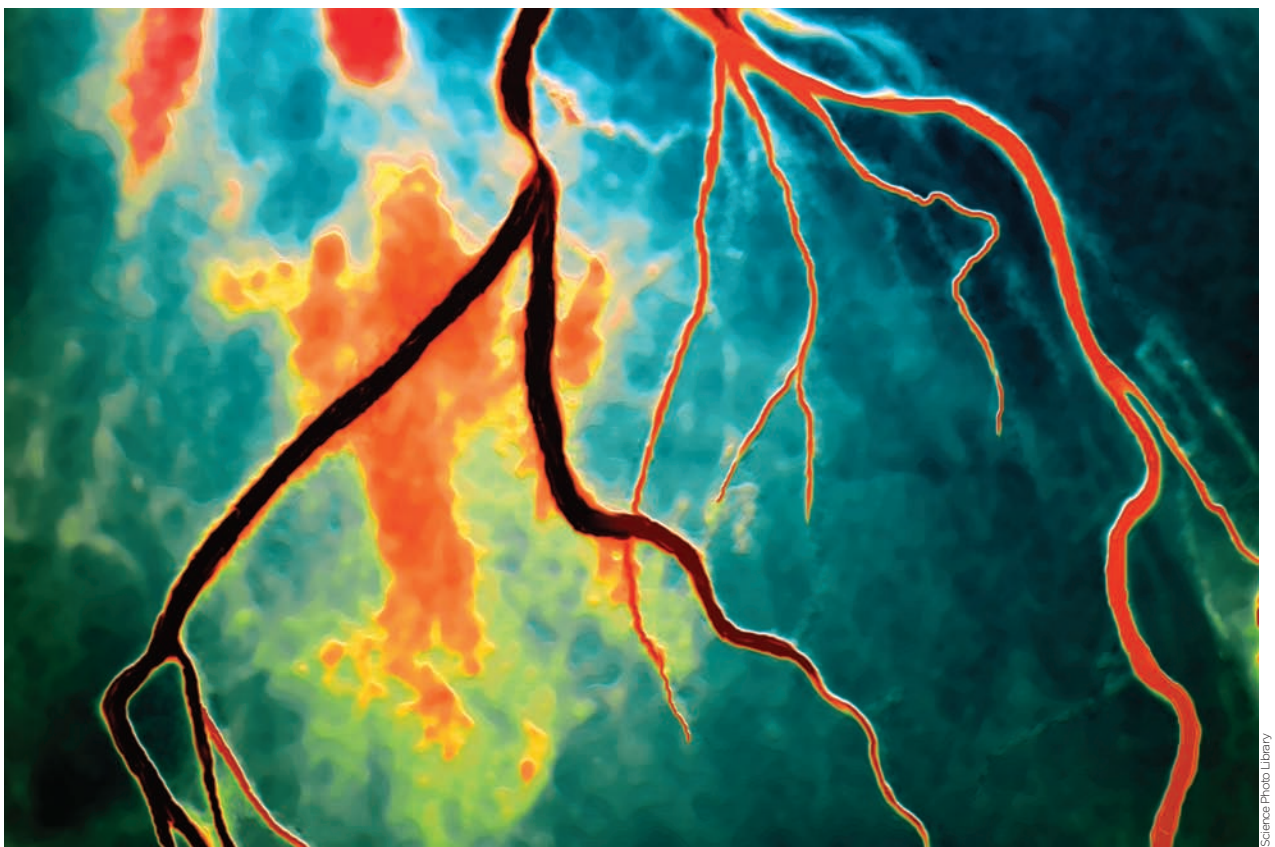
Early detection of erectile dysfunction may prevent CVD

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How should erectile dysfunction be investigated?

ERECTILE DYSFUNCTION (ED) IS A COMMON CONDITION DEFINED BY THE INABILITY TO achieve and sustain an erection adequate for sexual intercourse.

Population studies estimate a prevalence of between 32% and 52% for all grades of ED.[1,2] ED is more common as age increases, and one study found the prevalence of complete ED to triple, from 5% to 15%, between the ages of 40 and 70.[2]

The cause of ED may be multifactorial (see table 1, p22). However, there is increasing evidence to support a vascular aetiology. ED and cardiovascular disease (CVD) share common risk factors. So modifying risk factors for CVD may lead to an improvement in erectile function. Early identification of ED may also provide a window of opportunity in which clinicians can take preventative measures against the development of CVD. »

Table 1

Causes of ED[3,4]

Psychogenic
- Depression, schizophrenia, anxiety, situational

Organic

Vascular
- Risk factors: hyperlipidaemia, obesity, smoking, diabetes
- Secondary to trauma, Peyronie's disease, penile fracture, treatment for priapism

Neurological
- Multiple sclerosis
- Parkinson's disease
- Spinal cord injury

Endocrine
- Androgen deficiency
- Hypoprolactinaemia
- Hypo/hyperthyroidism
- Diabetes

