Paraplegia following thoracolumbar sympathectomy

Case report

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A case is presented in which a bilateral thoracolumbar sympathectomy and splanchnicectomy were followed by permanent paraplegia below T-10. The hypothesis is presented that coagulation of a bleeding intercostal vessel during surgery led to a propagating thrombus which involved, successively, the intercostal artery, a segmental medullary vessel, and the anterior spinal artery with resulting spinal cord infarction. Other possible mechanisms are mentioned. Several technical suggestions are offered with regard to prevention of this complication.

Key Words • intractable pain • sympathectomy • splanchnicectomy • paraplegia

ALTHOUGH thoracolumbar sympathectomy has been virtually abandoned as a method of treating hypertension, it remains an accepted procedure for the relief of intractable pain of benign pancreatic disease. It was for the latter purpose that we recently had occasion to perform this operation, and in doing so, encountered an unusual postoperative complication, that of spinal cord infarction. We reviewed the English literature and found 13 previously recorded cases of this occurrence. In all of these cases, however, the operation was performed for the purpose of relieving hypertension. In this paper we present the details of our case followed by a brief review of the pertinent anatomy and a discussion of the possible etiological mechanisms of this complication.

Case Report

This 48-year-old woman was referred to the Neurosurgery Service because of intractable epigastric pain due to chronic pancreatitis. She had suffered from this condition for approximately 17 years during which she had had multiple surgical procedures including cholecystectomy, sphincterotomy, and partial pancreatectomy with little if any benefit. She also had had sympathetic blocks on two occasions, one of which provided pain relief for approximately 6 months. At the time of admission, she required parenteral analgesics every 3 hours.

Examination. The general and neurological examinations were normal except for the presence of the scars of previous surgery. Routine laboratory studies were all normal.

Operation. A bilateral thoracolumbar sympathectomy and splanchnicectomy were performed through Peet's supradiaphragmatic approach. The legs were wrapped in elastic bandages preoperatively. The central
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segment of the eleventh rib was resected bi-
laterally, and the nerve chains were dissected
retropleurally and excised. A minimal
amount of bleeding was encountered when
the periosteum was stripped from the ribs.
This was thought to be of venous origin, pre-
sumably that of the intercostal vessels. The
bleeding was controlled with unipolar elec-
trocoagulation. The total blood loss of the
operation was estimated to be 150 cc. The
pleural cavity was not entered. At no time
during the operation was there a significant
drop in blood pressure.

Postoperative Course. The immediate
postoperative course was uneventful. On the
evening of the first postoperative day, how-
ever, the patient complained of numbness in
the lower part of the legs. Vital signs re-
mained stable. Upon awakening on the
morning of the second postoperative day, the
patient was paraplegic, and all sensation had
been lost below the tenth thoracic derma-
tome. Lumbar puncture showed an opening
CSF pressure of 70 mm H₂O; the fluid was
clear and colorless. Bilateral jugular venous
compression revealed a prompt rise of the
pressure to 140 mm with a rapid drop when
compression was released. Examination of
the CSF revealed 2 red blood cells, no white
blood cells, and 20 mg% protein. A Panto-
paque myelogram revealed a normal-sized
cord shadow with no evidence of obstruc-
tion. The patient's neurological deficit has
remained essentially unchanged over a 6-
month period except for a slight drop in the
sensory level to the twelfth thoracic derma-
tome.

Discussion

Anatomical Considerations

The anterior spinal artery is formed by the
union of a branch from each of the intracra-
nial vertebral arteries. It also receives contrib-
utions from two to 17 anterior segmental
medullary arteries that arise from branches
of the vertebral, subclavian, intercostal, and
lumbar arteries. These medullary arteries are
separate and distinct from the smaller ante-
rior and posterior radicular vessels that ac-
company and supply their respective nerve
roots but do not contribute to the spinal ves-
sels. The anterior spinal artery tapers from
above downward until just above its junction
with the great anterior medullary artery (of
Adamkiewicz) at which point it is difficult to
identify. The great anterior medullary artery,
the largest of the contributing vessels, usu-
ally arises on the left side as a branch of an
intercostal or lumbar artery or from the
aorta and may join at any level from the T-8
to L-4 cord segments.

