Letters to the Editor

A patient with recurrent hypothermia associated with thrombocytopenia

Sir, Chan and Beard conducted an important survey of platelet counts in 75 patients admitted with hypothermia. A few additional comments may be of interest.

Hypothermia is associated with haemorrhagic and thrombotic lesions. It is therefore of interest that the platelet and white cell (WCC) counts decreased and the mean platelet volume (MPV) increased in hypothermic dogs. Since this latter phenomenon was apparent within 3 hours, it is unlikely that it is a result of altered thrombopoiesis. The effect on MPV may indicate platelet activation since several platelet aggregating agents increase the MPV. Was the MPV or WCC altered in the patients in Chan and Beard's survey? If they were, these variables could become markers of platelet and white cell activation in hypothermia, even in the absence of thrombocytopenia. These markers would be clinically useful since they are now provided by automated counters. However, it is essential to consider that conditions that can precipitate hypothermia (for example, pneumonia and alcohol ingestion) may also affect the WCC and platelet function.

The benefits of defining, monitoring and possibly modifying the effect of cooling on platelets extends beyond hypothermia because there is evidence that the incidence of vascular disease is related to environmental temperature and to platelet function indices.


We have shown this letter to the authors who reply as follows:

Mikhailidis and Barradas have raised interesting questions with respect to the white cell count and the mean platelet volume and have suggested that these might be important markers of clinical usefulness in such cases. With our patient the lowest recorded white count that we can find in relation to one of these admission episodes was 3.1 × 10^9/l. Over the many years that this patient has been repeatedly admitted to hospital in a hypothermic state, the automated counter in the haematology laboratory has variably recorded the mean platelet volume. We could only identify three such incidences where the readings were 9.2 fl, 11.0 fl and 12.7 fl (normal range 7.6 fl–10.8 fl).

Thus, although these are important points, we do not think the data from our patient really serve to advance the argument that white cell count and mean platelet volume are useful clinical markers in patients with hypothermia and thrombocytopenia.

Hypertension and hypoparathyroidism — narrowed therapeutic safety with nifedipine

Sir, Sublingual nifedipine gives a rapid response and is used to treat severe hypertension. However, it may well produce severe hypotension and consequent myocardial ischaemia particularly in patients with angina pectoris with poor cardiac reserve, volume depletion and autonomic neuropathy, and chronic renal failure. We report a case where severe hypotension occurred in the hypocalcaemic setting of hypoparathyroidism. This deleterious effect of nifedipine was, ultimately, also used for the benefit of the patient.

A 35 year old obese and short female patient presented with sudden onset breathlessness and blood pressure of 200/120 mmHg. With 5 mg sublingual nifedipine, her blood pressure dropped to an unrecordable level within 5 minutes. The electrocardiogram taken revealed a QTc of 0.62 seconds, but was otherwise normal. Blood sample for biochemistry was withdrawn and a 20 ml bolus of 10% calcium gluconate backed up by slow calcium infusion was commenced, leading to normalization of the blood pressure to 130/80 mmHg. Her serum biochemistry revealed total serum calcium of 1.12 mmol/l, phosphorus 2.77 mmol/l and alkaline phosphatase of 96 IU/l. After excluding renal failure and secondary causes of hypertension including phaeochromocytoma a diagnosis of hypoparathyroidism and essential hypertension was made as the most likely explanation of her clinical state and serum biochemistry. Hypoparathyroidism was not characterized further because of the lack of parathyroid hormone assay facility.

References

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D. P. Mikhailidis and M. A. Barradas

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