The Journal of Laryngology and Otology
January 1998, Vol. **112,** pp. 88–91

Transverse sinus thrombosis and venous infarction of the brain following unilateral radical neck dissection

Z. Z. Mahasin, F.R.C.S.(C), M. Saleem, F.R.C.S., K. Gangopadhyay, M.S., D.N.B., F.R.C.S.

Abstract
Radical neck dissection is one of the commonest procedures performed in any unit dealing with head and neck surgery. Intracranial complications following this procedure are uncommon. Transverse sinus thrombosis and venous infarction of the brain following unilateral radical neck dissection have not been reported in the literature. We present a case in which this complication occurred following an uneventful radical neck dissection.

Key words: Cranial sinuses, Sinus thrombosis; Radical neck dissection

Case report

A 71-year-old female presented to King Faisal Specialist Hospital and Research Centre with a three-month history of a swelling in the left sub-mandibular area. She had undergone a resection of squamous cell carcinoma of the left half of the lower lip, with primary closure, nine months earlier in another hospital. She had no other complaints. On clinical examination she had a 2 × 2 cm lymph node in the left submandibular area. There was no other palpable cervical lymph node. The area of the lower lip where a previous excision had been performed did not reveal any abnormality or a recurrent lesion. Fine needle aspiration cytology of the sub-mandibular node showed a metastatic squamous cell carcinoma. Review of the slides and the paraffin blocks of the primary lesion obtained from the referring hospital confirmed a completely excised squamous cell carcinoma. Computed tomography (CT) scan of the neck showed a 2 cm node in the left sub-mandibular area with a necrotic centre and peripheral enhancement. Chest X-ray was clear.

A left radical neck dissection was undertaken. The procedure was uneventful as was the post-operative recovery. The drains were removed on the third post-operative day and the patient was discharged one week after surgery. Twenty-one days following the radical neck dissection the patient presented to Accident and Emergency with a three-day history of headache and a temperature of 38°C. No other abnormalities were present on clinical examination. The neck wound was not infected nor inflamed. The haematological profile was within normal limits. Urinalysis and chest X-ray did not reveal any abnormality. A blood culture specimen was taken. The patient was then discharged from Accident and Emergency with the advice to report back if her symptoms got worse. Three days later the patient came to Accident and Emergency again. She complained of headache and vomiting. On examination she was apyrexial, drowsy, disorientated and had slurred speech. Examination of the central nervous system showed brisk reflexes and upward plantars on the right side. The patient also had receptive dysphasia. The left pupil was fixed at 3 mm and the right showed a sluggish reaction. There was no papilloedema or signs of meningeal irritation. The rest of the central nervous system examination was normal. There were no signs nor symptoms of deep vein thrombosis. Haematological and coagulation profiles were again within normal limits. An urgent magnetic resonance imaging (MRI) scan of the brain showed venous infarction and haemorrhage in the left temporal lobe with mild surrounding oedema (Figure 1). There was a very high intensity signal from the left transverse sinus indicative of either thrombus formation or slow flow of blood (Figure 2). No mass effect was present. In the area of the left transverse sinus, sigmoid sinus and jugular bulb there was an area of increased signal intensity. After injection of gadolinium there was no significant enhancement in these areas. These findings are compatible with thrombus formation. The rest of the intracranial dural sinuses were patent. The patient was admitted. The blood culture results were available from the visit to Accident and Emergency three days earlier and showed growth of *Streptococcus viridans* sensitive to vancomycin which was commenced after consultation with Infectious Disease colleagues and continued for 10 days. To rule out an embolic phenomenon doppler examination of the carotids and echocardiogram was carried out. Doppler of the carotids showed good blood flow bilaterally with normal spectral analysis. Infective endocarditis was ruled out as both the precordial and trans-oesophageal echocardiogram were within normal limits.

On the night of admission she had a grand mal convulsion. The patient was given intravenous diazepam and phenytoin was commenced. Over the next 10 days the patient's condition stabilized. She remained apyrexial and did not have any more convulsions. The reflexes however remained brisk but pupillary reaction was still sluggish. A repeat CT scan of the brain before discharge did not show

From the Section of Otolaryngology – Head and Neck Surgery, Department of Surgery, King Faisal Specialist Hospital and Research Centre, Riyadh, Saudi Arabia.
Accepted for publication: 30 October 1997.

