Occlusion of radicular arteries—reasons, consequences and anastomotic substitution pathways

M Maliszewski^{*1}, P Ladziński¹, R Aleksandrowicz², H Majchrzak¹, G Bierzyńska-Macyszyn³ and J Wolańska-Karut³

¹Department of Neurosurgery, Mining Hospital, Sosnowiec, Poland; ²Department of Anatomy, Medical Academy of Warsaw, Chalubinski Str. 5, Warszawa, Poland; ³Department of Pathology, Silesian Medical Academy, Katowice, Medyków Str. 12, Poland

Aim of study: To investigate the cause of radicular artery occlusion.

Method: Fifty specimens of spinal cords were taken at postmortem from individuals in whom no symptoms of spinal cord and spine injuries were present in life. The spinal cord arterial system was filled with colour contrast material and then each radicular artery was studied individually at 5–25 operative microscope magnification.

Results: Two hundred and fifty two anterior radicular arteries joining the anterior spinal artery and 525 posterior radicular arteries joining the posterior spinal arteries were examined. There were five obstructed vessels: four were anterior radicular arteries including the artery of Adamkiewicz and one of the initial branches of the spinal artery originating from the vertebral artery.

Conclusions: As the presented examples show, the formation of a fully efficient collateral circulation is possible.

Keywords: radicular arteries; spinal cord ischaemia; substitution pathways

Introduction

Occlusion of radicular arteries is the most frequent reason for spinal cord ischaemia.¹ Disseminated fibrinoid and hyaline degenerations of small arterial vessels occur with almost the same frequency, mainly within the pial arterial plexus, developing at the age of senility.^{1–5}

Occlusion of radicular arteries may originate as a result of occlusion by thrombus (in cases of atherosclerosis,⁶ polyarteritis nodosa,⁷ trauma or metastases)³ or by emboli: with atheromatous masses, nucleus pulposus fragments or thrombi during endocarditis and valvular heart disease.⁶ Radicular arteries may be also compressed by spinal metastatic neoplasms, paraspinal tumours, in cases of spinal fractures,^{3,6} or from a high degree of spondylosis or side prolapse of the nucleus pulposus.^{3,6–10} Radicular arteries can be also damaged during operative procedures.¹⁰

Such situations are observed extremely rarely and the fact that epidemiological research has not been carried out yet and that the presentation of the phenomenon has been limited to individual reports,¹¹ should not be surprising.

The frequency of spinal vascular lesions in postmortem material is estimated at 0.25%–3% and in individuals of over 60 years it can reach 8%.^{1,3,11}

Comparing frequencies of the occurrence of ischaemic cerebral and spinal cord strokes, it appears that the latter occur ten times less frequently.³ The reasons can be investigated first of all in the specific organisation of the spinal cord arterial system.

Based on our observations we present cases illustrating the presence of efficient collateral circulation in radicular artery occlusion.

Materials and methods

Fifty specimens of spinal cords were taken at postmortem from individuals in whom no symptoms of spinal cord and spine injuries were present in life. There were 19 women and 31 men. Their age ranged between 20 and 80 years, (Table 1).

The spinal cord arterial system was filled with colour contrast material and then studied in detail under an operating microscope. Every effort was made to separate the spinal cord from the cadaver not later than 24 h after death. An incision of the dura mater and the arachnoidea was performed along the medial line at the anterior and posterior surface of the dural sac. The spinal cord was then placed in a water bath at 20°C for about 2 h, after which it was transferred to a water bath at 40°C. The vertebral arteries were ligated extradurally at their passage through the dura. Contrast medium catheters were inserted into the

*Correspondence: M Maliszewski, Department of Neurosurgery, Mining Hospital, Sosnowiec, PO Box 168, Poland

basilar artery and into 3–5 anterior radicular arteries of more than 600 μm in diameter. After a few unsuccessful attempts, no perfusions of the arteries with normal saline were made before injecting the contrasting medium because the ‘preliminary’ perfusion caused damage of the very long and minute arterioles and impaired the process of filling the arteries with the contrasting medium.

