

Subclavian vein stenosis and axillary vein 'effort thrombosis'

Age and the first rib bypass collateral, thrombolytic therapy and first rib resection

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Summary

Three patients presented with axillary vein 'effort thrombosis'. Intravenous streptokinase for 3 days followed by heparin for 10 days restored patency and relieved symptoms. Pretreatment diagnosis and the effect of streptokinase were confirmed venographically and an abnormality in the subclavian vein just medial to the first rib was demonstrated. This stenosis was most severe and had a prominent bypass collateral in the oldest patient. It is proposed that, in the absence of superimposed thrombosis, the damage to the vein in predisposed patients is progressive with age, with establishment of permanent collaterals. Transaxillary first rib resection is advised to prevent compression of the veins in the thoracic outlet.

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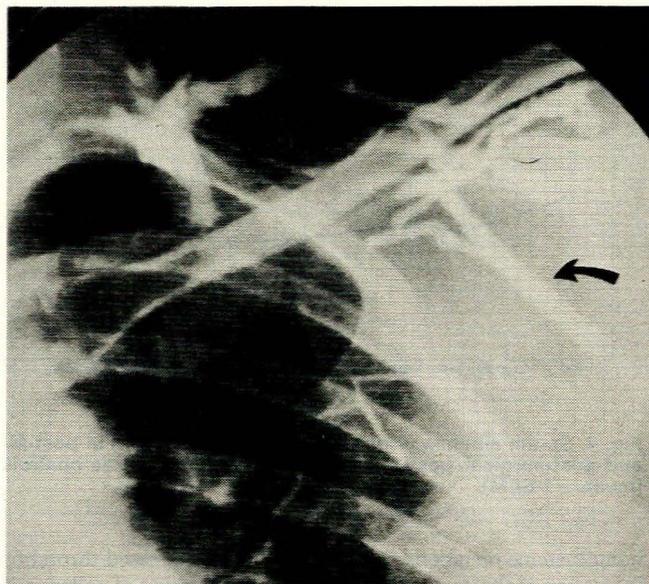


Fig. 1. Axillary vein thrombosis with prominent bypass collateral in a patient aged 42 years (cephalic vein arrowed).

The natural history and optimal management of patients with axillary vein thrombosis is controversial although a pattern of causes and sequelae is emerging.¹ Where several patients with a rare condition have been seen, it is worth documenting their management to stimulate discussion.

Patients and methods

Three patients were seen with axillary vein thrombosis. Two were aged 19 and 24 years and were seen within 2 days of the onset of swelling and cyanosis of an arm associated with distended visible veins over the shoulder. The third patient, aged 42 years, had had discomfort in the axilla for 10 days before the onset of clinical features of axillary vein thrombosis. A venogram confirmed the presence of occlusive axillary vein thrombosis in all 3 patients, but in the 2 younger patients minimal collateral veins were noted around the obstructed axillary vein. The oldest patient had extensive thrombosis of the brachial and axillary vein together with a prominent collateral vein (Fig. 1).

All 3 patients received thrombolytic therapy (streptokinase 600 000 U loading dose, followed by 100 000 U hourly intravenously in the non-affected arm) for 3 days, becoming asymptomatic within 24 hours, followed by intravenous heparin for 10 days. The post-thrombolytic venogram, done before discontinuing

the heparin, confirmed the restored patency of the axillary and the subclavian veins and again showed a distinct difference between the oldest and the two younger patients. The 42-year-old patient had a tight stenosis in the subclavian vein just medial to the first rib, with a persistent prominent first rib bypass collateral (Fig. 2). The two younger patients had only a minor irregularity in the subclavian vein at the medial border of the first rib with no prominent collateral vein noted (Figs 3 and 4).

The patients were discharged without further anticoagulant therapy and first rib resection was advised. This was declined by the 24-year-old patient who was still well 1 month after discontinuing heparin therapy. The other 2 patients had transaxillary first rib resection done within 1 month of discontinuing the heparin therapy and were asymptomatic 1 month after surgery apart from a small numb area behind the affected shoulder in 1 patient.

