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 relevant 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.

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References


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 x 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 QT 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.
Subsequently, the patient was put on oral calcium carbonate and vitamin D3 and thiazide diuretics. After one week though repeat serum calcium and phosphorus were 1.87 mmol/l and 1.96 mmol/l, respectively, she relapsed into hypertension of 200/120 mmHg. Attempts to bring down the pressure with a variety of drugs including beta blockers, enalapril and methyldopa did not help significantly. Ultimately, under controlled supervision, with a dose of oral nifedipine 2.5 mg twice a day, she maintained her blood pressure in a normal range.

The inter-relationship between hypertension and calcium is not well defined. Low calcium intake, hypercalcaemia, such as hyperparathyroidism, and hypercalcaemic states such as pseudohypoparathyroidism are also associated with hypertension. The final link postulated is suboptimum calcium in the vascular smooth muscle and high parathyroid hormone. This patient showed marked sensitivity to the nifedipine in the usual dosage in a setting of hypocalcaemia. Such a state may well be existing in many hypertensive states with chronic renal failure, and hypoparathyroidism. Hypocalcaemia may well be the cause behind such occasional, but marked, hypotensive spells noticed with sublingual nifedipine. Use of nifedipine sublingual therapy therefore, may not be advisable in hypertension associated with hypocalcaemia.

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References

Lymphopaenia in elderly in-patients

Sir,

We report data suggesting that lymphopaenia - traditionally defined as <1.5 x 10^9/l - occurs in the majority of ill elderly in-patients. We have prospectively recorded the peripheral blood lymphocyte count (PLC) of 625 consecutive admissions (344 women, 281 men, mean age (total n) = 81 years, range = 61–101 years) from the community to two acute medical geriatric wards at University Hospital, Nottingham, over 7 months. PLC was measured by Sysmex NE8000 haematology autoanalyzer.

The distribution of PLC in these admissions is positively skewed - such that in 72% (450) of in-patients PLC was <1.5 x 10^9/l and in 38% (237) PLC was <1.0 x 10^9/l (Table I). There is no gender difference in the distribution (χ^2 = 0.85, P > 0.5). PLC is significantly negatively correlated with age (Spearman rank correlation coefficient \( p = -0.14 \) (P = 0.006), \( p = -0.10 \) (P = 0.041) for females and males, respectively).

| Table I Ages and peripheral lymphocyte counts of all patients |
|-------------------------|--------------------------|
| PLC (×10^9/l)          | Female | Male |
| Median                 | 1.2    | 1.1  |
| Range                  | 0–10.2 | 0–6.6|
| Skew coefficient       | 5.24   | 3.19 |
| Age (years)            |        |      |
| Mean (± s.d.)          | 82 (±7) | 79 (±6) |
| Range                  | 61–101 | 65–95|

These results can be compared with those from past studies of old people in the community. While conflicting results have been reported in healthy volunteers, several community studies including small numbers of healthy elderly adults have documented a fall in PLC – particularly circulating T cells, with increasing age. Lymphopaenia and anergy in apparently healthy adults are associated with reduced survival at 1 and 3 years. Caird et al. studied 501 healthy old people living at home – in 45% PLC was <1.5 x 10^9/l.

Our findings suggest that lymphopaenia is very common in acutely admitted elderly medical patients and is associated with ageing and disease. Further work on the definition and clinical significance of lymphopaenia in ill elderly adults is warranted.

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