Late complications of surgery for coarctation of the aorta

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Ross, J. K., Monro, J. L., and Sbokos, C. G. (1975). *Thorax*, 30, 31–39. Late complications of surgery for coarctation of the aorta. The problem of the patient who has had one operation for coarctation of the aorta and who then requires another because of a late complication at or near the coarctation site is a demanding one. The safety of aortic cross-clamping at the second operation depends on the adequacy or otherwise of the collateral circulation, and this in turn depends on the presence or absence of residual or recurrent aortic obstruction. Three illustrative cases are described in which there was complete, incomplete, and no aortic obstruction respectively at the time of reoperation, two of the cases presenting the additional complication of local aneurysm formation. The various aspects of management of such individuals are discussed, and the relevant literature has been reviewed in an attempt to provide a systematic approach to these difficult patients.

The methods for assessing collateral circulation are both clinical and radiological with trial clamping of the aorta and pressure measurement as the most reliable ultimate test. A pressure of 50 mmHg in the distal aorta is accepted as indicating an adequate peripheral circulation, but it is recommended that the trial clamping should always include both the left subclavian artery and any particularly large local collaterals. The use of a perfusion technique to sustain the distal tissues is also recommended, although local bypass shunts have a place when their use is dictated in the interests of safety for the patient.

From the early days of coarctation surgery late complications have been recognized and recorded (Owens and Swan, 1963). These have been related to infection (Martin, Kirklin, and DuShane, 1956; Roesch and Bond, 1960; Oldham *et al.*, 1973), unfavourable anatomy, inadequacies of technique, and, in small children, the problem of growth failure at the suture line (Parsons and Astley, 1966).

The question of late reoperation is often not an easy one and the second operation itself can be difficult and dangerous. Apart from the local hazards in the region of the previous repair, which can include aneurysm formation, dense adhesion to surrounding structures, and abnormal thinning of the aortic wall (Cerilli and Lauridsen, 1965), the most serious risk in reoperation is that of spinal cord damage and consequent paralysis (Brewer *et al.*, 1972). This risk is related to the efficiency of the collateral circulation when the aorta is clamped or to the efficiency of the method

chosen to protect the distal tissues when aortic blood flow is interrupted. The state of the collateral circulation itself at the time of the second operation reflects the presence or absence of residual or recurrent aortic obstruction and its duration and degree when present.

This communication describes the problem presented by three patients, all requiring late second operation, who had respectively total, partial, and no obstruction to the aortic lumen at the time of their reoperation. The three patients represent, therefore, both in the local condition at the coarctation site and in the varying degree of aortic obstruction an almost complete illustration of the problems which have to be faced when a second operation is needed.

PATIENTS

CASE 1 M. K. aged 35. Coarctation of the aorta was discovered during the course of investigation for a urinary tract infection at the age of 22.

At the first operation, undertaken at another hospital in August 1961, a one-inch narrowed segment of aorta was resected and replaced by a tubular Teflon prosthesis. The femoral pulses did not reappear post-operatively and the patient experienced pain in the legs and an unusual amount of dyspnoea. She subsequently had two successful full-term pregnancies.

Twelve years after her first operation she presented at the Cardiothoracic Unit in Southampton, still with dyspnoea and severe hypertension (230/110 mmHg) in the upper half of the body. The femoral pulses were still absent, there was no coarctation bruit, and aortography demonstrated total obstruction of the aorta immediately below the left subclavian origin with late filling of the descending aorta and very large collaterals. The appearances were those of occlusion of the Teflon prosthesis (Figs 1a and b).

Reoperation (January 1973, JKR). At left thoracotomy the occluded and solid Teflon prosthesis was mobilized without difficulty and removed between clamps (Fig. 2a), aortic continuity being restored using a 20 mm diameter crimped Dacron prosthesis. During the mobilization process it had been noted that the ligamentum arteriosum was still intact and examination of the resected specimen suggested that the upper end still contained part of the original coarctation (Fig. 2b). Postoperative recovery was uneventful and one year later she was found to have full and equal femoral pulses, a blood pressure of 160/100 mmHg and a diminished heart size on the plain chest radiograph.