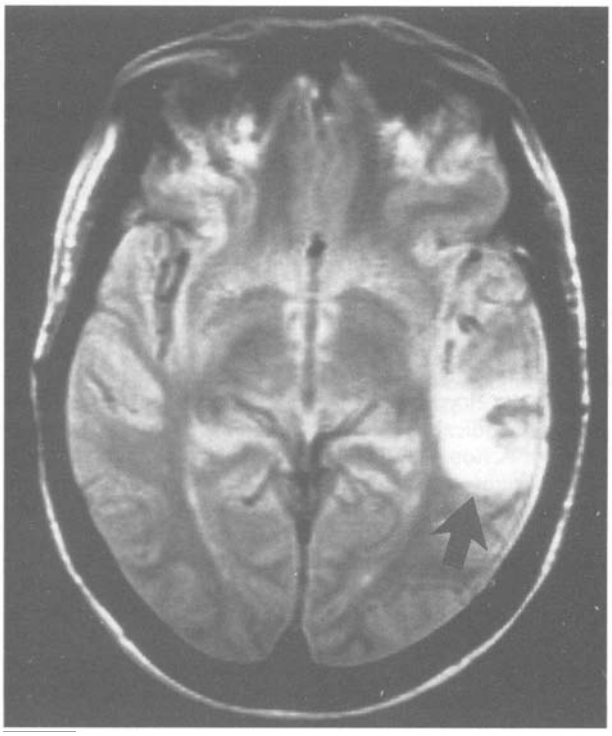

FIG. 1
MRI with gadolinium (axial cut) showing left temporal lobe haematoma (arrow).

any change from the earlier CT. The patient was discharged 10 days later on phenytoin. At the time of discharge apart from bilateral sluggish pupillary reaction, there were no other neurological abnormalities. The receptive dysphasia had improved. The patient was reviewed in Head and Neck follow-up clinic as well as in the Neurology clinic three months after discharge. She had recovered completely and did not show signs of any neurological deficit. A CT scan with contrast carried out on

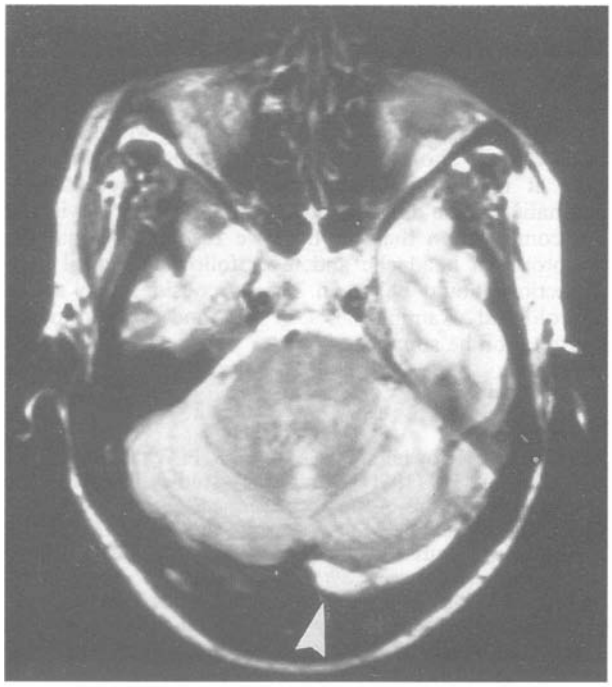

FIG. 2
MRI with gadolinium (axial cut) showing left transverse sinus thrombosis (arrow).