The posterior spinal arteries, or more cor-
rrectly, plexiform channels, are also branches
of the intracranial vertebral arteries. They
receive contributions from six to eight poste-
rior segmental arteries on each side. Only
rarely do anterior and posterior medullary
vessels contribute to their respective spinal
artery at the same level. Anastomosing
branches from both anterior and posterior
spinal arteries encircle the cord, forming the
pial (vasocoronal) plexus.

The intrinsic blood supply to the ventral
two-thirds of the cross-sectional area of the
cord is from central arterial branches of the
anterior spinal artery. The dorsal third is
supplied by penetrating branches of the pos-
terior plexiform channels. Branches of the
pial plexus supply the outer rim of the cord.
The intrinsic blood supply is furnished by
functionally end arteries; there are no arte-
rial or precapillary anastomoses. There are
anatomical considerations peculiar to the in-
trinsic supply of the thoracic cord which
would make this area more vulnerable to is-
chemic insult: the penetrating vessels in the
thoracic region are fewer and smaller and
branch from the anterior spinal artery at a
more acute angle than in other regions.14,5

Mechanism of Injury

Without pathological material, we can
only hypothesize as to the mechanism of in-
jury in this case. Because the clinical picture
was that of transverse myelitis rather than
the “usual” syndrome of anterior spinal ar-
tery occlusion, the possibility of an inflam-
matory process must be considered. How-
ever, there was no antecedent infectious pro-
cess, or CSF pleocytosis or elevated protein.
Venous thrombosis is another possible
mechanism to be considered. Although the
pattern of venous drainage of the spinal cord
has not been as thoroughly studied as the ar-
terial supply, it is generally assumed that the
veins are more numerous and the potential
for collateral supply is much greater than
with the arteries. In addition, one might ex-
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pect edema and consequent swelling of the cord with significant venous occlusion. This was not demonstrated in our case when myelography was performed.

The most plausible explanation, we feel, is that of arterial occlusion. Bleeding from the intercostal vessels is frequently encountered when rib resection is performed in this operation. In our case, hemostasis was achieved by electrocoagulation, which could have led to a propagating thrombus. If this intercostal vessel gave rise to a segmental medullary vessel, especially the great anterior medullary artery, the thrombus could have extended to the anterior spinal artery. Pertinent to this hypothesis is the recent experimental work of Fried, et al. They showed that ligation of the great anterior medullary artery alone or ligation of the anterior spinal artery above its junction with the former did not result in neurological deficit in the Rhesus monkey, whose spinal circulation is essentially the same as in man. Severe deficit did result, however, when both the great anterior medullary artery and the anterior spinal artery were occluded. In accord with this hypothesis, the 36-hour interval between surgery and the appearance of the neurological deficit in our case probably represents the time necessary for the thrombus to propagate from intercostal to medullary and thence to the anterior spinal artery. In most of the previously recorded cases, the deficit was present when the patient awakened from surgery. Most of these cases, however, suffered operative hypotensive episodes. In addition, all of these patients had been operated on because of hypertension, with its attendant peripheral vascular disease which would predispose them to ischemic insult.

Although spinal cord infarction is a rare complication of thoracolumbar sympathectomy, it is an extremely tragic one. It therefore behooves us to look for methods of prevention. The value of good anesthesia, especially in avoiding hypotension, cannot be overemphasized. Bleeding from intercostal vessels should be controlled with pressure and ligatures rather than coagulation. Perhaps preoperative selective angiography should be performed, as is done with arteriovenous malformations, to identify and possibly avoid that level at which significant arterial contribution exists.

References

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