Two types of contrasting medium were used: aqueous solution of Chinese ink heated to 50°C (36 specimens) or ethylene ether suspension of soot: 1 volume unit of soot per 3 volume units of ethylene ether (14 specimens). In seven cases the process of inserting the contrast was performed in a vacuum chamber at -0.1 at. The contrasting material was advanced by syringes, their pistons being moved by a micrometer screw.

The specimens were fixed and kept in 10% formaldehyde solution. Each radicular artery was studied individually at 5–25 operative microscope magnification. The following parameters were analyzed: the place of the penetration into the dural sac, the artery accompanying the radix, the level of the occurrence of the artery at the spinal cord, the type and function of branches, the location of the final branches, and the diameter of the trunk and branches.

Five occluded radicular arteries were embedded in paraffin and cut perpendicularly to their axes. The transverse cut sections were routinely stained with hematoxylin and eosin. To differentiate structural elements, we performed the following selective stainings: Masson trichrome, resorcin-fuchsin and van Gieson staining.

Results

Two hundred and fifty two anterior radicular arteries joining the anterior spinal artery and 525 posterior radicular arteries joining the posterior spinal arteries were encountered in the examined specimens. Among 777 radicular arteries, four vessels (0.51%), all of which were anterior radicular arteries of 150–1000 μm in diameter, were obstructed.

In two cases (specimens 1 and 4) atherosclerotic thrombi (Figures 1 and 2) were found, while in one case (specimen 2), the cause of the occlusion was degenerative hyperplasia of the intima and media which almost completely narrowed the arterial lumen (Figure 3). In specimen 3, where a small radicular artery 150 μm in diameter was occluded, we found hyperplasia of the intima, especially in the subintima,

and of the media with a significant thickening of the wall and narrowing of the lumen (Figure 4).

In another case (specimen 5) one of the initial branches of the anterior spinal artery, was obstructed by atheromatous embolic material (Figure 5). These obstructed vessels were detected in five specimens (10%). The specimens originated from three women and two men, who died at the age of 58–73 years. In three cases the atherosclerosis of cerebral vessels was an indirect cause of death. At the sides of occlusion the arteries were of fusiform shape at the length of 0.5–5 mm. Distal and proximal sections of the arteries

Figure 1 Case 1 (A) A diagram representing an obstructed artery of Adamkiewicz. e: place of occlusion. (B) Thrombus in the initial phase of formation and recanalisation in the arterial lumen. Magnified 300 times

Table 1 Age of the examined patients

Sex	Age (years)						Total
	20–30	31–40	41–50	51–60	61–70	71–80	
Men	1	3	4	7	13	3	31
Women	1	0	2	3	5	8	19
Total	2	3	6	10	18	11	50

Figure 2 A photograph of the specimen no. 4 (A) with an explanatory diagram alongside. (B) An obstructed anterior radicular artery on level L3 on the right side. asa: anterior spinal artery; cm: conus medullaris; e: place of occlusion. (C) Internal elastic membrane with intense elastosis. The lumen overgrown by the connective tissue. Visible recanalisation. Magnified 250 times

Figure 3 Case 2. Considerable narrowing of arterial lumen due to degeneration and hyperplasia of the intima and media. Magnified 300 times

in relation to the place of occlusion—except for case 4—were unobstructed. It should be stressed that in all cases the occlusion was situated directly past penetration of arteries into the dural sac—from 2.5–5 mm from the dura mater puncture.

The level of the occurrence of obstructed arteries as well as adjacent arteries, diameters of main trunks and final branches and location of side of occlusion are presented in Table 2. This table presents also the initial cause of death confirmed by a pathomorphological examination. In this examination the fact of generalised atherosclerotic changes in arterial vessels drew our special attention.