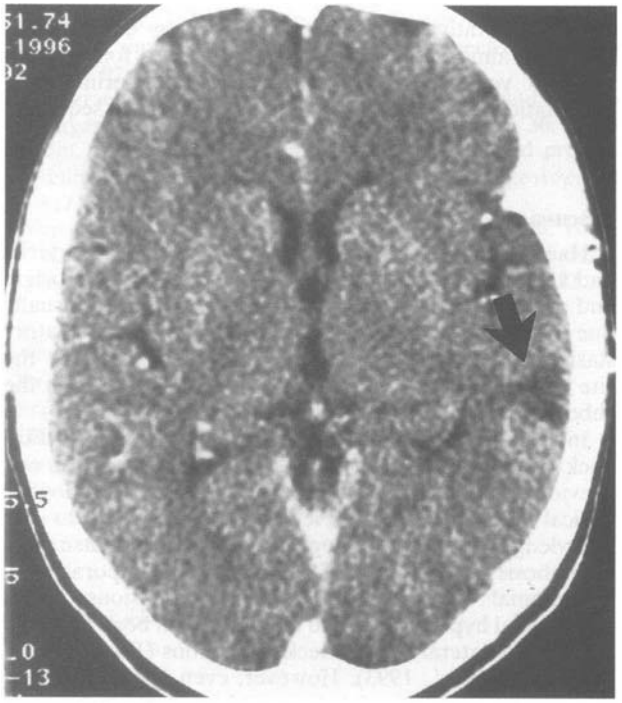

FIG. 3
CT scan (axial cut) one year later showing complete resolution of the haematoma and the area of infarction which has become smaller (arrow).

this visit showed complete resolution of the haematoma. The area of infarction had become smaller (Figure 3). The whole of the left transverse sinus up to the confluence of the sinuses did not fill with contrast indicating a thrombus (Figure 4). There was also a rich venous network adjacent to the tentorium on the left side, which was not present on

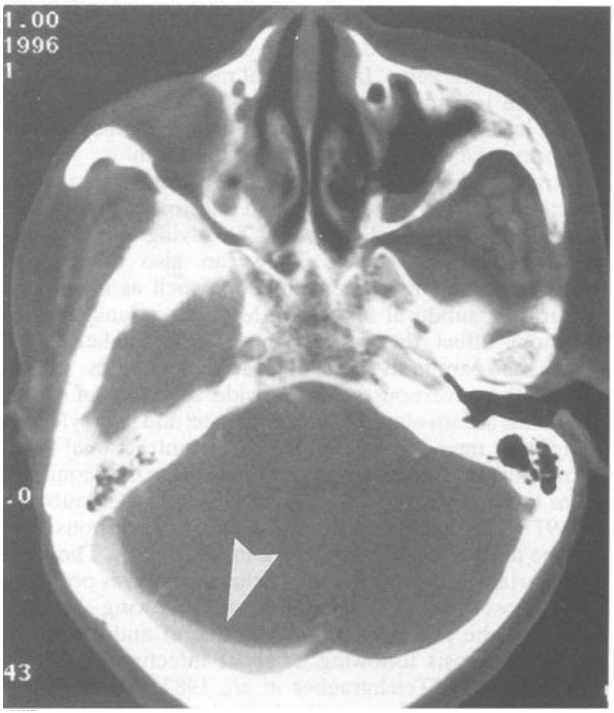

FIG. 4
CT scan with contrast (axial cut) one year later showing the whole of the left transverse sinus up to the confluence of the sinuses not filling with contrast indicating a thrombus. The patent right side can be visualised (arrow).

https://doi.org/10.1017/S0022215100139994 Published online by Cambridge University Press

the pre-operative CT, indicating opening of collateral venous drainage. The patient has now been followed up for two years. There is no evidence of primary or metastatic disease. She also has no neurological sequelae.

Discussion

Haemorrhagic infarcts are usually classified as arterial and venous. Arterial infarcts are usually embolic in origin and are located in the cortex. Venous infarcts are usually due to cerebral venous occlusion which causes circulatory stasis, extensive diapedesis and haemorrhage above the site of occlusion. These infarcts are usually located in the subcortical white matter (Moulin *et al.*, 1993).

