liver enzyme levels and the lipid profile that was sustained after discontinuing treatment with statins.

Lysosomal acid lipase deficiency is due to a reduction or complete absence of enzymatic activity due to mutations in the *LIPA* gene.[4] This lack of activity results in the intralysosomal accumulation of cholesteryl esters and triglycerides, leading to microvesicular steatosis, progressive liver disease and dyslipidaemia associated with rapidly progressing atherosclerosis, which are the characteristic features of this disease. A lack of clinical suspicion may lead to misdiagnosis, delaying initiation of appropriate treatment and follow-up. Generally speaking, the possibility of LAL deficiency should be contemplated in patients with unexplained persistent abnormalities in liver function tests or the lipid profile.[1–3]

In the first case, the diagnosis was reached through the measurement of LAL activity in fibroblasts through obtention of a skin biopsy. Advances in diagnostic techniques that have made it possible to study enzymatic activity in dry blood possible allows the assessment of this disease with considerably less invasive and more accessible methods, as demonstrated by the second case. If enzymatic activity is found to be nearly undetectable, genetic analysis of the *LIPA* gene should be considered with the purpose of identifying the causative mutation and confirm the diagnosis.[1,4]

Traditionally, the management of LAL deficiency was based on the control of dyslipidaemia through a low-fat diet and lipid-lowering drugs.[1,2] However, the use of lipid-lowering drugs is not always effective and does not correct the enzymatic defect or prevent lipid accumulation.[1,3] In 2015, the use of sebelipase alfa (a recombinant human lysosomal acid lipase) was approved in Europe for enzyme replacement therapy. This drug is used with the aim of restoring a LAL activity nearing physiological levels through the administration of exogenous enzyme, thus halting disease progression.[1,2,5] The fact that enzyme replacement therapy is now available makes awareness of LAL deficiency in health care professionals all the more relevant for the purpose of early diagnosis and treatment of the disease.

References

1. Kohli R, Ratziu V, Fiel MI, Waldmann E, Wilson DP, Balwani M. Initial assessment and ongoing monitoring of lysosomal acid lipase deficiency in children and adults: consensus recommendations from an international collaborative working group. Mol Genet Metab. 2019;129:59–66.
2. Camarena C, Aldamiz-Echevarria LJ, Polo B, Barba MA, García I, Cebolla JJ, et al. Actualización en deficiencia de lipasa ácida lisosomal: diagnóstico, tratamiento y seguimiento de los pacientes. Med Clin (Barc). 2017;148:429.e1–10.
3. Strebinger G, Müller E, Feldman A, Aigner E. Lysosomal acid lipase deficiency – early diagnosis is the key. Hepat Med. 2019;11:79–88.
4. Carter A, Brackley SM, Gao J, Mann JP. The global prevalence and genetic spectrum of lysosomal acid lipase deficiency: a rare condition that mimics NAFLD. J Hepatol. 2019;70:142–50.
5. Erwin AL. The role of sebelipase alfa in the treatment of lysosomal acid lipase deficiency. Therap Adv Gastroenterol. 2017;10:553–62.

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Suicide attempt with selective serotonin reuptake inhibitor antidepressants*

Intento autolítico con antidepresivos inhibidores selectivos de la recaptación de serotonina

Dear Editor:

Intentional drug poisoning with suicidal intent is an important cause of morbidity and mortality in adolescents.[1]

The drugs involved most frequently in these cases are paracetamol, ibuprofen and, third in frequency, selective serotonin reuptake inhibitors (SSRIs).[2,3] The frequency of the latter has been increasing in recent decades because these drugs are used for first-line treatment of major depression and anxiety disorders in this age group.[4]

The aim of our study was to assess the clinical manifestations, laboratory findings and electrocardiographic features associated with intentional ingestion of SSRIs with suicidal intent in adolescents.