Discussion

Axillary vein thrombosis is a rare form of deep-vein thrombosis and in the absence of a clear-cut cause or disease process such as direct trauma to the vein, use of an intravenous cannula or carcinomatous compression of the subclavian vein, the term 'effort thrombosis' has been used.^{1,2} The implied cause is mechanical venous compression in the thoracic outlet. Once the obstruction to venous return becomes significant, symptoms appear but intermittent non-thrombotic mechanical obstruction can also occur, so venographic confirmation of thrombosis is essential.^{1,3,4}

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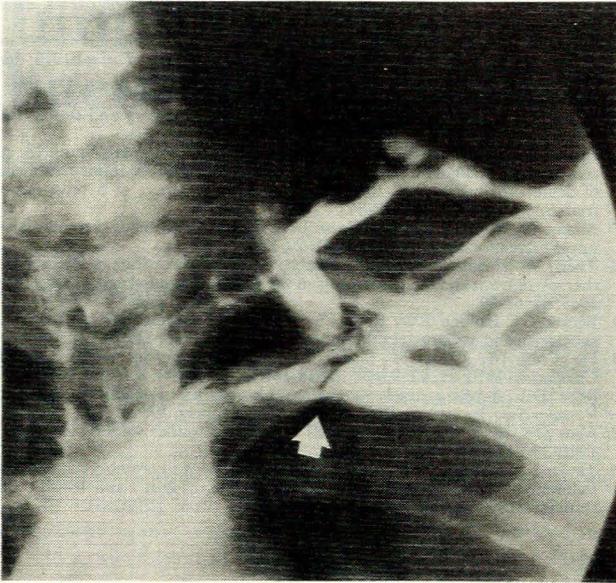


Fig. 2. Subclavian vein stenosis after thrombolytic therapy in a patient aged 42 years with a persistent prominent bypass collateral.

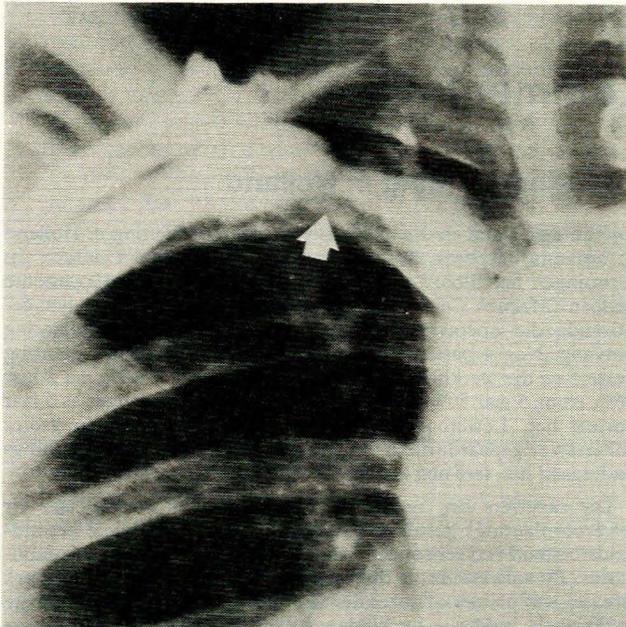


Fig. 3. Subclavian vein stenosis in a patient aged 19 years.

Our patients fell within the described age range of 17 - 68 years and presented with the typical symptom complex.^{1,5} The full spectrum of sequelae of deep vein thrombosis may occur in these patients and, although extension may be prevented, resolution of the thrombus is unlikely with anticoagulant therapy.⁶ Venous return will depend on flow through collaterals such as the 'prominent first rib bypass collateral' described by Adams *et al.*⁷

Patients with repetitive mechanical obstruction, which involves the collaterals, are most likely to have long-term disability after axillary vein thrombosis and may respond to thoracic outlet decompression.¹ For this latter group acute thrombectomy has been proposed but seldom done, and thrombolytic therapy to clear the recently occluded axillary

