nesium subsequently stabilised at the lower end of the reference range (figure), and no further intravenous magnesium replacement was required.

Comment

Calcium and magnesium share a transport system in the gut, and both 1-α-hydroxycholecalciferol and 1,25 dihydroxycholecalciferol have been successfully used in the treatment of hypomagnesaemia associated with the short bowel syndrome.[2] A recent report describes the use of 1-α-cholecalciferol in a patient with the short bowel syndrome in whom, the authors claim, renal tubular absorption of magnesium was increased.[3]

In our case oral magnesium supplements in high doses were associated with diarrhoea, and 1-α-hydroxycholecalciferol enhanced magnesium absorption and possibly reduced urinary losses. The figure shows that normal serum magnesium concentrations were maintained after the administration of 1-α-cholecalciferol, with resolution of symptoms. Two 24 hour collections obtained while the patient was taking 1-α-cholecalciferol contained 5 and 8 mmol magnesium (figure), suggesting that any effect of 1-α-cholecalciferol on renal magnesium conservation was minimal.

The role of vitamin D in the renal handling of magnesium is unclear. Levine *et al* found a diminution of magnesium tubular reabsorption in vitamin D deficient rats given 1,25 dihydroxycholecalciferol,[4] and Burnatowska *et al* showed an increased fractional excretion of magnesium in hamsters from which thyroid and parathyroid glands had been removed.[5] Fukumoto *et al*, however, described a decrease in fractional excretion of magnesium in a hypomagnesaemic patient with the short bowel syndrome who was given large doses of 1-α-cholecalciferol.[3] In their patient, as in our case, the serum concentration of 1,25 dihydroxy-cholecalciferol was abnormally low, and this was felt to contribute to impaired renal resorption of magnesium.

More work is required on the effect of 1-α-cholecalciferol on renal magnesium handling, but the case reported here suggests that this drug may be useful in the management of hypomagnesaemia induced by cyclosporin.

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Parathyroid hormone related peptide in ovarian carcinoma

Hypercalcaemia is one of the commonest paraneoplastic syndromes encountered clinically, being associated with an estimated 10–20% of all solid tumours. Many factors contribute to this syndrome of humoral hypercalcaemia of malignancy (HHM), including cytokines and prostaglandins of the E series.[1] Recently, a peptide structurally and immunologically distinct from parathyroid hormone (PTH), but with parathyroid hormone bioactivity, has been implicated in the pathogenesis of HHM and has been termed parathyroid hormone related peptide or PTHrP.[2]

Hypercalcaemia is associated with ovarian carcinoma frequently enough for the ovary not to be ignored as a primary tumour site in women presenting with clinically unexplainable hypercalcaemia.[3] We therefore felt it would be of interest to examine a number of ovarian carcinomas for the presence of PTHrP.

Immunocytochemistry was performed using an antibody raised against the first 34 amino acids of PTHrP (kindly donated by Drs GV Segre and H Jüppner, Boston, Massachusetts, USA) and standard indirect immunocytochemical techniques. Two cases of ovarian carcinoma associated with hypercalcaemia (one small cell and one non-small cell type, supplied by Dr GR Dickersin, Boston) were found to contain PTHrP. PTHrP was present throughout the cytoplasm of the tumour cells but was absent from inflammatory and stromal cells and areas of tumour necrosis. The immunoreactivity was completely abolished when the antibody was pre-incubated with PTHrP (1–34) overnight. Two cases of serous cystadenocarcinoma and two cases of mucinous cystadenocarcinoma of the ovary (from patients who were normocalcaemic) were found to be negative for PTHrP.

Normal adult ovary does not produce PTHrP, but the peptide has been detected in the human fetal gonad.[4] This is the first report of the presence of PTHrP in a hypercalcaemic ovarian carcinoma. While the pathophysiological role of PTHrP is yet to be elucidated, one possibility is that in addition to it inducing hypercalcaemia it may stimulate the growth of tumours in an autocrine manner.[5] It may also regulate the fetal calcium balance.[4]

The widespread presence of PTHrP in lung, renal cell, and squamous cell carcinomas and its presence in small cell ovarian carcinoma, as reported here, suggests that PTHrP is a common manifestation of the transformed cell.