Discussion

According to a number of reports occlusion of a large radicular artery leads to myelomalacia, which is irreversible and may lead to fatal results.^{3,6,8,9,12} This

Figure 4 A photograph of the specimen no. 3 (A) with an explanatory diagram alongside. (B) An obstructed anterior radicular artery joining the anterior spinal artery on level D5. asa: anterior spinal artery; e: place of occlusion. (C) Degenerative hyperplasia of the intima, especially in the subintima with a significant thickening of the wall and narrowing of the lumen, which is typical hyperplasia due to senescence and atheromatosis. Magnified 300 times

effect is caused by the lack of efficient paths of collateral circulation. According to Jellinger³ and Lazorthes *et al*¹³ anastomoses of arterial tributaries occurring both outside and inside the spinal canal are of little functional importance although they should not be underestimated. The anterior spinal artery is often discontinuous in the central thoracic area, where a so-called critical zone exists, and where the pial arterial plexus is least developed and consequently it cannot be considered as an efficient path of collateral circulation. Lazorthes *et al*¹³ claim that possibilities of substitution are significantly greater if

the site of an arterial occlusion is further from the spinal cord, that is, the closer it is to the main arteries—the aorta or vertebral arteries. Contraction or occlusion of intercostal, lumbar or vertebral arteries provide a greater chance of development of efficient collateral circulation than when a radicular branch is affected. These authors claim also that the slower the rate of artery occlusion, the greater is the chance of effective ‘intervention’ of substitute circulation paths, while with sudden occlusion of an artery the development of efficient substitution is unexpected.

Figure 5 Case 5. (A) A diagram representing obstruction in one of the initial branches of the anterior spinal artery. e: place of occlusion; asa: anterior spinal artery. (B) Occluded arterial branch among the pial arteries. Embolic material in the arterial lumen. Magnified 250 times

Corbin² points out that atherosclerotic changes develop slowly and provide a chance for development of collateral circulation. Such a situation is likely to be encountered in the cases described here. Atherosclerosis of spinal arteries occurs with a frequency of 1.2–3.5%, so it is significantly rarer than in the brain and does not reveal any correlation with the age and the occurrence of analogous changes in other arteries.³ Most often atherosclerosis takes the form of fibrosis and elastosis of the intima. Atheromatous plaques in spinal arteries are very rare.³ This is probably related

to the fact that atherosclerosis usually does not affect vessels under 2 mm in luminal diameter.⁷

The artery of Adamkiewicz, the largest artery supplying the spinal cord, has a diameter of 800–1500 μm .¹⁴ Thus, the four anterior radicular arteries of 150–1000 μm in diameter with atherosclerotic changes found in our study are unusual, especially in the light of the fact that the specimens were taken from individuals among whom as many as 78% died after the age of 50. Also 525 posterior radicular arteries of 100–850 μm in diameter were examined in the same specimens and none showed atherosclerosis. This explains the observations of Austin *et al*¹ that occlusion of posterior radicular arteries is much less common than in anterior arteries and ischaemic syndromes in the posterior arterial fields occur only very occasionally. Three obstructed arteries were found in section D10–L3 and this to some extent agrees with observations of some authors^{7,15} that spinal ischaemic lesions resulting from atherosclerosis occur usually in the thoraco-lumbar region.

Occlusion of arteries must be slow for a collateral circulation to develop. We found most evidence for that claim in case 4. The fact that the diameter of the obstructed artery is smaller than diameters of both its ascending and descending branches seems to be of critical importance. As the diameter of the artery decreased, the blood flow was increasing from the adjacent side situated at the level of the artery of Adamkiewicz, ie D10. As a result, the descending branch of the artery of Adamkiewicz of 850 μm in diameter took over the supply of the two-thirds of the anterior part of the whole lumbar enlargement, a phenomenon often encountered in regular conditions.¹⁴ Branches of the obstructed artery—included in the chain of anterior spinal artery—might have dilated, while the artery itself, which in the proximal section was filled with a thrombus, contracted. It cannot be excluded that in this case the additional blood flow to the anterior spinal artery occurred via branches of the terminal conus anastomotic system, which directly connected the anterior spinal artery with posterolateral spinal arteries, and via branches of radicular-pial arteries accompanying all cauda equina radices. These branches undoubtedly made the main circulation pathway of substitute circulation in the consecutive case, where the artery of Adamkiewicz was obstructed. A similar case was previously described by Lazorthes *et al*.^{16,17} Their 65 year old patient also did not have any clinical symptoms of a spinal cord lesion. However at the postmortem examination occlusion of the artery of Adamkiewicz was found. These authors also found that branches of the conus anastomotic system and arteries accompanying cauda equina radices were especially well developed. These anatomical observations were experimentally confirmed on dogs and monkeys.^{7,13,16,18} After ligation of the lumbar enlargement artery (Lazorthes proposed his own name for the artery of Adamkiewicz), it has been

