switched for a short period of time to deferiprone, and then deferiprone was discontinued. While apparently not on any chelation therapy, the patient developed a cardiomyopathy and then, while still not on any chelation therapy, progressed to heart failure. Despite his poor cardiac status, he underwent a bone marrow transplant and the patient died in heart failure. The authors speculate that the event might have been induced by deferiprone. While it is not possible to categorically exclude deferiprone, at least in the differential, the evidence seems to strongly suggest that iron-induced cardiomyopathy, precipitated by a period without chelation therapy and bone marrow transplantation were the cause. We are concerned that the authors’ assessment of causality was inadequately documented and that their opinion might compromise treatment of patients with iron-induced cardiac dysfunction despite the published evidence of the benefits of deferiprone alone or in combination with deferoxamine in reversing cardiac dysfunction [5]. Indeed there are at least three reports on the complete reversal of cardiomyopathy in patients previously on deferoxamine alone with intensive combination therapy with deferoxamine and deferiprone [6—8]. There are also some promising reports that deferasirox may reduce cardiac iron and may help prevent death from cardiac failure although its efficacy in reversing cardiac dysfunction must be demonstrated on the long term [9].

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Conflict of interests


References


Fatal congestive heart failure with deferiprone: Answer to letter from Ladis et al.

Insuffisance cardiaque congestive fatale lors d’un traitement par déféripnone : réponse à la lettre de Ladis et al.

We would like to thank Dr Ladis and his colleagues for their interesting comments about our recently published case report.

We fully agree with their comments about underlying cardiac iron overload, which may have been the cause of preexistent asymptomatic cardiac disease. Although we did not perform cardiac magnetic resonance imaging, this was highly probable, due to the amount of iron transfused [1]. It is also clear that the bone marrow transplant was an aggravating factor and played a major role in the fatal outcome in this patient.

The key point is that 15 days following the administration of a 6 week course of deferiprone dramatic cardiac failure occurred. We searched for a factor explaining this sudden failure (the patient had been completely asymptomatic until then) but could not find one. It should be noted that the cardiac decompensation was preceded by severe arthralgia. Also, this event was strikingly similar to the features reported after vitamin C treatment in highly overloaded hemochromatosis and attributed to redistribution of iron with release of non transferrin bound iron which is a toxic form of iron [2]. Therefore, the hypothesis that deferiprone might have been responsible for a rise in NTBI seems logical, which could play a role both in the arthro-

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pathy and in the cardiac decompensation resulting in the patient being admitted to the intensive care unit two weeks after the cessation of treatment. Ladis et al. think that the responsibility of deferiprone should be ruled out because clinical signs of cardiac failure only occurred two weeks after withdrawal of the drug but in fact the presenting signs were those of acute pulmonary oedema suggesting that worsening of cardiac function had occurred earlier.

As we mentioned in our conclusion, although the temporal relationship suggested that deferiprone was responsible, no definite conclusion can be established from a single case report. We think the information that Ladis et al. did not observe any cardiac complications with deferiprone is important, suggesting that it could be an exceptional event. However, when searching for similar existing cases in the literature, we found a case reported by Agarwal et al. of a patient who died four weeks after withdrawal of deferiprone due to intolerable arthropathy [3].

In conclusion, we considered this case to be important to inform clinicians of a potentially harmful complication.

References

