PROGRESSIVE VESICAL-NECK STENOSIS IN THE FEMALE

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Organic obstruction at the neck of the urinary bladder in the adult female is a very real condition and not an uncommon one; despite this, it is seldom recognized and certainly poorly documented. Since its presence is no less damaging to the renal tract than is bladder-neck obstruction in the adult male, its early recognition and treatment is of considerable importance. The first requirement for this is an increased awareness of the possibility that such pathology exists. There appears at present to be a hiatus in urological teaching regarding this clinical entity and there is only sparse literature on the subject.

The condition is not perfectly understood, particularly with regard to pathogenesis, but certainly there is a clear-cut clinical picture which, providing there is a high index of suspicion, is easy to recognize on investigation. If it is recognized early in its natural history, the disastrous effects on the upper renal tract of prolonged obstruction to the outflow of urine may be prevented, and a substantial cause of irreversible chronic renal failure, not uncommonly terminating in uraemia and death, will be eliminated. Furthermore, the vast number of women attending urological clinics complaining of recurrent cystitis which fails to respond to any form of treatment, would be considerably reduced if more of them were investigated for an obstructive element at the bladder neck. The diagnosis is missed most often through lack of knowledge of the occurrence of urinary obstruction in the female.

This paper is intended to draw attention to a group of clinical pictures which it is felt have a common underlying cause, and are merely stages in the natural history of what I have called progressive bladder-neck stenosis. The condition, in its fully developed state, has been described as the female prostate.[1] It would be more correct, I think, to draw an analogy between the condition and certain forms of Marion's disease in the male.

In Marion's disease the following can occur: (*a*) stenosis of the bladder neck as a result of either subepithelial fibrosis or muscular hypertrophy, and (*b*) median-bar obstruction which may be due to either a localized muscular hypertrophy or an adenoma of outlying glandular foci. The pathogenesis of acquired Marion's disease is not fully understood; neither is that of progressive bladder-neck stenosis in the female. As the symptom-complex is described it will become apparent that, although an inflammatory process could satisfactorily explain most of the changes that take place, it cannot explain them all.

ANATOMY AND PHYSIOLOGY

The bladder neck is the junction between the bladder and the urethra, and minor changes at this junction can lead to gross interference with micturition. The detrusor muscle of the bladder is continued into the proximal urethra, and there is also muscle posteriorly under the trigone, partly derived from ureteric muscle, which enters into the posterior urethra. When micturition occurs contraction of the detrusor muscle leads to shortening of the urethra and pulling open of the sphincter muscle at the bladder neck. Any rigidity of the bladder neck from whatever cause will act as a resistance to this mechanism and will result in obstruction of varying degree.

The female urethra is surrounded by many glands. Huffman[2] prepared wax models of the gland ducts of the female urethra which showed these structures in close proximity to the internal meatus on both the anterior and posterior aspects of the channel. Many believe that these represent the homologue of the prostate in the male. Folsom[3] and Young[4] both commented on the similarity of the tissue excised from the bladder neck in the female to that of the prostate in the male. Young[4] described a case of adrenal virilism in the female, in which hypertrophy of these glands was so marked that to the naked eye the structure below the bladder neck resembled the prostate. The true role of these glands in the proximal urethra is problematical.

PATHOLOGY

'Recurrent Cystitis'

For many years it has been taught by some authorities that the para-urethral glands become infected and are responsible for 'recurrent cystitis'. This has been disputed by others, since the urine is so very often sterile and free from pus cells, and other signs of infection such as pyrexia and leucocytosis are absent. Examination of the proximal urethra by urethroscopy in these cases, however, will often demonstrate definite signs of an inflammatory process. These take the form of small granular vegetations or pseudopolypi in the posterior urethra and on the bladder neck, and there are often associated changes on the trigone resulting from congestion and oedema. Similar appearances are seen in males with posterior urethral inflammatory states. Non-proliferative inflammatory signs, such as oedema with hyperaemia, and patches of thickened epithelium, are also commonly seen.

Intermittently the infection may burst forth from these glands, frequently after intercourse, causing a true attack of cystitis. Most of the attacks of frequency, urgency and dysuria are not, however, true cystitis, but result from the irritation caused by the hyperaemia, granulomata and pseudopolypi. In these attacks the urine is abacterial and apyurial, and this has led to considerable confusion of thought.