Intracranial complications following unilateral radical neck dissection are very rare. Although there has been one previous case report of lateral sinus thrombosis following radical neck dissection (Fielding *et al.*, 1973), to our knowledge this is the first reported case of transverse sinus thrombosis and venous infarction in the temporal lobe. Intracranial complications such as convulsions, coma, intracranial hypertension and death have all been reported following bilateral radical neck dissections (Razack *et al.*, 1980; Weiss *et al.*, 1993). However, even in these reports there is no mention of intracranial sinus thrombosis or venous infarction. Similarly, although there are numerous reports in the literature of increased intracranial pressure and disturbances in visual acuity (Morfit and Cleveland Jr., 1958; Fitz-Hugh *et al.*, 1966; Tobin, 1971) following unilateral radical neck dissections, there are no reports of venous infarction of the brain or thrombosis of the transverse sinus.

Venous drainage of the head is a complex network which is very variable. The blood leaves the brain via internal jugular veins, the occipital plexus, orbital plexus, pterygoid plexus, emissary veins and pharyngeal plexus. There is a communication between the intracranial and extracranial venous systems through the emissary and diploic veins. The predominant venous outflow is via the internal jugular veins. In unilateral radical neck dissection where the ipsilateral jugular vein is removed the contralateral internal jugular vein along with the other venous networks take over the drainage of the brain.

Intracranial dural sinus thrombosis is known to occur in infections of the middle ear and mastoid air cells, the paranasal sinuses and infections involving the mid-face area (Schlossberg, 1987). They can also occur as a complication of an adjacent infection such as meningitis, epidural or subdural abscesses. Middle ear and mastoid infections affect the lateral sinus (Teichgraeber, 1982) while the paranasal and mid-facial infections usually involve the cavernous sinus. A wide spectrum of micro-organisms are involved with streptococci and staphylococci being the most common. However, intracranial sinus thrombosis as a post-operative complication is uncommon. It has been reported following nasal surgery (Casaubon *et al.*, 1977; Teichgraeber and Russo, 1993). Cavernous sinus was the main sinus to be involved in these cases. There are no reported cases of transverse sinus thrombosis occurring as a post-operative complication following surgery. Although the pathogenesis of the lateral and cavernous sinus thrombosis following adjacent infections has been widely studied (Teichgraeber *et al.*, 1982), there are no reports or studies to show the behaviour of blood flow in the ipsilateral dural sinuses following ligation of the internal jugular vein following radical neck dissection. It is possible that following the ligation of internal jugular vein there is obstruction to the blood flow in the sigmoid and lateral sinus which may result in the opening of other venous channels to direct the venous drainage of the ipsilateral brain to the contralateral venous sinuses.

Variations in the anatomy of intracranial dural sinuses have been widely studied. Durgun *et al.* (1993) have evaluated the lateral dominance of the dural venous sinuses by angiography in 206 cases. They found that in 2.1 per cent of cases the drainage is limited to the right side only and in 0.5 per cent it is to the left side only. Modic *et al.* (1983) found that portions of dural sinuses may be absent or variable in size. Kaplan *et al.* (1972) report that in 58 per cent of cadavers, there was a 1–3 cm atresia of the rostral end of the anterior superior sagittal sinus and this resulted in variations of cortical veins draining the cerebrum. Anlyan *et al.* (1951) describe a case where a patient developed a right hemiplegia immediately following a left radical neck dissection and died two weeks after surgery. Autopsy of this patient revealed a pinpoint lumen of the right transverse sinus which was obviously not sufficient to cope with the venous drainage of the brain.

In our patient it is difficult to determine the exact cause of the dural sinus thrombosis and venous infarction of the brain. There are two possible reasons how this may have occurred. One possibility is infection in the blood clot in the proximal portion of the internal jugular vein as the blood culture showed growth of *Streptococcus viridans*. This infection may have resulted in ultimate thrombus formation in the intracranial sinuses. However, it is difficult to pinpoint the exact source of *Streptococcus viridans*. There was no wound infection in the post-operative stage. This organism is part of the normal microflora of the oral cavity, upper respiratory tract, female genital tract and all regions of the gastrointestinal tract (Johnson and Tunkel, 1995). Any of them could have been the cause of bacteraemia although the patient did not have any specific complaints related to this area. Another possibility is that following the ligation of the internal jugular vein, alternate venous channels were not available to provide adequate venous drainage for the temporal lobe which not only resulted in venous congestion and eventual development of temporal lobe haematoma but also caused transverse sinus thrombosis due to venous statis.