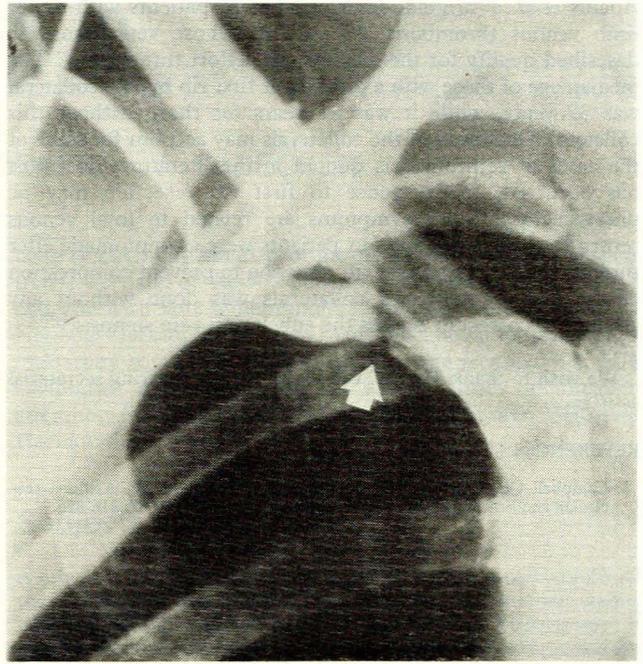


Fig. 4. Subclavian vein stenosis in a patient aged 24 years.

vein is justified.^{1,5} One of our patients had extensive occlusive thrombosis with a long history before diagnosis, and where the long-term consequences of the thrombus may be grave a liberal policy in using thrombolytic agents would seem to be correct.⁸ The post-treatment venogram was important to prove patency, to establish any source of extrinsic compression other than in the costoclavicular space, and to note any intrinsic stenosis of the vein at the first rib.^{1,3,4} Stenosis, with perivenous fibrosis and internal synechiae noted at thrombectomy, at the point of compression between the clavicle and the first rib has also been described after thrombolytic therapy.^{9,10}

Having succeeded in clearing the veins, the risk of recurrent thrombosis and a plan of action had to be considered — thrombectomy, repeat thrombolytic therapy or anticoagulation with later management of an established post-phlebotic syndrome.^{1,5,11} The risk of clinical 'recurrent thrombosis' in patients treated with heparin or elevation would seem to be small, and any episode may be due to thrombosis of collaterals rather than rethrombosis of a recanalised axillary vein. Since there is nothing in the literature which addresses this problem after thrombolytic therapy, it seemed prudent to decompress the thoracic outlet prophylactically on the affected side, using an approach with minimal cosmetic deficit.^{1,12} Since the incidence of bilateral 'effort thrombosis' is low and the incidence of subsequent symptoms in patients with radiographically demonstrable venous compression or intrinsic stenosis is unknown, contralateral venography and first rib resection was not advised.^{1,3,5,9}

Although all 3 patients treated with streptokinase had a demonstrable residual abnormality in the vein at the thoracic outlet, only 1 had a stenosis which appeared tight, with a prominent collateral in spite of the patent axillary and subclavian vein. The pressure gradient across this stenosis was not measured. To what extent such changes precede axillary vein 'effort thrombosis' will become apparent as more cases are described in which thrombolytic therapy has been used. One could easily postulate that, in the absence of superimposed thrombosis, the progressive narrowing of the distal subclavian vein in predisposed patients is a factor of age, with the development of permanent enlargement of collateral veins. In

Adams *et al.*'s⁷ original description of 25 patients with upper limb venous thrombosis, 13 patients whose venograms are described qualify for the diagnosis of 'effort thrombosis'. The median age of those with a prominent first rib bypass collateral was 39 years, while it was 21 years for those without this collateral. Occlusion of the collaterals may account for some of the cases of rethrombosis quoted in the literature; once this has occurred, the response to first rib resection may be unsatisfactory. Since symptoms are related to total venous return from the arm, and our patients were asymptomatic after thrombolytic therapy, first rib resection to prevent compression of the axillary vein and collaterals was done without any attempt to correct or bypass the subclavian vein stenosis.^{1,9,13}

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News and Comment/Nuus en Kommentaar

Explaining medicine to the media

From time to time there have been clashes between the medical profession, and indeed the scientific community, on the one hand and the news media on the other hand, through failure of each party to understand the other's point of view. Some readers may recall that in 1969, after a certain amount of fluttering in the dovescotes in relation to the first heart transplant, Dr Pieter van Biljon, then editor of the *SAMJ*, arranged a conference in Pretoria to bring the warring parties together to try to improve matters. Over the years, this conference probably achieved slightly more than the recent confrontation at Reykjavik between the Soviet and American leaders.

