aggressive treatment with intravitreal and systemic antibiotics combined with steroid therapy.

Endogenous *E coli* endophthalmitis appears to occur almost exclusively in diabetics, and is invariably related to a urinary tract infection. Treatment of any infection in diabetics should be aggressive and care should be taken to cover the likely pathogens. The outcome of *E coli*

endogenous endophthalmitis is invariably poor and so any deterioration in vision should be assessed promptly as it is not necessarily painful. The alteration of the red reflex to a whitish reflex is an important early sign. In settings where patients cannot communicate, periodic examination of the fundi should be undertaken. Thankfully this condition remains rare.

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Elevated serum prostate-specific antigen and pancreatic carcinoma

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Summary
We report a case of elevated serum prostate-specific antigen-like immunoreactivity in a man with a disseminated pancreatic carcinoma

Keywords: prostate-specific antigen, prostatic biopsy, pancreas

Measurement of serum prostate-specific antigen (PSA) is said to be a specific and sensitive tool in the diagnosis of prostatic cancer.¹ It has been said that PSA is not present in any other normal tissue obtained in men apart from the prostate, nor is it produced by cancers of the breast, lung, colon, rectum, stomach, thyroid or pancreas.²

Case report

A 79-year old man was admitted with a four-month history of weight loss, diarrhoea and abdominal pain. Physical examination and investigations including ultrasound, computed tomography (CT) of abdomen and fine needle biopsy of a retroperitoneal mass suggested the diagnosis of adenocarcinoma of the pancreas with multiple secondary metastases in the liver. Bone scans and chest X-ray excluded any skeletal or thoracic spread. Routine haematology and biochemistry investigations were nor-

mal except for serum γ -glutamyl transpeptidase, amylase and erythrocyte sedimentation rate which were noted to be 85 U/l (ref range 1-28), 150 IU/l (ref range 30-110) and 33 mm/h, respectively. Serum PSA and acid phosphatase measurements were requested at the time of admission and showed an unexpectedly high value for PSA. Prostate-specific acid phosphatase levels of 1.6 and 2.2 IU/l (tartrate labile fraction) (ref range 0.1-2.8)

Causes of raised serum PSA
Analytical interference
Prostatic
• prostatitis
• prostate infarcts
• benign enlargement
• carcinoma
Non-prostatic
• breast milk
• apocrine sweat glands*
• salivary gland*
• para-urethral gland*
• renal cell adenocarcinoma**
• breast tumours*
• apocrine sweat gland tumours*
*not detected to be high in blood
**only with assays using polyclonal antisera

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Accepted 15 September 1995

Postgrad Med J: first published as 10.1136/pgmj.72.848.363 on 1 June 1996. Downloaded from <http://pmj.bmj.com/> on April 26, 2023 by guest. Protected by copyright.

were noted on two occasions while serum PSA levels of 233, 206 and 272 $\mu\text{g/l}$ (ref range 1.0–4.0) were shown on separate occasions. The patient denied any prostatic symptoms and evaluation by digital rectal examination, transabdominal and transrectal ultrasonography as well as needle biopsy (four cores of prostatic tissue) showed no evidence of carcinoma of prostate. He became extremely ill with marked weight loss during his hospital stay prior to his demise.

Serum PSA measurements are routinely analysed in our laboratory by using a Hybritech™ immunoassay (Tandem™-E PSA) which uses monoclonal antisera of mouse origin. Measurement of serum PSA using an immunoradiometric assay kit (CIS Biointernational™) in a different laboratory was concordant with measurements in our laboratory. Tests for heterophilic antibody using mouse serum were negative. Serial dilution of serum for PSA measurements was linear and excluded non-heterophilic antibody interference.

Discussion

The history, physical examination and investigations performed in this man showed an apparent carcinoma of pancreas with secondary spread into the liver. The unexpected elevation of serum PSA was associated with the absence of prostatic symptoms in the history as well as a normal digital rectal examination. A transabdominal and transrectal ultrasound of the prostate showed a normal sized prostate. Prostatic needle biopsies which enabled four cores of prostatic tissue to be sampled showed no evidence of malignancy. Acid phosphatase has been shown to increase reliably in those with disseminated prostatic disease³ but our patient had normal values on two occasions.

The investigations on the PSA analysis in this man excluded analytical interference and confirmed that our routine laboratory methodology was consistently detecting PSA-like immunoreactivity.

PSA is described as prostate-specific although not prostate cancer specific.⁴ There is, however, increasing recognition that non-prostatic tissues also produce PSA.⁵ PSA-like immunoreactivity has been shown in breast milk, breast tumours, and apocrine sweat glands.⁵ Histochemical staining in carcinoma of salivary and Skene's glands has shown PSA-like immunoreactivity^{6,7} although elevation of PSA has not been seen in the blood. Rasmuson and colleagues have demonstrated elevated PSA values in sera of women with renal cell cancers using polyclonal antisera,⁸ although these measurements could not be reproduced with assays using monoclonal antisera. The Hybritech™ kit for PSA immunoreactivity measurement specifically states that PSA is not present in pancreatic cancer tissue. Non-cancerous pancreatic tissue is rich in kallikrein (a peptide belonging to the same superfamily as PSA), but has not been shown to contain PSA.⁹

Recent molecular studies have located the PSA gene to human chromosome 19, giving both sexes two copies of this gene.¹⁰ In addition, androgen-response element sequences have been separately identified in the promoter region of the PSA gene¹¹ and human androgen receptor has been shown to specifically bind to these promoter sequences.¹² These findings suggest that PSA may be expressed in tissues where an appropriate steroid receptor is present. It is tempting to speculate that neoplastic pancreatic tissue in this man may have secreted PSA-like immunoreactive substance.

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