe hepatic cytolysis. The patient improved with treatment, but on the seventh day another episode of kidney failure occurred which did not require kidney replacement therapy. This progression, which is ambiguously named "mixed syndrome" together with the opinion of an expert mycologist, who is neither named as a co-author nor listed in the acknowledgements, led the authors to suspect ingestion of both *Amanita phalloides* and *Cortinarius orellanus;*[1] the latter contains the orellanine toxin that gives its name to orellanus syndrome.

In our opinion, there was no orellanus syndrome. Rather, the condition was more likely **intoxication with hepatotoxic mushrooms** of the *Amanita* or *Lepiota* genus; these contain amatoxins, which in a third of all cases cause secondary renal failure between the fifth and tenth day following ingestion.[2] In a series of 77 appraisable cases, Piqueras[2] detected secondary nephropathy in 28, of which 27 also presented renal failure at the onset, as with the case in question.[1] This could fundamentally be due to initial hypovolaemia with renal hypoperfusion, and in some cases to persistent diarrhoea after the improvement of the hepatic analysis which permitted discharging the patient from the ICU and discontinuing intensive fluid therapy. All of the above stresses the importance of replacing the liquids that are lost during the gastrointestinal phase, during both the first days and in later days.[3]

We feel that the possibility of *Cortinarius orellanus* being involved in this case is remote, since intoxication from mushrooms containing orellanine has been observed in Northern and Eastern Europe, and is hardly known in the Mediterranean region.[4]

The review by Saviuc et al.[4] of 245 cases of orellanus syndrome showed a mean delay of 8.5 days before acute renal failure, with 50% developing chronic kidney disease. The worst prognosis was presented by those with a prior kidney disease and early appearance of renal failure. However, the case described by Gallego et al.[1] progressed favourably with no need for kidney replacement therapy, despite being a relatively early presentation of acute renal failure for orellanus syndrome.

This leads us to question the botanical identification of these mushrooms, a week after they were eaten. We suspect that it was done according to a description and/or photographs of the mushrooms provided by the patient or the gatherer. This method has been shown to be unreliable for identifying fungal species[5] if we compare it with studying the fresh material and then analysing it with an optical microscope.

We conclude with the reminder that continuous nasogastric suction alternated with activated carbon, together with sustained intensive diuresis, are the fundamental pillars for initial treatment of intoxication from hepatotoxic mushrooms.

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