Urinary 6-oxo prostaglandin $F_{1\alpha}$ in myocardial infarction

Platelet activity, which may play a part in both atherogenesis and thrombosis, is inhibited by prostacyclin in vitro. We report a study of urinary 6-oxo prostaglandin $F_{1\alpha}$, one of the stable prostacyclin metabolites, in myocardial infarction.

Patients, methods, and results

Eleven patients (mean age 57.9 years) with a diagnosis of myocardial infarction (classic history and electrocardiographic and enzyme changes) who denied having taken any drug for the previous 10 days gave an early morning two hour urine specimen after an overnight fast. Samples were collected on days 5 and 6, and eight to 10 weeks after myocardial infarction. Control samples were obtained under the same conditions from otherwise fit inpatients before operation who did not have the above diagnostic criteria. Family practitioners confirmed that no drug treatment was given after the patients' discharge from hospital.

Prostaglandins were extracted from urine samples using octadecylsilyl silica C18 minicolumns and the prostanoid fraction eluted with methyl formate. 6-Oxo prostaglandin $F_{1\alpha}$ was separated from the other prostanoids by high performance liquid chromatography. The recovery of a known quantity of tritium labelled 6-oxo prostaglandin $F_{1\alpha}$ (Amersham International) during the preliminary stages was 60-70%. 6-Oxo prostaglandin $F_{1\alpha}$ was measured by radioimmunoassay using antiserum obtained from rabbits together with a radiolabelled conjugate of histamine and 6-oxo prostaglandin $F_{1\alpha}$. Each urine specimen was assayed in triplicate.

Urine taken on days 5 and 6 after myocardial infarction gave a mean (SEM) 6-oxo prostaglandin $F_{1\alpha}$ concentration of 163.4 (25.0) pmol/mmol creatinine ($n=10$), which was significantly higher than the control concentration of 111.4 (12.8) ($n=11$) ($p<0.05$, Student's t test) (figure). In contrast the mean urinary 6-oxo prostaglandin $F_{1\alpha}$ concentration recorded eight to 10 weeks after myocardial infarction was 57.2 (15.0) ($n=7$), significantly lower than that in the controls ($p<0.001$, Wilcoxon's rank sum test).

Urinary 6-oxo prostaglandin $F_{1\alpha}$ concentrations expressed as ratio of urinary creatinine.

Comment

Recent work has suggested that plasma 6-oxo prostaglandin $F_{1\alpha}$ concentrations are too low to be measured easily by radioimmunoassay.¹ In contrast, urinary 6-oxo prostaglandin $F_{1\alpha}$, representing a store of excreted prostanoid, offers a convenient method of studying prostacyclin metabolism in man. It is still uncertain, however, how much urinary 6-oxo prostaglandin $F_{1\alpha}$ is derived from the renal and

urinary tract. In this study variations in renal production were minimised by selecting patients with normal renal function who were not taking any drugs.

A common feature of inflammatory or mechanical tissue injury is increased release of prostaglandins from the damaged tissue. Prostacyclin is released from many tissues including those of the heart.² Thus the increased urinary 6-oxo prostaglandin $F_{1\alpha}$ concentration recorded shortly after myocardial infarction may have reflected tissue damage within the heart or transient hypoxic damage to other organs. The decreased amounts of 6-oxo prostaglandin $F_{1\alpha}$ excreted two months after myocardial infarction, when extensive healing should have occurred, may have been due to a late response to the myocardial infarction itself or may indicate that there is a generalised reduction in basal production of 6-oxo prostaglandin $F_{1\alpha}$ in patients prone to myocardial infarction. There is evidence that vascular production of prostacyclin is decreased in rabbits with experimentally induced atheroma.³ One possible explanation for this decrease might be the inhibition of prostacyclin synthetase by lipid peroxides,⁴ concentrations of which may be raised in atherosclerosis,⁵ which is a major aetiological factor in myocardial infarction.

These findings should encourage further investigation into the hypothesis that defective prostacyclin production by vascular endothelium may be associated with myocardial infarction in man.

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Male genital self mutilation after paternal death

Deliberate male genital self mutilation is rare.^{1 2} We report the first two cases after paternal death; one patient later committed suicide.

Case reports

Case 1—A 24 year old single van driver presented as an emergency one and a half hours after an attempt at self circumcision with a pair of scissors. He admitted to being depressed after the death of his father one week previously and said that he "thought this might lift him out of his depression." There was no medical history apart from an appendicectomy and no psychiatric history. He denied drinking alcohol or drug abuse. There was no hallucination or obvious thought disorder. He described himself as normally level headed. On examination he had three 3-4 cm lacerations over the distal penile skin and mucous membrane. These were debrided and sutured under general anaesthetic. He made a satisfactory recovery but declined psychiatric help.

Case 2—A 37 year old unemployed single man was admitted as an emergency in a state of shock two hours after an attempt at self castration. He was resuscitated with intravenous colloid and then became agitated and refused blood transfusion as "an unsupportable invasion of his privacy."

