Partial splenic embolization in a child with hereditary spherocytosis

M. Jiménez *
C. Azcona *
L. Castro *
J. I. Bilbao #
P. León *
L. Sierrasesúmaga

* Department of Paediatrics
# Department of Radiology

Clínica Universidad de Navarra.
Avda. Pio XII s/n. E-31080.
Pamplona. Navarra. Spain

Partial splenic embolization (PSE) was introduced as an alternative to splenectomy to avoid the risk of post-splenectomy sepsis and to reduce the number of situations in which surgery would be dangerous or not feasible [5]. PSE is an accepted therapy for the treatment of secondary hypersplenism in various disorders, including oesophageal variceal haemorrhage [3], thalassaemia major [5], portal hypertension [1], painful splenomegaly [2], Gaucher disease [9], hypersplenism in cirrhosis [5], and prior to myelosuppressive treatment in hepatocarcinoma and active chronic hepatitis [2, 3].

As an alternative to surgical splenectomy, PSE was performed on a 7-year-old girl with hereditary spherocytosis and hypersplenism who initially presented with a haemolytic crisis; haemoglobin values of 6.8 g/dl, jaundice and splenomegaly. She needed blood transfusions on two occasions due to persistent anaemia, with Hb values between 6.8-9 g/dl. PSE was performed following the Spigos protocol [8].

Peripheral blood cell counts were repeated on days 1, 3, 7 and 15, and then 1, 3, 6, and 12 months after PSE (see Table 1). Abdominal ultrasound 1 month after PSE showed a 17% reduction in spleen size, with embolization of approximately 75% of the spleen.

Maddison [7] performed the first successful splenic arterial embolization in 1973. The procedure was initially associated with significant morbidity and mortality, but subsequent series report substantially improved results [6]. We performed the embolization technique described by Spigos [1, 3, 6, 8] because it has a very low risk of complications, although Israel et al. [5] recently reported a lower rate of complications using polyvinyl alcohol.

The increase in haemoglobin values found in our patient has been observed in patients affected by other diseases who have required PSE [5]. The thrombocytosis observed in our patient during the first 15 days after PSE that returned to normal values over 3 months, has been previously reported [3, 5]. This increase in platelet count appears to be

due to the increase in sequestration in the spleen. There were no modifications in the white cells. The clinical course in our patient was similar to that described by others [3, 6], with moderate fever for 3 days, mild abdominal pain and vomiting (known as the postembolization syndrome) and alleviated by symptomatic treatment [2]. We did not observe any other complications (e.g. pleural effusion, pancreatitis, pneumonia [1]).

We attempted to infarct between 70% and 80% of the splenic parenchyma, as it has been clinically and experimentally demonstrated that a 30% remnant of splenic tissue can provide protection against infection [4].

We suggest PSE to be an acceptable alternative to splenectomy in a child with hereditary spherocytosis and secondary hypersplenism, owing to the simplicity of the technique, the favourable outcome and the absence of complications in our case.

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Table 1. Haemoglobin, platelets, leucocytes and total bilirubin data before and after PSE									
	Before PSE	**1 day**	**3 day**	**7 day**	**15 day**	**1 mo**	**3 mo**	**6 mo**	**12 mo**
Haemoglobin (g/dl)	6.8-8	9.1	9.5	10.1	10.5	12.6	12.8	12.5	12.2
Platelets (10^9/1)	334	374	420	635	902	571	471	450	436
Leucocytes (10^9/1)	7.4	8.0	7.3	7.4	8.1	7.9	7.1	7.2	8.6
Total bilirubin (mg/dl)	3.4			0.5			0.6		0.1