Fibrotic Changes

Ultimately the inflammatory changes involve the subepithelial and muscular layers, with subsequent fibrosis. The resulting rigidity of the bladder neck leads to obstruction with its sequelae. The common histological findings are squamous metaplasia of the epithelium of the bladder neck, infiltration with inflammatory cells, oedema, and fibrosis. Occasionally muscular hypertrophy of the internal sphincter is also present.

Chronic Cervicitis

The part played by chronic cervicitis has been suspect from time to time. Although there is often an association between the occurrence of the two conditions, the precise role, if any, played by the cervical infection in the development of bladder-neck pathology is not established. It is interesting, however, to note in this connection that Winsbury-White[5] showed experimentally that, when Indian ink was injected into the cervices of guinea-pigs, particles of this ink could be demonstrated around the bladder neck and could even be shown to extend up to the kidney *via* the peri-ureteric lymphatics.

CLINICAL FEATURES

The cases under discussion fall into two main groups: (*a*) those with urinary symptoms, but no signs of obstruction; and (*b*) those with symptoms and signs of obstruction.

There is often no firm demarcation, since there is a

gradual progression from one stage to the next. Early obstruction, showing no signs, is occasionally present and can often only be diagnosed in retrospect by the response to dilatation incidental to diagnostic endoscopy.

Patients with no Signs of Obstruction

This is essentially the group of females who attend for recurrent attacks of so-called cystitis. The attacks consist of bouts of urgency, frequency and dysuria, which are variable in length, but usually of only a few days' duration. In some patients, but not in the majority, there is a history of intercourse as a precipitating cause. Repeated urine examinations have usually been negative, showing no bacteria or pus cells. There is often a history of frequency of micturition between attacks. Urgency is a striking symptom, and these patients' symptoms are often susceptible to aggravation by emotional disturbances. Other symptoms are often present, such as aches and pains in the loins and groins or about the lower part of the trunk.

The chief feature is the recurrence of attacks of 'cystitis' associated with sterile urine. In an occasional attack the urine is infected. Physical examination is usually negative, although an infected cervix is sometimes found. Intravenous pyelography is essential to exclude any other lesion of the renal tract; in this group evacuation of the bladder is complete. The cystoscopic and urethroscopic findings are diagnostic — the bladder is as a rule normal, except for some possible congestion and oedema of the trigone. Careful urethroscopy will provide valuable diagnostic information. The proximal urethra is as a rule hyperaemic, and proliferative changes of varying degree may be present; these consist of small friable granulomata or pseudopolypi situated in the proximal one or two centimetres of the urethra and on the bladder neck, usually on the posterior quadrant.

Patients with Signs of Obstruction

The presenting features in these cases are dependent on the degree and duration of the obstruction and the extent of any renal damage. In the early stage of obstruction there is frequency of micturition, and recurrent or persistent urinary infection may occur. This is due to secondary infection of residual urine. At this stage in the disease process dysuria, frequency, and even haematuria can occur, but these have a different mechanism of causation to similar symptoms in the first group. Ascending infection may result in pyelonephritis, and the first presenting signs and symptoms may be those of pyelonephritis.

Careful questioning of patients will elicit a history of difficulty with micturition, particularly on initiating the act, a stream that has deteriorated in force, intermittency, and often a feeling of not emptying the bladder completely.

In the early stages the bladder-neck obstruction may be well compensated and there may be minimal residual urine. Chronic retention of urine with a painlessly distended bladder occurs at a very late stage. Secondary infection in patients with a large residual urine, particularly where long-standing back pressure has already caused insidious impairment of renal function, may precipitate uraemia. This may be the presenting feature and cause difficulty in diagnosis. The following case illustrates this type of problem:

