

Deep vein thrombosis associated with distension of the urinary bladder due to benign prostatic hypertrophy - a case report

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ABSTRACT

A 76-year-old man was admitted with a first episode of deep vein thrombosis (DVT) of his left leg. It was associated with a distended urinary bladder, due to benign prostatic hypertrophy. Screening for malignancy was negative. Laboratory testing revealed protein S deficiency. Although a distended bladder may induce venous stasis, it is not a proven risk factor for DVT. Clinical expression possibly depends on the concomitance of other risk factors, such as inherited or acquired thrombophilic defects. However, it is also possible that the association of a distended bladder with DVT of a lower limb has not been recognised yet. As a distended bladder is rather common in elderly men, a proper study is warranted to estimate the prevalence of associated DVT.

INTRODUCTION

Deep vein thrombosis (DVT) can occur due to external compression of the pelvic veins by a malignant or benign tumour. Particularly in case of a benign tumour, venous stasis is the supposed mechanism to explain DVT.

However, benign tumours have rarely been reported in association with DVT.

A distended urinary bladder is diagnosed in 0.8% of elderly men annually¹ and may induce venous stasis.^{2,3} Remarkably, only three case reports have been published that described a possible relation with DVT.⁴⁻⁶ Here we describe another patient in whom a causal relationship between a distended bladder and DVT seemed to be plausible. However, the DVT in this patient could at least partly or even completely be explained by the concomitant presence of protein S deficiency.

CASE REPORT

A 76-year-old retired gynaecologist was admitted to our hospital with a one-day history of moderate swelling and a heavily prickling sensation of the left leg. His tentative diagnosis was DVT. It was confirmed by compression ultrasonography, which showed a thrombus in the left femoral and popliteal veins.

On the day of admission he developed acute urinary retention. He had accepted the mild micturation problems, which had already been present for a while, as normal for his age. Previous exposure to risk factors for venous thromboembolism had occurred when surgery for otitis media and sinusitis was performed at an age of 19 and 66 years, respectively. He was physically active and not obese. He had no history of either venous thromboembolism or any other disease. His mother experienced idiopathic DVT when she was 35 years old. She and one of her sisters died of colon carcinoma at an older age. The only abnormal findings on physical examination of the patient, apart from a swollen left leg, were a very enlarged bladder and a large prostate without palpatory signs of malignancy. The results of routine laboratory tests were within normal ranges, including haemoglobin level, white blood cell and platelet counts, and serum levels of electrolytes, urea nitrogen and creatinine. The D-dimer plasma level was 4804 µg/l (normal range 70 to 500), the serum level of prostatic specific antigen 2.4 µg/l (normal <6.5 µg/l). Abdominal echography revealed an overfilled urinary bladder without distension of the pyeloureteral systems. A Foley catheter was introduced suprapubically into the bladder and about 2.5 litres of clear urine was evacuated. Echography, chest X-rays and colon X-rays provided no evidence of a malignant tumour.

A transurethral resection of the prostate was performed four months later. Histology of the removed prostatic tissue showed benign hypertrophy.

Treatment of the DVT consisted of subcutaneous tinzaparin for seven days and acenocoumarol for three months. Because the patient was not convinced that venous stasis by the distended urinary bladder was a sufficient explanation for DVT, he requested laboratory testing of thrombophilic defects. Protein S deficiency was demonstrated at repeated measurements. Free protein S plasma levels amounted to 37 and 46%, respectively (normal range 76 to 120%) in samples that were collected four months after the acute DVT, when he was no longer receiving acenocoumarol. Plasma levels of antithrombin (87%), protein C (84%) and plasminogen (98%) were within normal ranges. Lupus anticoagulant and anticardiolipin antibodies were not detectable. DNA analysis did not reveal factor V Leiden or the prothrombin G20210A mutation.

DISCUSSION

A distended bladder can compress the iliac veins. Hopkins *et al.* demonstrated in six volunteers with normal bladder capacity (400-600 ml) that the pressure in the femoral veins rose significantly when the bladder contents approximated 300 ml, although variations were observed.² The pressure elevation was on average much higher in five patients with urinary retention due to benign prostatic hypertrophy and a bladder capacity that ranged from 1000 to 2200 ml. Three of these patients also had oedema of the lower limbs. They had the highest pressure elevation, which was even greater than 50 cm H₂O. Similar results were reported by Nilsson *et al.* during transurethral resection of the prostate when the urinary bladder was intermittently filled with irrigating fluid.³ In both studies, measurements were performed in supine position. Compression of the iliac veins due to bladder enlargement is expected to occur rather frequently in elderly men with benign prostatic hypertrophy. It may lead to oedema of one or both legs,^{7,8} comparable with oedema of the legs in women during the last trimester of pregnancy as a result of compression by the uterus.

Venous stasis is a generally accepted predisposing factor for DVT, as Virchow already postulated in 1865.⁹ It is therefore remarkable that only three cases have been reported since 1993, in which DVT of a lower limb was associated with a distended urinary bladder,⁴⁻⁶ as the latter condition is rather common in elderly men. Unfortunately, no information was given about other thrombotic risk factors in the three patients mentioned above. Based on the few cases reported in the literature, a distended urinary

bladder does not seem to be, or is only a mild, risk factor, clinical expression of which depends on the concomitance of other risk factors, such as the thrombophilic defect in our patient. Such a conditional causal relationship is in accordance with the current view that venous thromboembolism is often a multicausal disease.¹⁰ However, even then, a higher prevalence of DVT in elderly men would be expected, considering the increasing number of more or less prevalent inherited or acquired thrombophilic defects that have been identified as risk factors.¹⁰ One can therefore speculate about a protective mechanism against the development of DVT, such as an increased release of urokinase-type plasminogen activator from the distended bladder wall.¹¹ In conclusion, a distended urinary bladder may compress the iliac veins but is surprisingly very seldom associated with DVT of a lower limb. Apparently, other thrombotic risk factors are required to express venous thrombosis in this condition, such as the presence of protein S deficiency in our patient.

In our opinion, a prospective study is warranted to obtain an accurate risk estimate of venous thrombosis in consecutive patients with an urinary bladder distension to assess its clinical implications.

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