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Prorenin in ovarian cyst fluid

Aspartic proteinase renin is secreted by the kidneys and, as the initial enzyme in the renin/angiotensin cascade, it is important in the regulation of blood pressure and fluid homeostasis. Recently, high concentrations of the zymogen prorenin have been shown in female reproductive organs.[1] The concentrations of prorenin in follicular fluid collected from women undergoing in vitro fertilisation are at least 10 times higher than those in plasma.[2] Immunohistochemical staining has shown that the prorenin is present in the theca cells lining the follicles from where it is, presumably, secreted into the fluid.[3] As only very low concentrations of active renin are found in the ovary, it has been postulated that ovarian prorenin may be biologically active without any necessity for prior removal of the propart sequence.[1] The role of ovarian prorenin is still unclear but it probably operates through the formation of angiotensin II (AII). Studies with the AII antagonist, saralasin, have indicated a direct role for AII in ovulation,[4] and, in addition to its effects on steroidogenesis, the vasoconstrictor and angiogenic properties may be important for follicular growth.

Cysts are commonly derived from the ovarian follicles and frequently contain a large volume of liquid. It was thus considered of interest to determine whether this fluid might also contain a high concentration of prorenin. The fluid from six ovarian cysts removed at laparotomy was collected, stored at −20°C, and assayed for prorenin by the trypsin-activation method of McIntyre *et al*.[5] Renin activity was estimated by measuring the rate of angiotensin I (AI) production from human angiotensinogen.[6]

Prorenin was detected in the fluid from four of the cysts assayed—three of follicular origin and one from a mucinous cystadenoma—with two others being virtually negative (table). The concentration of active renin detected in all of the fluids was very low (less than 5% of total) and may have been due to partial activation of the prorenin during collection. This predominance of prorenin over renin is in keeping with previous results for normal ovarian follicular fluid.[1,2]

Concentrations of prorenin in ovarian cyst fluid

Age	*Diagnosis*	*Prorenin (ng AI/ml/h)*
18	Follicular cyst	41
29	Follicular cyst	9
42	Follicular cyst	24
44	Mucinous cystadenoma	15
32	Serous cystadenofibroma (16 weeks pregnant)	1
83	Serous cystadenofibroma	0

The concentration of prorenin quantified in the ovarian cysts was, however, comparable with the range of values reported for normal plasma (10–40 ng AI ml^{-1} h^{-1}).[2 3]

These data show the presence of prorenin in some ovarian cysts. The concentration of prorenin, however, was not sufficiently high to consider fluid from these cysts as a useful source of the zymogen for further study. Future work is important to define the role of the ovarian renin-angiotensin system in human reproduction.

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BOOK REVIEWS

Animal Cell Culture. Ed JW Pollard, JM Walker. Methods in Molecular Biology. Vol. 5. (Pp 713; £69.50). The Human Press Inc. 1989. ISBN 0-89603-150-0.

This volume is the latest in the very useful *Methods in Molecular Biology Series*. The editors have assembled contributions from 78 expert authors in 55 chapters covering the whole spectrum from basic culture techniques for mesenchymal, neuronal, epithelial, and haemopietic cells through to detailed methods for cytogenetics, gene transfer, and in situ hybridisation. There are 10 chapters covering the production and characterisation of hybridomas and monoclonal antibodies. There are high quality line drawings and monochrome photographs throughout and the chapters are, in general, well referenced. A particular feature of the series is the inclusion of a Notes section at the end of chapters, where one finds expert tips that can enable one to apply these techniques even as a novice. Although there are some omissions, such as the freezing of cells other than hybridomas, I would recommend this book to all those considering using tissue culture in their work.

PA HALL

Immunosuppression and Human Malignancy. D Naor, B Klein, N Tarcic, J Duke-Cohan. (Pp 271; $69.50.) The Humana Press Inc. 1989. ISBN 0-89603-149-7.