Case 1. A woman aged 43 years was admitted to hospital with pain in the left loin of 3 weeks' duration, and with vomiting and constipation. She was gravely ill. She gave a history of difficulty with micturition and was reported not to have passed urine for 48 hours before admission. Her blood chemistry was as follows: Blood urea 274 mg. per 100 ml., serum sodium 132 mEq./l., serum potassium 3·0 mEq./l., serum chloride 79 mEq./l., and CO_2 combining power 20·1 mEq./l. A diagnosis of uraemia was made, and since she had a history of tuberculosis I was asked to see her in case she had renal tuberculosis. On examination there was a full bladder, and on catheterization 20 oz. of urine were removed. The urine was found to be grossly infected. A diagnosis of bladder-neck obstruction with pyelonephritis and severe renal impairment was made. Continuous bladder drainage, using a sterile closed circuit, was instituted, and the regime for acute renal failure was begun. Drainage was continued for 4 weeks, and on removing the catheter spontaneous micturition was restored. The serum electrolyte pattern slowly returned to normal, and on discharge the blood-urea level was 49 mg. per 100 ml. Examination 4 months later showed complete bladder evacuation, but pyelonephritis persisted for 5 months in spite of intensive therapy.

This was a patient with bladder-neck obstruction giving rise to chronic retention of urine, pyelonephritis and uraemia.

Patients may develop retention of urine on being confined to bed for an illness, and some present in this way following surgery for unrelated conditions. Two of my patients presented like this after haemorrhoidectomy. A history of long-standing difficulty with micturition was elicited in both.

For reasons which are ill understood, females with urinary symptoms are usually referred initially for a gynaecological consultation. Cystocele *per se* is not a cause of urinary symptoms other than stress incontinence, but it is often operated on in the mistaken belief that it is. This has been done most commonly where there has been a failure to distinguish between urge and stress incontinence. Colporrhaphy is of necessity associated with postoperative fibrosis in the region of the bladder neck, and if an undiagnosed bladder-neck obstruction was originally present, the condition is aggravated and retention of urine may even occur immediately after operation. The following is an illustrative case report:

Case 2. A woman aged 44 years had a colporrhaphy performed for cystocele with urinary symptoms. Postoperatively she developed retention of urine, and on the 15th day I was asked to see her. During this time catheterization, combined with attempts at stimulating the bladder detrusor, had failed to restore spontaneous micturition. On careful questioning she gave a history of difficulty with micturition, frequency, and retention of urine following each of 6 births. Endoscopic examination revealed a marked ridge, about half-an-inch in width, on the posterior quadrant of the bladder neck. The overlying mucosa was white and immobile. She had obviously had unrecognized bladder-neck obstruction before her colporrhaphy. I resected the ridge, which consisted of fibromuscular tissue, and following this spontaneous micturition was restored.

Chronic cervicitis is also often held responsible for a variety of urinary symptoms. If this is treated and the possibility of existing bladder-neck pathology is ignored, the original urinary symptoms persist or may even be aggravated.

An *intravenous pyelogram* provides valuable information in diagnosis. Catheterization for the estimation of residual urine is undesirable in view of the danger of introducing infection, and this information can be obtained readily from the post-evacuation antegrade cystogram. Residual urine in small amounts tends to go unrecognized in females as a rule, since scant attention is usually paid to this aspect of the pyelogram. In all females with urinary symptoms on whom an intravenous pyelogram is done, post-evacuation films of the bladder should be routine; if not, they should be requested specifically.

No residual urine or only a trace does not, however, exclude obstruction since, as mentioned above, in well-compensated bladder-neck obstruction there may not yet be any residual urine. Quantitative estimations of residual urine, as in the male with bladder-neck obstruction, are misleading and of no prognostic value. The presence of residual urine, in no matter how small a volume, is positive evidence of well-established obstruction with failure of compensation. In advanced cases there may be virtually no difference between the pre- and post-evacuation films of the bladder. Dilatation of the upper renal tract may be present.

Cysto-urethroscopy is the most important diagnostic procedure. A ridge may be felt at bladder-neck level as the instrument is passed along the urethra. On inspection a prominent ridge, of varying width, can be seen in the posterior quadrant. The ridge may be circumferential in nature, virtually forming a collar around the bladder neck. The mucosa overlying this is often white and immobile, or polypoid and hyperaemic when there is an associated active inflammatory process present.

It must be stressed that these changes can only be recognized with a forward-viewing lens system, such as is found in a panendoscope or urethroscope, and are not recognized readily with a cystoscope, which has a right-angled viewing system. Awareness of this pitfall is of the utmost importance, since on many occasions I have seen patients with bladder-neck pathology who have been reported previously as normal on cystoscopy. Unless the endoscopic examination has included examination of the bladder neck with an adequate instrument, it will have been valueless in establishing the diagnosis. It is often useful to fill the bladder with fluid and to apply suprapubic pressure while viewing the bladder neck, the rigidity of which will then become more obvious. In some instances there is evidence of detrusor hypertrophy in the form of varying degrees of trabeculation. This is of course not always present and is dependent on the duration and degree of obstruction. Saccule and diverticulum formation can also occur.