CASE 2 S. O. aged 13. This child was found to have a cardiac murmur at birth and was thought to have a persistent ductus arteriosus and coarctation of the aorta. This clinical diagnosis was confirmed at the age of 5 years. At the first operation in March 1968, a 5 mm ductus was ligated, the coarctation was resected, and continuity of the aorta was restored by end-to-end anastomosis using continuous and interrupted 5/0 sutures. Opinions varied about the state of the femoral pulses after operation but 18 months later they were clearly diminished and delayed, and a shadow had developed in the left upper mediastinum which was noticeably widened in the early postoperative chest films. The shadow was continuous with the aortic shadow and soon developed evidence of calcification (Figs 3a and b). A calcified false aneurysm at the coarctation site was confirmed at aortography in 1972. There was also a gradient of 40 mmHg between the aorta proximal and distal to the aneurysm. The blood pressure was 130/85 mmHg in both arms and there was loud systolic bruit to be heard both at the back and the front of the left upper chest. Plain chest radiography at this time showed that the aneurysm was static in size. Review of the aortograms suggested that the aneurysm did not involve the origin of the left common carotid artery and that it had become secondarily adherent to the left subclavian artery whose lumen was also intact. Collateral vessels were

(a)

(b)

FIG. 1. Case 1. (a) Ascending aortogram showing complete aortic occlusion at the level of the origin of the left subclavian artery. (b) Large collateral vessels shown on aortography in a later film in the series.

FIG. 2. Case 1. (a, b) The resected occluded Teflon prosthesis. A black ligature marks the point of attachment of the ligamentum arteriosum. The appearances seen end-on suggest that some of the original coarctation is still present.

seen to be well developed, and it appeared possible that the aneurysm itself could have been partly responsible for the aortic narrowing by compression from without.

Reoperation (October 1973, JKR). At left thoracotomy the lung was mobilized off the aneurysm and control of the distal arch, left subclavian, and descending aorta was obtained. Preparations had been made for left atrial-femoral bypass but on trial clamping of the aorta and left subclavian artery proximal to the aneurysm the mean pressure only fell from 80 to 60 mmHg in the distal aorta. This was considered sufficient to proceed without bypass and the aneurysm was uneventfully resected (Figs 4a and b) and aortic continuity restored using a 3 cm length of 18 mm diameter crimped woven Dacron tube. Postoperative recovery was uneventful with return of normal femoral pulses.

CASE 3 A. D. aged 43. Seventeen years before admission to the Cardiothoracic Unit at Southampton this patient had undergone surgery for coarctation of the aorta at another hospital. Six months before admission he had begun to develop hoarseness. A small polyp was removed from one vocal cord but the hoarseness persisted and a subsequent chest radiograph showed an aortic aneurysm (Fig. 5a). At this time his blood pressure was 140/90 mmHg, he had no murmurs, and his femoral pulses were normal. Aortography confirmed the diagnosis although the exact way in which the aneurysm communicated with the aortic lumen was not clear (Fig. 5b).

Reoperation (January 1974, JLM) At left thoracotomy the aneurysm was exposed by dividing vascular adhesions and mobilizing the lung. Proximal and distal control was obtained by passing tapes round the aorta above and below the aneurysm. Femoral vein to femoral artery perfusion (with a Temptril oxygenator in the circuit) was then started and a flow of 1.7 l/min was maintained in the lower half of the body while the aorta was cross-clamped. The aneurysm proved to be a fusiform dilatation of the whole area without a discrete neck. It was incised longitudinally and a 16 mm crimped woven Dacron tube prosthesis was put in, after which the residual aneurysm wall was sutured over it. The postoperative recovery was complicated only by slow re-aeration of the left lung, and the patient left hospital on the eleventh day following surgery. After operation an account of the first procedure was received which stated that a piece of homograft aorta had been used to reconstruct the aorta after resection of the coarctation. The homograft had been sterilized with beta propiolactone and freeze-dried.