Table 2 The level of the occurrence of obstructed arteries as well as adjacent arteries, diameters of main trunks and final branches and location of site of the occlusion

No., sex, age	Initial cause of death	Level of occurrence of obstructed artery; diameters of trunk and branches [μm]	Artery occurring above the place of occlusion; diameters of trunk and branches [μm]	Artery occurring below the place of occlusion; diameters of trunk and branches [μm]	Artery occlusion site	Number of anterior R - A arteries existing in the specimen
1. F 67	Ischaemic stroke of the right cerebral hemisphere	L ₂ Adamkiewicz artery main trunk - 1000 asc. branch - 250 desc. branch - 900	D ₁₁ main trunk - 850 asc. branch - 180 desc. branch - 850	not occurring	3 mm after penetration to dural sac - fusiform place of occlusion 3.5 mm long	9
2. F 73	Spontaneous intracerebral haematoma with penetration to ventricular system	D ₁₀ main trunk - 400 asc. branch - 400 desc. branch - 200	D ₇ main trunk - 550 asc. branch - 400 desc. branch - 500	L ₁ Adamkiewicz artery main trunk - 1000 asc. branch - 250 desc. branch - 1000	2.5 mm after penetration to dural sac - place of occlusion 3 mm long	7
3. F 60	Endometrial carcinoma	D ₅ main trunk - 150 no division into asc. and desc. branches	D ₃ main trunk - 350 asc. branch - 100 desc. branch - 300	D ₉ main trunk - 450 asc. branch - 350 desc. branch - 400	2.5 mm after penetration to dural sac - place of occlusion 3 mm long	7
4. M 63	Bronchial carcinoma	L ₃ main trunk - 720 asc. branch - 800 desc. branch - 850	D ₁₀ artery of Adamkiewicz main trunk - 850 asc. branch - 220 desc. branch - 850	not occurring	5 mm after penetration to dural sac - place of occlusion 1.85 mm long	6
5. M 58	Myocardial infarction	right initial branch of anterior spinal artery, diameter - 350 μm	left initial branch of anterior spinal artery, diameter - 600 μm	C ₃ main trunk - 550 asc. branch - 280 desc. branch - 400	after 3 mm of convergent course branches join an azygous trunk of anterior spinal artery. Embolism of right branch occurs 0.5 mm before junction and is 0.5 mm long	7

shown that a fluorescent agent administered into the abdominal aorta quickly accumulates within the lumbar enlargement area, thanks to the above-mentioned vessels. These authors consider that the vessels in physiological conditions supply cauda equina nerve roots and in the situation of flow disorders in the main lumbar enlargement area arterial pathways can fully replace them. Having in mind the observations of Lazorthes' and others, we compared diameters and numbers of discussed branches occurring in our examined case with the remaining 49 specimens. Contrary to expectations we have not found any significant differences.

In case 2 the occlusion of anterior radicular artery at level D10 was undoubtedly compensated by the ascending branch of the artery of Adamkiewicz occurring at level L1 and a large 500 μm descending branch of the artery occurring at level D7.

The radicular artery occurring in case 3 at level D5 joined the anterior spinal artery, which was more than double the diameter. In consequence the obstructed

artery could not make its significant inflow and it might be supposed that it was a certain type of reserve vessel of the anterior spinal artery. The descending and ascending branches of adjacent anterior radicular arteries, occurring at levels D3 and D9 and forming the trunk of anterior spinal artery in this section, could easily compensate for the lack of a small radicular artery.

Compensation for one of the initial branches of the anterior spinal artery occlusion is probably easiest to explain. A system of two branches originating from vertebral arteries and joining into an azygous trunk of the anterior spinal artery sometimes called 'a small Willis circle', automatically explains possibilities of compensation. The obstructed smaller branch of 350 μm in diameter was substituted by a branch of 600 μm in diameter and an ascending branch of the anterior radicular artery occurring at level C3.

As the examples show, the formation of a fully efficient collateral circulation, substituting even the main arterial inflows of the spinal cord, is possible.

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