Drugs
- Antihypertensives (especially diuretics and beta-blockers)
- Antidepressants
- Antipsychotics
- Antihistamines
- Alcohol
- Recreational drugs (eg cocaine/heroin)

Combination of organic and psychogenic

Table 2

Investigations in the management of ED

In all patients:
- Fasting blood glucose

Suspected hypogonadism/reassurance:
- Testosterone; if low, measure prolactin and luteinising hormone

Suspected liver disease:
- Liver function tests

Suspected renal disease:
- Renal function
- Dipstick urine analysis

Prior to instigating hormone treatment:
- PSA

Erectile dysfunction may be a clinical manifestation of endothelial dysfunction, affecting penile circulation. This may be part of a generalised vascular disorder that leads to myocardial infarction, angina and stroke.[5] As the penile arteries are smaller in diameter than the coronary or carotid arteries, the clinical manifestation, ED, may appear before symptoms of CVD or cerebrovascular disease.

A study investigating the vascular structure of patients with ED and age-matched controls found that those with ED were significantly more likely to have endothelial defects in major blood vessels before the onset of CVD.[6]

If ED is part of the same pathological process that leads to CVD, it is no suprise that they share the same risk factors. Cigarette smoking may double the likelihood of developing ED (from a lifetime prevalence of 14% in non-smokers to 34% in smokers).[2] One study, investigating the prevalence of ED in primary care, found that diabetes and raised fasting blood glucose levels were associated with ED. Increased coronary risk scores have also been associated with an increased rate of ED.[2,7]

ED AS A SCREENING TOOL FOR CVD

The prevalence of ED is higher in men with CVD. However, the cause of ED may be multifactorial and factors such as medication, depression and decreased exercise tolerance may play a role.

In patients with known CVD, the prevalence of ED varies between 49% and 81%.[5,8,9] Roumeguére et al found that the prevalence of hypercholesterolaemia was significantly higher in men with ED (71% vs 52%).[10] More than half the ED group in this study had an increased 10-year CVD risk, compared with 33% in the non-ED group.

The presence of ED may be an indicator of CVD severity. Min et al evaluated ED as a predictor of severe CVD in patients referred for nuclear cardiac stress testing.[11] The study found the prevalence of ED in these patients was 55%, and that ED was associated with severe CVD and left ventricular dysfunction. Another study investigating ED in patients with a high risk of CVD found that the prevalence of ED was 81%, and that severity of ED correlated significantly with the number of coronary arteries affected by stenosis.[9]

However, the development of ED may predate a cardiovascular event, thus serving as a warning for the future development of CVD. Montorsi et al found that 49% of men attending an acute chest pain clinic had ED.[5] Of these patients, 51% had severe ED and 67% reported ED before CVD was diagnosed. The mean time interval between the onset of ED and CVD symptoms was 38 months.

Similarly, Hodges et al found that of 207 patients attending CVD rehabilitation programmes, 66% reported ED, with a mean duration of five years before diagnosis of CVD.[8] Only 53% of these men had consulted a healthcare professional about their ED.

In a population undergoing cardiac stress testing, the presence of ED has also been found to be an independent risk factor for abnormalities associated with high cardiac risk in patients without established CVD.[11]

Therefore, when assessing a patient in primary care for CVD risk factors, specific questions should be asked about sexual function, as ED can serve as a predictor for subsequent CVD.

GPs should take a detailed sexual history. History taking should not only focus on cardiovascular risk factors that may contribute to the development of ED, but also aim to elicit other causes, whether psychological, psychosocial or physical (see table 1, left).

Blood pressure measurement and examination of the genitalia should be carried out and, if hormone therapy may be indicated, digital rectal examination. Specific examination may be guided by information elicited during history taking.

Guidelines for the management of ED have been developed in association with the British Association of Urological Surgeons, the Association for Genitourinary Medicine and the British Diabetic Association.[12]

The guidelines recommend the measurement of fasting plasma glucose in all patients. Testosterone levels should be measured if hypogonadism is suspected, or if the patient needs reassurance, and, if low, prolactin and luteinising hormone levels should be measured (see table 2, left). »

key points

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Erectile dysfunction may be a clinical manifestation of endothelial dysfunction, affecting penile circulation. This may be part of a generalised vascular disorder that leads to myocardial infarction, angina and stroke. As the penile arteries are smaller in diameter than the coronary or carotid arteries, the clinical manifestation, ED, may appear before symptoms of CVD or cerebrovascular disease.

Cigarette smoking may double the likelihood of developing ED (from a lifetime prevalence of 14% in non-smokers to 34% in smokers). Diabetes and raised fasting blood glucose levels have been associated with ED. There is a significantly raised risk of ED in patients with established CVD. Similarly, the development of ED may predate a cardiovascular event, and serve as a warning for the future development of CVD.

