Just another boring anaemia?

Introduction

Normochromic, normocytic anaemia is commonly encountered in clinical practice and may be due to co-existent ‘chronic disease’ or a primary haematological disorder. However, in about one quarter of cases, no underlying cause may be found.

Case History

A 75 year old male presented with lethargy. Past history included treated hypothyroidism (GP diagnosis). Hb was 96g/l (mcv 86fl) with normal WCC and platelets. All investigations were normal (Bone marrow declined by patient). A diagnosis of myelodysplasia was made and he received intermittent blood transfusions. 3 years later, he presented with a visual field loss. MRI demonstrated a pituitary macroadenoma with chiasmal compression. He was referred to an endocrine clinic and was noted to be pale with decreased facial hair and gynaecomastia. Synacthen test was normal, prolactin 772mU/l (<500), consistent with pituitary stalk compression, testosterone 0.4nmol/l (10-30), LH 1.7mU/l (1.3-13) and FSH 3.6 (0.9-15), consistent with hypogonadotrophic hypogonadism. Review of his initial thyroid function tests four years previously demonstrated a low TSH (0.71 mU/l) and free thyroxine of 8 pmol/l (10-27), consistent with secondary hypothyroidism.

The patient underwent trans-sphenoidal resection of the pituitary tumour and received post-operative radiotherapy for residual tumour. Androgen deficiency was corrected by monthly intramuscular injection of testosterone esters (Sustanon 250). The normochromic, normocytic anaemia resolved (Hb139g/l, Hct 0.41, MCV 85fl) and for 2 years he continued to have normal haematological indices with no transfusion requirement. Unfortunately he died of ischaemic heart disease.

Discussion

Erythropoiesis is a hormone dependent phenomenon. Erythropoietin is secreted by the kidney and, to a lesser degree, the liver in response to hypoxia. Androgens increase the sensitivity of erythroid progenitors to erythropoietin, resulting in a larger red cell mass in males, despite similar erythropoietin concentrations. This explains the 10 – 20 % rise in haemoglobin that occurs in males at puberty and the adult male/female differential in haemoglobin concentration.

It is well recognised that exogenous testosterone therapy can result in erythrocytosis and, thus, an increase in haematocrit.

Androgen deficiency may be caused by pituitary disease or primary testicular failure, e.g. following chemotherapy, trauma, or mumps orchitis. Clinical features include azoospermia, loss of libido, lethargy and/or low bone mineral density. Differentiation of primary and secondary androgen deficiency relies on the interpretation of luteinizing hormone, follicle stimulating hormone and testosterone measurements. In the present case, it is hypothesised that androgen deficiency, secondary to a pituitary tumour, caused a mild normochromic, normocytic anaemia. The earlier identification of hypothyroidism with a low TSH concentration (secondary hypothyroidism) should have pointed to the presence of pituitary disease. The anaemia resolved following correction of the androgen deficiency and no further blood transfusions were required.
Testosterone levels should be measured in patients with unexplained normochromic, normocytic anaemia and endocrine referral if indicated.

References
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