SHORT REPORT

Haemorrhage associated with silastic dural substitute

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Abstract
Three cases of haemorrhage after the use of a silastic dural substitute are presented. In all cases the implant was removed and further haemorrhage has not occurred. Published work is reviewed and the implications for the continued use of silastic are discussed.

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In an attempt to reduce the incidence of tethering of the intradural contents to the overlying dural scar we have used silastic (Dacron polyester fibre backed silicone sheet; Codman, USA) as a dural substitute. A full report of the indications for its use together with a discussion of its benefits and risks has been prepared. It is with regard to the curious complications of haemorrhage that this report is concerned and we describe three patients in whom this has occurred.

Case reports
Case 1
A 33 year old woman presented with a history of disabling cough headaches and underwent a foramen magnum decompression for herniation of the hindbrain in April 1984. During the operation porcine pericardium (Xenoderm) was used as a dural substitute. Although there was an initial resolution of the presenting symptoms these recurred four years later and an MRI scan revealed posterior tethering of the intradural contents at the previously operated site. A second operation was carried out and on this occasion the brainstem and spinal cord junction was untethered, the original dural substitute was removed, and a silastic sheet inserted in its place. Once again the patient recovered well and became symptom free but six months later she presented again, this time with a subarachnoid haemorrhage confirmed by lumbar puncture. Both CT and MRI scans failed to identify an underlying cause for the haemorrhage. The operation site was re-explored and the silastic patch removed. The patient made a good recovery and was discharged home with full resolution of his symptoms. He has remained free of further haemorrhages during two years of follow up.

Case 2
A three year old Greek boy with symptoms and signs of lower brainstem compression due to herniation of the hindbrain underwent a foramen magnum decompression in September 1990. Silastic sheeting was used to make good the dural defect. Apart from a transient CSF leak controlled by lumbar puncture he made a good recovery with improvement in his presenting symptoms. Two months after discharge he suffered a sudden episode of headache while playing football and was admitted to a hospital in Greece where his symptoms gradually resolved. Two weeks later the same thing happened again and on this occasion he was readmitted to our unit with a subarachnoid haemorrhage confirmed by lumbar puncture. Both CT and MRI scans failed to identify an underlying cause for the haemorrhage. The operation site was re-explored and the silastic patch removed. The patient made a good recovery and was discharged home with full resolution of his symptoms. He has remained free of further haemorrhages during two years of follow up.

Case 3
A 60 year old man with a history of headache and visual deterioration was found to have a right sphenoid wing meningioma. At craniotomy the tumour together with a large area of infiltrated dura was completely excised and a silastic patch inserted to close the dural defect. He presented again 11 months later complaining of mental confusion and ataxia of one month duration. A CT scan revealed a large subdural and intracerebral haematoma adjacent to the original craniotomy (figure). The craniotomy was reopened, the haematoma evacuated, and the silastic removed. The patient made an uneventful recovery with resolution of his symptoms and remains well two years later.
Discussion

The reported vascular complications of silastic dural substitute include extradural, subdural, and subarachnoid haemorrhage. The table summarises the previously reported cases. It is evident that the risk of haemorrhage persists for many years after insertion of the silastic.

The presence of silastic in surgical wounds has been shown to evoke a local tissue reaction leading to the formation of a connective tissue capsule or neomembrane between the prosthesis and the adjacent tissues. The vasculature of these neomembranes comprises a delicate capillary network and it is proposed that rupture of these fragile vessels in response to minor trauma leads to the haemorrhagic event. An alternative explanation is that the bleeding is the result of direct trauma of the silastic sheet on local vessels. Of course, the possibility exists that the haemorrhage was not associated with the silastic at all; however we consider that this possibility was excluded by means of angiography in case 1, and in case 2 where two episodes of haemorrhage had occurred it was "proved" by the lack of further haemorrhages after removal of the silastic. Whatever the correct mechanism is, it might be anticipated that haemorrhage is more likely to occur when the silastic has been used in an area of particular mobility such as the craniovertebral junction. Boop and Chaddock reported no haemorrhagic complications in a series of 33 patients. They attributed this to the use of non-reinforced silastic, which is a thinner and more pliable material than the Dacron backed alternative used here, and one which they claim is associated with minimal local tissue reaction and neomembrane formation.