History taking should not only focus on cardiovascular risk factors that may contribute to the development of ED, but also aim to elicit other causes, whether psychological, psychosocial or physical. Blood pressure measurement and examination of the genitalia should be carried out and, if hormone therapy may be indicated, digital rectal examination. Patients should have a fasting plasma glucose measurement and testosterone levels should be measured if hypogonadism is suspected, or if the patient needs reassurance.

As the cause of ED can be multifactorial, both organic and psychogenic aspects should be addressed. If the predominant cause is psychogenic, the patient should be referred to a psychosexual counselling service. Lifestyle modification, such as weight loss and increased physical activity, has been shown to improve ED symptom scores and should be encouraged.

Oral therapy with a PDE5 inhibitor (sildenafil, tadalafil or vardenafil) may be initiated. PDE5 inhibitors are safe and effective drugs. However, they are absolutely contraindicated in patients taking nitrates or with hepatic impairment. PDE5 inhibitors are also contraindicated in patients who have recently had a stroke, myocardial infarction, retinal disorder or hypotension. If PDE5 inhibitors are ineffective or contraindicated alprostadil may be used either transurethrally or via intracavernosal injection. Some patients may benefit from the use of a vacuum device to obtain an erection. Patients for whom drug therapy is unsuccessful may consider insertion of a malleable or inflatable penile prosthesis.

If renal disease is suspected, renal function tests and urine dipstick analysis should be performed.

Abnormal liver function may be associated with ED, and therefore if risk factors for liver disease are elicited (eg alcohol misuse, chronic liver disease), liver function tests should be performed.

If testosterone therapy is to be initiated, PSA should be measured before commencing treatment as hormone supplementation/replacement may increase the risk of prostate cancer. However, the evidence supporting this link is extremely limited.

MANAGEMENT

Treatment options

As the cause of ED can be multifactorial, both organic and psychogenic aspects should be addressed. If the predominant cause is psychogenic, the patient should be referred to a psychosexual counselling service.

'Weight loss and increased physical activity have been shown to improve ED symptom scores and should be encouraged'

Treatment should tackle the cause as well as the symptoms, and it is therefore important to modify risk factors. Cardiovascular risk factors should be addressed and managed appropriately.

Lifestyle modification, such as weight loss and increased physical activity, has been shown to improve ED symptom scores and should be encouraged.[13]

It is important to understand the needs and expectations of patients and to tailor management accordingly.

Oral therapy with a PDE5 inhibitor (sildenafil, tadalafil or vardenafil) may be initiated. PDE5 inhibitors are safe and effective drugs. However, they are absolutely contraindicated in patients taking nitrates or with hepatic impairment. PDE5 inhibitors are also contraindicated in patients who have recently had a stroke, myocardial infarction, retinal disorder or hypotension.

A drug should be tried four times and the dose increased before it is considered ineffective. It may be helpful to consider an alternative PDE5 inhibitor before specialist referral is sought.

Certain patient groups are eligible to receive treatment with PDE5 inhibitors on the NHS. These include:

- Multiple sclerosis
- Parkinson's disease
- Polio
- Single-gene neurological conditions
- Spinal cord injury and spina bifida
- Diabetes
- Following kidney transplant (or in patients on dialysis)
- Following transurethral resection of the prostate, major pelvic surgery or injury.

Secondary care options

If PDE5 inhibitors are ineffective or contraindicated there are a number of further treatment options that may be offered in secondary care.

Alprostadil is a synthetic prostaglandin and may be used either transurethrally or via intracavernosal injection. When administered transurethrally, 69% of men reported an erection adequate for intercourse following use at home.[14]

Intracavernosal injection is also an effective treatment, achieving an erection in 70% of men. Patients and their partners report satisfactory sexual activity in 87% and 86% of cases respectively.[15] However, intracavernosal injections are an invasive treatment and up to 50% of patients suffer penile pain following injection, although this is usually mild.[16]

Some patients may benefit from the use of a vacuum device to obtain »

an erection. Reports of efficacy are varied, although in one study 67% of men found these devices effective.[17]

'The evidence that ED is a sentinel event for the development of CVD is increasing'

Patients for whom drug therapy is unsuccessful may consider insertion of a malleable or inflatable penile prosthesis.

CONCLUSION

Erectile dysfunction is a common condition and may be caused by a combination of psychogenic and organic factors. Care should be taken to identify and modify risk factors, especially for cardiovascular disease, and make lifestyle interventions. There is increasing evidence that erectile dysfunction is a sentinel event for the development of cardiovascular disease.[10]

Useful information

The European Association of Urology website contains their guidelines on erectile dysfunction
www.uroweb.org

The Sexual Dysfunction Association provides information and factsheets for patients and their partners
helpline: 0870 7743571
www.sda.uk.net

FURTHER READING

Ralph D, McNicholas T. UK management guidelines for erectile dysfunction. *BMJ* 2000;321:499-503

ACKNOWLEDGMENT

We would like to thank Mr Angus MacCormick, nurse specialist, Musgrove Park Hospital, Taunton for his advice in the preparation of this article.

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