deaths recorded between 2010 and 2015, 55% corresponded to ARD associated to allogenic blood transfusion: 39 cases of TACO, 9 cases of TRALI and three cases of TRD.

In view of the above, and considering the clinical importance, healthcare impact and therapeutic relevance involved, we consider it essential to contemplate episodes of ARD associated to blood transfusion in this document. Likewise, we wish to underscore the obligation to report all cases to the hematological surveillance system, and the fact that ''one unit may be enough''.

Conflicts of interest

There are no conflicts of interest in relation to this study.

Dr. García-Erce has given talks and has acted as moderator at congresses and sessions, with grants or funding from Vifor-España, Sandoz, Amgen, Alexion, Braun, GSK, Octapharma, Novartis and Sanofi.

Acknowledgements

Thanks are due to all those transfusion centers that supply the best and freshest blood components.

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2173-5727/

In reply to ''Acute respiratory distress secondary to blood transfusion''

En respuesta a «Distrés respiratorio agudo secundario a la transfusión sanguínea»

Dear Editor,

We would like to thank the authors of the letter entitled ''Acute respiratory distress secondary to blood transfusion'' for their interest in our article.[1] We agree with them that blood derivatives transfusion (whole blood cells, red cells, apheresis platelets, fresh frozen plasma, cryoprecipitates, stem cell products and endovenous inmunoglobulins) are well recognized, but infrequent, risk factors for Acute respiratory distress syndrome (ARDS).[2] In the original manuscript,[1] blood derivatives products and other ARDS risk factors are not mentioned because it is focused on what is required to define a ''disease'' and the relation between ARDS and diffuse alveolar damage (DAD). From our point of view, the effect of each risk factor in ARDS susceptibility or outcome should be clarified after the ARDS has been agreed upon as a disease (see below).

Currently, given that it has been demonstrated that only half of ARDS patients present DAD[1,3] – which is considered the histological ARDS hallmark[4] – as well as the effect of DAD in the ARDS outcome,[3,5] we consider that including DAD as an ARDS diagnosis criteria would increase the accuracy of the definition.[1] If this proposal is accepted, the ARDS with DAD should be considered the real disease and the others (ARDS without DAD) as a misdiagnosis or mimic.[6] This new interpretation determines that old paradigms and approaches should be changed.

In reference to the transfusion related acute lung injury (TRALI), firstly, its association with DAD is not well demonstrated, as because studies with pathological analysis are scarce and biased, due to the short time between the blood product administration and the deceased.[7] Secondly, accepting that TRALI is risk factor for ARDS with DAD, it is unknown if the DAD induced by blood transfusion is similar to those induced by other risk factors. This fact could be of paramount importance because ARDS with DAD is a complex entity with several different physio-pathological pathways. For that reason, different subtypes of DAD would be recognized according to the processes which predominate (e.g. apoptosis, tight junction dysfunctions or alveolar clearance impairment). Finally, all of these pathological findings should be settled in a specific clinical context, in which

differential clinical diagnosis, like transfusion associated circulatory overload (TACO), have been ruled out.

As a conclusion, we recognize that blood transfusion derivatives can trigger episodes of severe respiratory insufficiency, but their relation to ARDS with DAD is still unknown. It is evident that improving the diagnosis accuracy seems to be an initial and basic requirement to enhance the efficacy and effectiveness of future treatment.

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2173-5727/

Aspirin desensitization in patients with coronary artery disease: Cost savings☆

Desensibilización al ácido acetilsalicílico en pacientes con cardiopatía isquémica: ahorro de costes

Dear Sir,

Antiplatelet drugs play a key role in the management of ischemic heart disease and other diseases, exerting their effects though different pathways. The most useful of these drugs in application to coronary disease are the cyclooxygenase inhibitors: acetylsalicylic acid (ASA, aspirin), which is the most widely studied and used substance,[1] and triflusal; and the P2Y12 antagonists: ticlopidine, clopidogrel, prasugrel and ticagrelor.

According to the current ischemic heart disease guides, in allergic patients were ASA is necessary, a rapid desensitization protocol must be applied,[2] involving the administration of increasing doses of the drug until tolerance is achieved. Different rapid desensitization protocols have been described,[3–5] with a duration of 2–5 h, that can be used in unstable patients, with excellent efficacy and safety.

Despite the lack of clinical evidence to the effect (since no studies have suppressed the use of ASA), in patients who are hypersensitive to nonsteroidal antiinflammatory drugs and suffer confirmed chronic ischemic heart disease (detection of coronary atherosclerosis by computed axial tomography or positive ischemia testing), it is common to empirically prescribe triflusal or clopidogrel in monotherapy. In the event of percutaneous coronary intervention with the placement of a stent, even double-dose clopidogrel (or the prescription of prasugrel–ticagrelor) during one year has been used. In patients with acute coronary syndrome, dual antiplatelet treatment with triflusal and a P2Y12 inhibitor has been used on an empirical basis.

From the pharmacoeconomic perspective, ASA desensitization in patients with ischemic heart disease is comparatively less expensive in the context of both monotherapy and dual antiplatelet treatment (Tables 1 and 2).

In monotherapy, the annual cost of clopidogrel or triflusal is respectively 1142.12% (218.13 vs 17.64€) and 662.76% (134.56 vs 17.64€) greater than the cost of ASA. These differences could greatly increase (between 1408.05 and 3778.23%) in the case of treatment during the first 1–6 months with prasugrel (cost between 266.02 and 515.52€) or ticagrelor (cost between 294.12 and 684.12€), followed by clopidogrel, as recommended by some guides.[2]

☆ Please cite this article as: Cubero JL, Simó Sánchez B, Millán P, Colás C. Desensibilización al ácido acetilsalicílico en pacientes con cardiopatía isquémica: ahorro de costes. Med Intensiva. 2017;41:446–447.