Nevertheless, things are not nearly as bad in South Africa in respect of science reporting as they apparently are in Britain.

Last October Sir Walter Bodmer, Chairman of an *ad hoc* group set up by the Royal Society to report on the public understanding of science, claimed in his Bernal Lecture to the Royal Society that much of the responsibility for this state of affairs lies with the scientists. One good suggestion he made was that all Ph.D. candidates should be required to explain their research to a lay audience. But the media are also not blameless; according to the lecturer, the *ad hoc* group found that editors of almost all major national newspapers in Britain were not interested in science coverage. Another problem is that interviewers on television in particular tend to turn the programme into a Perry Mason-type court room scene, which certainly stimulates the public but does not always shed light on the controversial and difficult points of the programme. However, in Britain there are some steps being taken in the right direction. The Royal Society is now considering the appointment of a full-time public relations officer solely to explain science to the media while the Ciba Foundation has set up a Media Resource Service as a free referral service for the media. But communication is a two-way affair and the media must themselves do something to recruit personnel with at least a basic understanding of the way that science, including medical science, works and a realisation that the areas under discussion cannot be presented in terms of black or white but are still in various shades of grey.

Leweroorplanting in Holland

Die huidige stand en die toekoms van leweroorplanting in Holland is onlangs bespreek in twee artikels in *Medisch Contact*. In Groningen het Slooff *et al.* 54 leweroorplantings op 49 pasiënte gedoen (*Medisch Contact* 1986; **41**: 1552). Vier pasiënte het gedurende die operasie gesterf en 5 het 'n tweede oorplanting ontvang. Nog 4 pasiënte het binne 'n maand na die oorplanting gesterf en die werklike oorlewingsyfer na 1 jaar is 60%, na 2 jaar 56%, en na 5 jaar 50%. Tans is daar 11 pasiënte wat langer as 5 jaar oorleef het. Leweroorplantings by kinders gaan met 'n groter suksesyfer gepaard; die 6 kinders wat in Groningen oorplantings ondergaan het, leef nog almal.

Die skrywers het 'n boonste ouderdomsperk van 60 jaar vir leweroorplantings gestel en die pasiënt moet in 'n goeie gesondheidstoestand verkeer sonder ernstige kardiaale of pulmonale komplikasies. By volwassenes is die belangrikste funksionele aanduidings sirrose, wat primêr of sekondêr kan wees, terwyl die algemeenste funksionele indikasie by kinders biliêre atresie is. Kwaadaardige tumore van die lewer bied besondere probleme, met voorkeur vir gedeeltelike reseksie van die lewer waar moontlik.

In 'n tweede artikel deur Terpstra (*Medisch Contact* 1986; **41**: 1623) word die probleem van aanvullende of heterotopiese leweroorplanting bespreek. Soos by 'n abhartoerplanting, is die idee om 'n versakende lewer te help eerder as om die funksie daarvan te vervang. Hoewel hierdie aangeleentheid reeds jare lank bestudeer word, is daar slegs 2 gevalle aangeteken waar pasiënte onderskeidelik 7 en 13 jaar na 'n aanvullende leweroorplanting met 'n atrofiese oorspronklike lewer geleef het.

Die prosedure het nooit verder as die eksperimentele fase ontwikkel nie en verdere ondersoek word in Rotterdam beoog. Die grootste probleem is die gebrek aan ruimte in die buikholte, die ontoereikende bloedvoorsiening aan die oorgeplante orgaan en veneuse stase in die oorgeplante orgaan. Om die ruimtelike probleem te oorbrug word voorgestel dat slegs 'n gedeelte van die skenkerlewer oorgeplant word, en dat spesiale aandag geskenk moet word aan pasiënte wie se lewerfunksie besig is om te verswak, maar by wie die proses van versaking nog omkeerbaar is.