Cases 1 and 2 represent an incidence of 5% of haemorrhagic complications associated with the use of silastic dural substitute seen over a five year period. The largest group in whom we have used the silastic is that with spinal dysraphism and it is interesting that the complication has not been encountered in this group.

Two of the three cases presented haemorrhaged at the foramen magnum and in each there were two episodes of haemorrhage. The tendency for repeated bleeds in the presence of silastic has been documented by others.

Haemorrhage after the use of silastic in the posterior fossa was also reported by Simpson and Robson. Although it might be argued that the predilection for this site is associated with the degree of mobility of the craniovertebral junction it does not explain the intracranial haemorrhages.

Silastic, however, seems to prevent tethering or retethering after intradural procedures. In our experience and that of others, autologous tissues (pericranium, fascia lata, and fat), although readily available, are not generally as effective. Cadaveric and animal dural substitutes have the same disadvantage and there is the added concern of the inoculation of transmissible (particularly PRION associated) diseases. We consider that there is still a place for silastic as a dural substitute, perhaps in its non-reinforced form in cases of intramedullary spinal lesions and in the dysraphic population as well as in those procedures designed specifically to treat tethering from previous surgery.

In summary we wish to highlight the haemorrhagic complications associated with the use of silastic dural substitute. We propose that (1) this phenomenon is due in part at least, to a mechanically induced disruption of vascular neomembranes and as such the risk may vary with the anatomical site; (2) the risk of haemorrhage seems to continue long after the time of implantation; and (3) there is a tendency for this complication to recur. We therefore advocate removal of the prosthesis.

Table Previously reported haemorrhagic complications of silastic dural substitute

<table>
<thead>
<tr>
<th>Source</th>
<th>Site of haemorrhage</th>
<th>Extradural</th>
<th>Subdural</th>
<th>Subarachnoid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Banergee et al. (2 cases)</td>
<td>6 years</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
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<tr>
<td>Adehite et al. (1 case)</td>
<td>7 years</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ongkiko et al. (1 case)</td>
<td>9 weeks</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Simpson and Robson (3 cases)</td>
<td>19 days</td>
<td>Cerebellar astrocytoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Misra and Shaw (1 case)</td>
<td>4 months</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aowad et al. (2 cases)</td>
<td>6 years</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fontana et al. (2 cases)</td>
<td>12 years</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thompson et al. (3 cases)</td>
<td>6 months</td>
<td>Hindbrain herniation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 months</td>
<td>Hindbrain herniation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>11 months</td>
<td>Cranial meningioma</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

For each case the original diagnosis is given with the time between insertion of the silastic and the presentation of the haemorrhagic event.
once the diagnosis of haemorrhage has been made.


John and James Parkinson on appendicitis

Though straying from the strict confines of neurology, one of the classic, early accounts of appendicitis may be of interest. The extraordinarily versatile James Parkinson 1 read this paper on 21 January 1812. A boy about 5 years of age, who died under the following circumstances:

He had been observed for some time to decline in health, but made no particular complaint until two days before his death when he was suddenly seized with vomiting and great prostration of strength. The abdomen became very tumid and painful upon being pressed; his countenance pale and sunken, his pulse hardly perceptible. Death preceded by extreme restlessness and delirium, took place with 24 hours.

Upon examination the whole surface of the peritoneum was found inflamed with a thin coat of coagulable lymph; ... The viscera independent of the inflammation of the peritoneal covering, appeared in a perfectly healthy state, excepting the appendix vermiformis of the coecum. No dissected appearance was seen in this part near to the coecum; but about an inch of its extremity was considerably enlarged and thickened, its internal surface ulcerated, and an opening from ulceration which would have admitted a crow quill, was found at the commencement of the diseased part, about the middle of the appendix, through which it appeared, that a thin dark-coloured and highly fetid fluid, had escaped into the cavity of the abdomen. Upon opening the appendix, a piece of hardened feces was found impacted ...

... It is often overlooked that the title of this paper is subheaded: “By John Parkinson, Esq., Surgeon. Communicated by James Parkinson, Esq.”

James had married Mary Dale in May 1781 and they had six children: two died in infancy, one son John qualified in Medicine and shared his practice. I remain unsure as to who made this major contribution.

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