He talked of his previous incarnation in eighteenth century Russia when he had led a large religious movement, was persecuted, and had castrated himself with a red-hot poker. There was no psychiatric history, but his father had died three days previously. A consultant psychiatrist diagnosed an acute reactive psychosis, detained the patient formally, and sedated him. He was then transfused, and his wounds were explored under general anaesthetic. There was a 10 cm scrotal laceration, and he had successfully excised the left testis and the lower pole of the right. Haemostasis was achieved and the wound debrided and sutured. He made a satisfactory recovery and was reviewed by the consultant psychiatrist, who found him to be lucid and rational. He was discharged on sedation with psychiatric and surgical follow up arranged but subsequently committed suicide by swallowing weedkiller.

Comment

In a recent review of 53 reported cases of deliberate male genital self mutilation only seven patients were considered not to be psychotic at the time of self mutilation.³ Ten had made previous attempts at genital self mutilation, and at least half had received previous psychiatric treatment, many being long term inpatients. The two patients described here are unusual in that genital mutilation was the first indication of a psychiatric disorder. They are the first cases to be recorded after the recent death of a father, though in one previous case the patient had just lost his pet dog and a tame bird.³

Genital injuries varied from those without lasting harm to complete amputation of the genitals with other bodily injury, but no difference has been observed in the severity of injuries between the psychotic and non-psychotic groups.³

Genital self mutilation after paternal death might be expected on theoretical grounds in individuals with an unresolved oedipal complex. The son may have regarded his father as a potential rival and have felt guilty about his continuing sexual attachment to his mother. Thus the guilt that is part of any bereavement reaction⁴ might occasionally be greatly exaggerated. The son might then punish himself by genital injury or castration, thereby carrying out what he considered was his father's wish in an attempt at restitution.

Further cases may be prevented by increased awareness. Sons at risk would probably have an undue attachment to mother, an intense ambivalent relationship to their deceased fathers, and a very intense or otherwise abnormal grief reaction.

The subsequent suicide risk has been estimated at 50% by one group,⁵ but Greilheimer and Groves thought that only five of their 53 reviewed cases were acutely suicidal.³ The second case reported here emphasises the risk in these patients.

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Is measurement of girth of value in assessing intraperitoneal bleeding after trauma?

Measurement of abdominal girth is frequently used to aid the assessment of intraperitoneal bleeding after trauma. As the circumference (C) of a sphere increases in proportion to the cube root of the volume (v) ($C = \sqrt[3]{6\pi^2 v}$), it seemed to us that a considerable volume of blood might accumulate in the abdomen with little change in girth. We

decided to test this hypothesis and assess the reproducibility of the measurement in a ward setting.

Patients, methods, and results

Three groups of adult patients were studied. All measurements of abdominal girth were performed using a non-elastic tape at a premarked level.

In group 1 six patients undergoing peritoneal dialysis for chronic renal failure had their girths measured during infusion of dialysate. All intra-abdominal cannulas had been in place for one week or more without abdominal discomfort and girths were measured by one person. The table gives the results. The increase in girth varied by a factor of three despite identical volumes of dialysate injected.

In group 2 interobserver variation was assessed by taking measurements in convalescent patients with limb trauma but without abdominal injury. Measurements were taken on three patients by six nurses and one doctor within a 20 minute period. The results showed a scatter either side of the mean but in no case was the difference between minimum and maximum measurements less than 2 cm, and in one patient it was as high as 3 cm. Yet the change in girth after a 2 litre fluid infusion was as small as 2.5 cm in one patient (table).

Finally, in group 3 we studied diurnal variation in girth, again in convalescent patients with limb trauma but without abdominal injury. One observer made four measurements at specified times during the day. The observed changes varied from 1.3 cm to 3.0 cm. Again the maximum variation was greater than or equal to that obtained after a 2 litre infusion (cases 1 and 4; table).

Variation in girth with volume (cm)

Case No	Before infusion	After infusion of:		Change after 2 l infusion
		1 l	2 l	
1	77.5	80.0	80.0	2.5
2	118.0	119.0	121.5	3.5
3	86.5	90.0	92.5	6.0
4	88.0	90.0	91.0	3.0
5	86.5	90.0	94.0	7.5
6	106.5	110.5	111.5	5.0

Comment

Intra-abdominal bleeding may require urgent surgery to save life. Measurement of abdominal girth is sometimes used as one of the many indicators of such bleeding, but London stated that late swelling of the belly is more likely to mean ileus than intra-abdominal bleeding.¹

There are several other factors, apart from bleeding, which may cause abdominal distension. The bowel may become distended due to ileus which may be secondary to the initiating trauma or to various anaesthetic and narcotic drugs used in the treatment of that trauma. Pain and fear at the time of injury may cause air swallowing, thus adding to the distension. The volume of the bladder is often neglected and may be an important factor, particularly after infusion of large volumes of fluid and in inebriated patients. Fluid may also accumulate in the abdomen due to transudation with any inflammatory process, including visceral perforation. Further, if used in the patient with thoracic injury, and especially with positive end expiratory pressure ventilation, diaphragmatic splinting may erroneously accentuate abdominal distension.

We have shown that in uninjured people observer and diurnal variation may be greater than the increase in girth after infusion of two litres of fluid. Our findings suggest that an increase in girth may occur due to bleeding but that large volumes are required to produce a clear increase, if observer error and diurnal variation are considered. Hence we think that this measurement is of little clinical value in the assessment of intraperitoneal bleeding and that it may distract the unwary from more sensitive haemodynamic indicators of blood loss.

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