Conclusion

Radical neck dissection is one of the commonest procedures performed on any unit dealing with head and neck cancer. Short of performing angiography in every case, it is not possible to be aware of the different anomalies of the dural sinuses pre-operatively. In view of the complication that we had, we recommend that the symptoms of headache and fever following radical neck dissection should be given serious consideration and a neurological examination, CT scan and a neurological consultation be sought.

References

Anlyan, A. J., Browning, H. C., Black, S. P. W., Clifton, E. E., Janzen, A. H. (1951) Dural sinus studies in relation to radical operations of the neck. *Surgical Forum* 300.

Casaubon, J., Dion, M. A., Larbisseau, A. (1977) Septic cavernous sinus thrombosis after rhinoplasty. *Plastic Reconstructive Surgery* **59:** 119–123.

Durgun, B., Ilglt, E. T., Cizmeli, M. O., Atasever, A. (1993) Evaluation by angiography of the lateral dominance of the drainage of the dural venous sinuses. *Surgical Radiologic Anatomy* **15:** 125–130.

Fielding, I. R., Grant, J. M. F., Selby, G. (1973) Lateral sinus thrombosis following radical neck dissection for malignant melanoma. *Australian and New Zealand Journal of Surgery* **43:** 228–231.

Fitz-Hugh, G. S., Robins, R. B., Craddock, W. D. (1966) Increased intracranial pressure complicating unilateral neck dissection. *Laryngoscope* **76:** 893–906.
Kaplan, H. A., Browder, A. A., Browderm, J. (1972) Atresia of the rostral superior sagittal sinus: Associated cerebral venous patterns. *Neuroradiology* **4:** 208–211.
Johnson, C. C., Tunkel A. R. (1995) Viridans streptococci and Group C and G streptococci. In *Principles and Practice of Infectious Diseases.* (Mandel, G. L., Bennett, J. E., Dolin, R., eds.) Churchill Livingstone, New York, p 1846.
Modic, M. T., Weinstein, M. A., Starnes, D. L., Kinney, S. E., Duchesneau, P. M. (1983) Intravenous digital subtraction angiography of the intracranial veins and dural sinuses. *Radiology* **146:** 383–389.
Morfit, H. M., Cleveland, Jr., H. (1958) Permanent increased intracranial pressure following unilateral radical neck dissection. *Archives of Surgery* **76:** 713–723.
Moulin, T., Crepin-Leblond, T., Chopard, J., Bogousslavsky, J. (1993) Haemorrhagic infarcts. *European Neurology* **34:** 64–67.
Razack, M. S., Sako, K., Baffi, R., Patel, J. (1980) Simultaneous bilateral neck dissection. *Journal of Surgical Oncology* **15:** 387–392.
Schlossberg, D. (1987) Infections of the eye and orbit. In *Infections of the Head and Neck.* (Ostler, H. B., Okumoto, M., eds.) Springer-Verlag, New York, pp 39–40.
Teichgraeber, J. F., Per-Lee, J. H., Turner Jr., J. S. (1982) Lateral sinus thrombosis: A modern perspective. *Laryngoscope* **92:** 744–751.
Teichgraeber, J. F., Rosso, R. C. (1993) Treatment of nasal surgery complications. *Annals of Plastic Surgery* **30:** 80–88.
Tobin, H. A. (1971) Increased cerebrospinal fluid pressure following unilateral radical dissection. *Laryngoscope* **81:** 817–820.
Weiss, K. L., Wax, M. K., Haydon, R. C., Kaufman, H. H., Hurst, M. K. (1993) Intracranial pressure changes during bilateral neck dissections. *Head and Neck* **15:** 546–552.

Address for correspondence:
Dr M. Saleem, M.B.B.S., F.R.C.S.,
Section of Otolaryngology – Head and Neck Surgery,
Department of Surgery, M.B.C. #40,
King Faisal Specialist Hospital and Research Centre,
P.O. Box 3354,
Riyadh 11211,
Saudi Arabia.

Fax: 966 1 442 7772