We conducted a single-centre retrospective observational study in patients aged less than 18 years managed between February 2013 and May 2018 in the paediatric emergency department (PED) of a tertiary care hospital that is a reference centre for psychiatric disorders with a discharge diagnosis of ''intentional drug poisoning'' or ''suicide attempt''. We selected patients in who the main drug ingested in the episode was a SSRI (Table 1). We defined

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Table 1 Type of SSRI used.

SSRI	*n* (%)	Number of patients that ingested a dose exceeding the toxic threshold[a]
Fluoxetine	14 (58.3%)	13
Sertraline	4 (16.6%)	2
Escitalopram	4 (16.6%)	3
Paroxetine	1 (4%)	0
Trazodone	1 (4%)	1

SSRI, selective serotonin reuptake inhibitor.
[a] The toxic dose for each drug was defined based on the data collected by the Toxicology Data Network: fluoxetine > 100 mg; paroxetine > 800 mg (for patients aged more than 12 years), sertraline > 250 mg; escitalopram > 50 mg; trazodone > 20 mg/kg.

Table 2 Neurologic signs and symptoms.

Neurologic signs and symptoms	*n* (%)
Decreased consciousness	9 (37.5%)
Glasgow ≤ 8 points	1 (4.1%)
Glasgow 9–13 points	4 (16.6%)
Glasgow 14 points	4 (16.6%)
Delirium	2 (8.3%)
Agitation	1 (4.1%)
Ataxia	1 (4.1%)
Headache	1 (4.1%)

intentional drug poisoning as ingestion of a drug in amounts exceeding the maximum dose established in the marketing authorisation with suicidal intent. The statistical analysis consisted of parametric tests performed with the software Stata version 15.

In the period under study, the PED received 306 583 visits. Twenty-four patients were treated for intentional ingestion of SSRI, all of them female, with a median age of 15.7 years (interquartile range [IQR], 14.4–16.7). Twenty-three (95.8%) had an underlying mental health disorder. The median time elapsed from ingestion of the drug to assessment at the PED was 1.5 h (IQR, 1–2), and 13 (54.2%) were deemed stable in the initial paediatric assessment triangle (PAT); 10 (41.7%) presented with neurologic impairment and 1 had compensated shock (4.1%). All patients with neurologic impairment had ingested not only SSRIs, but also benzodiazepine or antipsychotic drugs. The most frequent symptoms were neurologic (Table 2), followed by vomiting (4; 16.6%). The neurologic symptoms had resolved spontaneously by a median of 8 h post ingestion (IQR, 6.5–18), and all patients maintained a patent airway and spontaneous breathing at all times without haemodynamic or respiratory abnormalities.

Sixteen (66.7%) patients underwent urgent gastrointestinal decontamination. We did not find clinically relevant abnormalities in any of these patients in the electrocardiogram or the serial laboratory tests performed (blood gases, kidney and liver enzymes, creatinine phosphokinase), so no additional treatment was required. All patients were admitted to hospital: 11 to the intensive care unit (ICU) for continuous monitoring, and 13 in the inpatient ward, and none required respiratory or haemodynamic support. The median length of stay was 8 days (IQR, 4–20).

In conclusion, the patients that received care in our hospital for ingestion of SSRIs with suicidal intent did not exhibit any relevant electrocardiographic or blood test abnormalities. Larger studies are required to confirm these findings.

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References

1. Mintegi S, Grupo de Trabajo de Intoxicaciones de la Sociedad Española de Urgencias de Pediatría. Manual de intoxicaciones en pediatría. 3.ª ed. Madrid: Ergon; 2012.
2. Shreidan DC, Hendrickson RG, Lin AL, Fu R, Horowitz BZ. Adolescent suicidal ingestion: national trends over a decade. Adoles Health. 2017;60:191–5.
3. Tyrrel EG, Kendrick D, Sayal K, Orton E. Poisoning substances taken by young people: a population-based cohort study. R J Gen Pract. 2018;68:e703–10.
4. Moller HJ. Evidence for beneficial effects of antidepressants on suicidality in depressive patients: a systematic review. Eur Arch Psychiatry Clin Neurosci. 2006;25:329–43.

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