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Postsplenectomy sepsis: the need for lifelong prophylaxis

Dr Evans's report of three cases of late severe postsplenectomy sepsis¹ rightly draws attention to the fact that such sepsis is often rapidly fatal and can occur as long as 45 years after splenectomy.

I feel, however, that the opportunity to discuss the aetiology of this phenomenon was missed. This is of fundamental importance because successful prophylaxis of overwhelming postsplenectomy sepsis depends on rationally based treatment, and it can be argued that Dr Evans's recommendations were inadequate.

Overwhelming postsplenectomy sepsis is almost always associated with bacteria such as *Pneumococcus meningococcus* and haemophilus. These bacteria have a polysaccharide capsule, which evades ingestion by most macrophages and enhances their pathogenicity.² Specialised dendritic macrophages however, have evolved within the marginal zone of the spleen, and these have the ability to take up and present carbohydrate antigens,³ which are commonly of the T cell independent type.

Evidence from studies on animals shows that a specialised subset of B cells, capable of responding to carbohydrate, exists in close proximity to the marginal zone macrophages.⁴ This subset of B cells is non-recirculating and resides in the spleen. Gaps in the adjacent endothelium allow direct contact between these specialised immune cells and the blood. Thus it can be seen that the spleen is optimally developed to police the blood for the presence of dangerous encapsulated pathogens.

This explains why the response to infections with encapsulated bacteria or to vaccination with pneumococcal vaccines is inadequate after splenectomy as the response by lymph node macrophages is insufficient. Unless a splenunculus is present the patient is permanently vulnerable to such encapsulated pathogens.

It is well known that postsplenectomy sepsis can be fatal in as little as 12 hours from the onset of symptoms: medical attention, however well informed, may arrive too late. It is therefore my belief that it is the responsibility of the physician or surgeon who recommends splenectomy to discuss the implications with the patient, explaining that lifelong prophylaxis with penicillin will be needed and that pneumococcal vaccine will be given before the operation, although the vaccine may only be beneficial for five years. The family doctor should also be alerted.

I have found that, provided the risks and benefits of splenectomy are clearly explained, patients comply very carefully with prophylaxis with penicillin. For those allergic to penicillin, alternatives are available.

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Dr Evans replies as follows:

Dr Kay's comments are apposite and mainly true. It was a desire for brevity which restricted my report. I am more cynical than he. He believes, however, that it is the responsibility of the doctor recommending or performing the operation to explain that lifelong penicillin will be needed. This may be true; but I do not believe that patients, in the main, will take

drugs lifelong, or that general practitioners or specialists will prescribe them. These patients all have an abdominal scar: the evidence of splenectomy is there for everyone to see. What is needed is recognition of the risk, not only by doctors but also by the patients themselves.

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Glycosylated haemoglobin

The excellent review by Dr Ian Peacock contains some biochemical information which, in my opinion, is not completely correct.

The author rightly refers to HbA_{1c} as $\alpha_2(\beta\text{-Val-1-deoxyfructose})_2$, but its formation is expressed as:

A non-enzymatic reaction should lead to the production of a monoglycosylated compound and, to a much lesser degree, of a diglycosylated one. HbA_{1c} is really diglycosylated, while the monoglycosylated haemoglobin, when present, is in low concentration.^{2,3} Thus the above scheme is an oversimplification of the reaction and an equilibrium, producing symmetrical haemoglobin, should be postulated:

It is unclear whether it plays an important part within the erythrocyte or it derives from in vitro manipulation of the sample. A similar equilibrium is well known for some haemoglobin variants, such as Hb_S, and generally for haemoglobins which are modified near the NH₂ terminus of the α chain.

For the reasons given above the heterogeneous glycosylation on some late α lysines should provide principally monoglycosylated products. In this case the equilibrium can not be postulated and these haemoglobin components should be collectively indicated as:

These observations are based principally on theoretical considerations and sur-