TREATMENT

Without Obstruction

In the group without obstructive features, treatment is directed towards eliminating the proliferative inflammatory changes which are present at the bladder neck. Endoscopy is carried out and all granulomata and pseudopolypi are fulgurated or resected, dilatation being effected simultaneously. In early cases with absent or minimal fibrosis the combination of the two procedures is usually sufficient to remove all symptoms. Very often, even in the absence of obvious proliferative lesions, the dilatation is in itself curative. Patients so cured are often those who are mistakenly labelled as having functional trouble, since diagnostic cystoscopy has in itself apparently effected the cure.

With Obstruction

Patients with obstruction are treated by perurethral resection of the bladder neck. It is quite possible, using a resectoscope, to resect adequate amounts of mucosa and underlying internal sphincter accurately and without any risk. If the cutting diathermy loop is accurately placed, there is no possibility of damaging the external sphincter, and in experienced hands fistula formation is unknown. The internal sphincter is not important in urinary continence. I have resected varying amounts of tissue without interfering in any way with continence. The amount of tissue to be resected varies with each case and experience is the guide in this respect; often only minimal tissue need be removed. After resection, the bladder is drained with a Foley catheter for a few days.

The following report illustrates a typical example of the usual uncomplicated type of case encountered:

Case 3. A woman, aged 42 years, presented with a history of frequency of micturition of several years' duration. A few months before she was seen by me, the frequency became worse and urgency became a marked symptom. She complained of difficulty with micturition and felt as though she was not emptying her bladder properly. An intravenous pyelogram showed a normal upper renal tract, but there was a large residual urine in the bladder on the post-evacuation film. Endoscopy showed a trabeculated bladder and a marked ridge on the posterior quadrant of the bladder neck. This was removed by perurethral resection and micturition was restored to normal.

When obstruction has caused severe renal dysfunction, preliminary drainage by indwelling urethral catheter may be necessary. Any biochemical imbalance which exists is attended to, and this is combined with vigorous treatment of any infection that may be present. Drainage is maintained until maximal correction of all abnormalities is achieved. It is permissible to proceed to operation once the general condition is satisfactory, the normal electrolyte pattern of the plasma has been restored, and the blood urea is stabilized, even if its level is still above the accepted normal. Patients admitted in acute renal failure must be treated by suppression of endogenous protein metabolism until renal function has improved. The principles in the management of these patients differ in no way from those in the management of renal failure from any other cause.

Results of Treatment

The results of treatment are very satisfactory. In early cases the attacks of 'cystitis' cease and the patients become asymptomatic. In spite of the uncertain role of associated cervical pathology, I have usually advised that this be dealt with gynaecologically, either before or at the time of operation. Treatment of the cervical infection alone will, of course, not remove the urinary symptoms. In the group of patients with obstruction, transurethral resection has been eminently successful in removing the obstruction and restoring the normal function of the urinary tract.

SUMMARY

Bladder-neck obstruction can and does occur in the female. Its presence is no less damaging to the renal tract than is bladder-neck obstruction in the male. *Anyone treating uraemia in the male, with or without urinary infection, is very quick to suspect the presence of bladder-neck obstruction and undertake the necessary investigations and remedial therapy. There is, however, a marked tendency to overlook the comparable possibility in women.*

The stenosis which occurs appears to be the end result of an inflammatory process commencing in the proximal para-urethral glands. The earliest manifestation of the condition is recurrent abacterial and apyurial cystitis, often incorrectly attributed to gynaecological pathology in the first instance.

Treatment in the early stages consists in removing any proliferative chronic inflammatory changes present in the proximal urethra and bladder neck by means of endoscopic diathermy, and dilatation.

In the obstructive stage adequate perurethral resection of the bladder neck is highly successful in removing the obstruction and restoring normal function. Renal damage is the price paid for failure to diagnose obstruction early enough.

A few examples of the mode of presentation of patients with progressive vesical-neck stenosis have been given.

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