This book contains four large chapters dealing with induction of suppressor cells by immunostimulants, control of natural killer cells by suppressor cells, suppressor cells in human malignancies, and finally suppressor cells and malignancy in experimental animal models. It is a very curious book. The authors go into great depth in the description of experiments designed to elucidate aspects of suppression. All of the conflicting data from different studies and different models, however, served to confuse this reviewer rather than enlighten him. Some attempt is made to summarise the results with large tables at the end of certain chapters, but these are lists and are not particularly useful. The central question stated at the beginning of the book, of whether suppressor cells permit malignancy or are a result of it, remains unanswered, and since the question of suppressor cells is rather contentious these days, it really is rather hard to make sense of the central theme of the book. There is a lot of discussion of suppressor macrophages but no mention anywhere of tumor necrosis factor α. The book would have been topical in the early 80s but now it seems slightly anachronistic, dealing as it does with cellular rather than molecular immunology. Virtually all of the experiments described are rather old, as is the literature cited. Towards the end of the book is a section on the T cell receptor with references up to 1987, but this appears to have been added on at the end in an attempt to make the book more topical. It is very poorly illustrated, containing only three figures, the first of which appears on page 50. Clearly a great deal of work has gone into the book and it contains 860 references, but more attention to presentation and less to documentation would have helped. It is unlikely to be of use to the general pathologist, clinician, or immunologist but might be of interest to afficianodos of tumour immunology.

TT MACDONALD

Progress in Surgical Pathology. Vol X. Ed CM Fenoglio-Preiser, M Wolff, F Rilke. (Pp 265; DM 164.00.) Springer. 1989. ISBN 3-540-51360-4.

This book consists of a series of review chapters written by specialists from several European countries and centres in the USA on different topics relating to surgical pathology. The text is generally easy to read and well illustrated. Most articles are above average interest and this was shown clearly by the book being temporarily appropriated by my wife (who is also a histopathologist) so that she could read several chapters.

I found the best chapters were those on cutaneous histiocytoses in children, Hodgkin's disease, chromogramin A and B in neuroendocrine tissues, thymic tumours, campylobacter in gastroduodenal disease, mucosal prolapse syndrome, and the surgical pathology of the anal canal, but that probably reflects my range of interests. Other topics covered were extrinsic allergic alveolitis, pulmonary vascular neoplasms (I hadn't realised there were so many possibilities), telangiectatic osteosarcoma of bone, sarcomatoid carcinomas of the breast and micropapillary hyperplasia of the breast. Chapter 1 discussed the importance of right and left handedness in pathologists and its association with happiness and other factors. The author admits it is a relatively unscientific enquiry but nevertheless it is thought provoking.

Overall, a worthwhile book which I would recommend to practising surgical pathologists to borrow or buy. I should say, however, that it falls a little short of the consistently high standards achieved by *Recent Advances in Histopathology* edited by Anthony and MacSween.

DR TURNER

Fine Needle Aspiration Cytology: Lymph Node, Thyroid & Salivary Gland. PS Feldman, JL Covell, TF Kardos. (Pp 278; $184.) Raven Press. 1990. ISBN 0-89189-293-1.

This is a highly illustrated book written by three American authors who perform their own fine needle aspirations; it could have been subtitled "A manual of head and neck cytopathology". It is divided into five chapters. The first provides a practical guide to the technique of performing a fine needle aspiration (FNA), as well as preparing smears and staining them. There are useful guidelines on the general interpretation of FNA material and the reporting of results. Three separate chapters follow on lymph node, thyroid, and salivary gland cytopathology. A final general chapter discusses branchial cleft cysts, lymphangioma, carotid body tumour, and neuroblastoma.

Apart from the use of a North American classification scheme for the non-Hodgkin's lymphomas, I cannot fault this book. Each chapter contains a body of text in which the organ system concerned is discussed. Problems of interpretation and differential diagnosis as they pertain to FNA material are presented clearly and succinctly, including some useful tables. The text is followed by illustrated case reports which show in both colour and in black and white, the cytological, histological, clinical, and occasionally, radiological appearances of the pathology under discussion. Some electron micrographs are also provided.

The book is generally well balanced, the illustrations are of adequate quality, and several up to date reference lists are provided. I think it should appeal to a general audience, and I would recommend it to pathologists,