DISCUSSION

Reasons for reoperation in our three patients need not cause much debate. In the first case a tubular prosthesis became blocked immediately after it was put in and the evidence suggests that the original coarctation was incompletely excised. It is reasonable to suppose that this caused reduced flow through the Teflon tube, which then rapidly thrombosed. It is interesting that thrombotic occlusion of the aorta had been described after resection of a coarctation (Cerilli and Lauridsen, 1965) without prosthetic reconstruction.

In our second case there is little doubt that a suture line leak led to a peri-aortic haematoma which then became organized into a false aneurysmal sac. Infection, although not a likely cause, cannot be entirely excluded. The calcification in the aneurysmal wall may have helped to prevent the aneurysm from growing larger.

(a)

(b)

FIG. 3. *Case 2. (a) PA and (b) lateral chest radiographs showing calcified multilocular false aneurysm at site of previous coarctation resection.*

FIG. 4. Case 2. (a) The resected specimen opened to show narrowing of aortic lumen and false aneurysm containing clot. (b) Radiograph of intact specimen with probe lying in residual aortic lumen.

(a)

(b)

FIG. 5. *Case 3. (a) PA chest radiograph showing aortic aneurysm. (b) Aortogram confirming the presence of a large fusiform aneurysm.*

In the third case, the fusiform aneurysm was due to degenerative change in the segment of chemically sterilized, freeze-dried homograft aorta inserted at the primary operation. Aneurysmal dilatation of a Dacron graft has also been reported (Stankowiak and Soots, 1973).

Aneurysms can occur in the region of a coarctation repair due to infection on the suture line (Martin *et al.*, 1956; Roesch and Bond, 1960; Davis, Fell, and Taylor, 1965) when the problems of management have much in common with the spontaneously occurring mycotic aneurysm (Khazei and Cowley, 1967; Oldham *et al.*, 1973) although the unpredictable early regression of collaterals should influence the surgical tactics in the postoperative variety. Aneurysms can also occur at the site where a clamp was applied to the aorta (Gazzaniga, 1966) or can involve the area of the suture line without identifiable aetiology (Blickman, 1949; Frederiksen, Poulsen, and Davidsen, 1961).

The presence of an aneurysm, provided that it is not infected, is less important than the problem of the reliability of the collateral circulation when a second operation is needed, and this in turn depends on the degree of aortic obstruction present. If, following any type of operation for coarctation, complete obstruction of the aortic lumen remains (as in case 1), there can be no doubt that if it was considered safe to clamp the aorta at the first operation it will be so at the second. Any collaterals that had to be divided at the first procedure represent the only difference between the two preoperative situations. Conversely, if there is no aortic obstruction (as in case 3), it must be assumed that there are no effective collaterals. It is the intermediate type of case that represents the real problem, when the recurrent or residual obstruction is incomplete (as in case 2). This is typified by the patient whose primary repair was in infancy (Parsons and Astley, 1966; Khoury and Hawes, 1968; Ibarra-Pérez *et al.*, 1969; Tawes *et al.*, 1969; Eshaghpour and Olley, 1972) but the uncertainty about how much regression and re-establishment of the collateral circulation has occurred applies to all individuals in this category regardless of their age at the primary procedure.

PREOPERATIVE ASSESSMENT OF COLLATERAL CIRCULATION Clinical assessment of a collateral circulation may be difficult or misleading unless there are obviously palpable chest wall vessels combined with diminished and delayed or absent femoral pulses. Conversely, easily felt femoral pulses and a

loud coarctation bruit, indicating less important aortic obstruction, must raise doubts about the state of the collateral supply.

Radiological assessment is more valuable but demands high quality aortography with sufficient time lapse during the study for the collateral vessels to fill and be clearly demonstrated.

The velocity of flow in superficial collaterals may be measured by the Doppler method (Ben-chimol, Baldi, and Dessler, 1973) but this technique has not yet been used enough for one to be sure of its usefulness.

A study of the records of the first operation and the patient's postoperative course can give some indication of both the duration and the severity of any residual or recurrent aortic obstruction and, therefore indirectly, of the likely state of the collateral circulation.

ASSESSMENT OF COLLATERAL CIRCULATION AT OPERATION An estimate of the size and number of collateral vessels may be made as the chest is opened, and failure to encounter the characteristic vessels in the chest wall may be the first indication that the collateral circulation is poor. If, as is likely, the chest is being reopened in the line of the previous incision, this may not be a valid sign.

Trial clamping of the aorta with measurement of intra-aortic pressure proximal and distal to the clamp (Hughes and Reemtsma, 1971) is accepted as a method of judging the efficiency of the collateral circulation. A mean pressure of 50 mmHg distal to the clamp is regarded as the critical pressure below which some form of circulatory support is needed for the lower half of the body. Ideally, this manoeuvre should be combined with estimates of blood flow in the descending aorta, and this would be a logical extension of the flow studies reported by Schenk, McDonald, and Andersen (1965). We agree that trial clamping is a valuable manoeuvre and that a pressure of 50 mmHg (mean) is an acceptable arbitrary level to set, but the method should routinely include occlusion of the left subclavian artery and also of any particularly large collaterals entering close to the coarctation distally which would have to be ligated or clamped during the subsequent repair. We also feel that although trial clamping may be regarded as the most precise way of assessing the adequacy of the collateral circulation, it is dangerous to base the judgement of the safety of aortic cross-clamping on a single parameter. The final decision must therefore take into account all the available clinical and radiological

evidence and must always err on the side of safety if there is the slightest doubt, although this point of view would not be shared by Crawford and Rubio (1973), who advocate aortic cross-clamping at normal temperature without the use of shunts or circulatory support when treating aneurysms of the descending thoracic aorta.

There are various methods available for protecting the tissues distal to the site of aortic clamping, assuming that preservation of intercostal and other collateral vessels is an integral part of the surgical technique. Here the technique of plastic reconstruction of the aorta described by Vosschulte (1961) has much to recommend it as the need for extensive mobilization of the aorta, and therefore the threat to collateral vessels, is much reduced. Some favour hypothermia (Dubost, cited by Brewer *et al.*, 1972) as a routine adjunct to all secondary procedures. Others use temporary or permanent bypass shunts electively in such instances (Lam and Arciniegas, 1973; Skagseth and Froysaker, 1973; Weldon *et al.*, 1973), and in difficult cases the technique described by Weldon, which does not involve aortic cross-clamping, is attractive. It is possible, however, that the abnormal thinning of the aortic wall which may be found at secondary procedures make these methods difficult to apply universally with safety.

In common with many other authors, we favour the use of a perfusion technique, either left atrial-femoral bypass (Gerbode *et al.*, 1957; Braimbridge, 1963) or veno-arterial femoral bypass (Neville *et al.*, 1968), not only as a safe and flexible method which ensures an adequate and measurable blood supply to the lower half of the body but as a means of decompressing the proximal circulation when the aorta is cross-clamped. Further, we would agree with the opinion expressed by others (Hughes and Reemtsma, 1971) that such facilities should always be available when surgery of this kind is undertaken.

Spinal cord complications with coarctation of the aorta have recently been well reviewed (Brewer *et al.*, 1972) together with the vagaries of the spinal cord's blood supply and the factors considered important in causing and preventing spinal cord damage. It is clear that no method for protecting the spinal cord at primary or secondary operation can be regarded as totally safe, and the fact that coarctation can be associated with spontaneous cord lesions without surgery emphasizes the unpredictable nature of the disease complex. Perfusion support for the distal circulation represents the most reliable method

available for protection of the tissues beyond the level of aortic cross-clamping. Temporary or permanent local bypass shunt procedures, which can be made without aortic clamping, also have a place, particularly when mobilization of the aorta proximally and distally is likely to be more dangerous than the introduction of the shunt.

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