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PERMANENT DDDR CARDIAC PACING FOR HYPERSENSITIVE CAROTIC SYNDROME AFTER SURGERY

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Abstract: *We report a case of a 53 year old patient with recurrent syncope episodes following neck surgery intervention for a tong spinocelular epidermoid carcinoma. During the syncope, he experienced severe bradycardia (15 beats/min) and hypotension with systolic BP 50 mm Hg (mixed VASA 3 like response). A DDDR pacemaker was then implanted with no further syncopal episodes and significant improvement in quality of life.*

Key words: *sinus carotis hypersensitivity, vagal-mediated syncope, permanent cardiostimulation, spinocellular carcinoma somatic.*

Background

The usefulness of cardiac pacing for the prevention of vagal syncope is still controversial, especially when a vaso-depressor component is associated.

We report a case of a 53 year old patient, admitted in hospital in December 2008 for recurrent syncopal episodes associated with falls and physical injury.

Two month ago the patient has underwent surgery for a spinocelular epidermoid carcinoma of the tongue (through neck incision and tissue dissection). During the surgical intervention a left side neck ade-nectomy was also performed. Anatomopatho-logical examination of the removed tumour tissue revealed squamous cell carcinoma. After the intervention he received chemo-therapy. He was discharged after surgery and has remained free of any symptoms until he starts exhibiting syncopal episodes.

The patient was a heavy smoker (30-40 cigarettes/day), quit smoking 5 years ago, was alcohol abstinent with no other medical or surgical history of note.

The physical examination at admission was unremarkable except a post-operative scar on the left side of the neck. We found normal BP values (120/70 mmHg), normal regular heart rhythm (80 beats/min), no additional cardiac and/or vascular murmurs. Carotid sinus massage on each part was negative. Blood samples were in normal range.

A cardiac echocardiography examination (Sonos Agilent 4500, Philips Healthcare, Best, The Netherlands) showed a normal heart and normal aspect of the duplex ultrasound scanning of the carotid arteries.

Neurological examination was negative.

Due his oncological history, a complete CT scan (head, neck, thorax), native and with contrast substance was performed, in order to rule out any other possible aetiology of the syncope (eg. brain metastasis). The examination was negative.

During hospitalization, the patient presented few syncopal episodes which always appeared in orthostatic position. Sometimes, prodromes like sharp neck aches radiating to the mandible were preceding the episode. A sudden drop of a

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heart rate until 15 beats /minute (fig.1) which lasted more than 30 seconds was then documented. This severe bradycardia was associated with a systolic BP decrease until 50 mm Hg mimicking a mixed VASA 3 like response. [9]

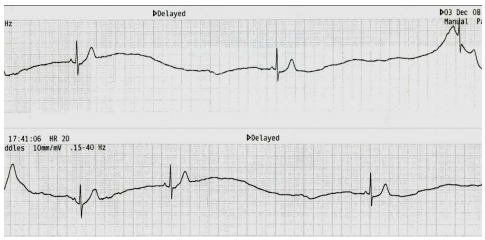

Fig. 1. *ECG during the syncope episode*

The clinical presentation, together with the laboratory investigations suggested recurrent vagal syncope possibly triggered by the stimulation carotid sinus due to the left neck scar.

As it is already known, drugs have a limited role in the management of vagal syncope. [4] In highly symptomatic vagal fainters with relative bradycardia, dual-chamber cardiac pacing was proven to be more effective than drugs in reducing the likelihood of syncope recurrences. [1]

The patient was discharged and a pacemaker implantation was proposed after one month follow-up. He was advised to avoid sudden head movements and sudden changes from horizontal to erect position and to increase the fluids and salt intake. He was also advised to lay down immediately in case of the prodroms occur.

After one month follow-up he continued to present syncope almost daily, with a subsequent significant impact on quality of life.Although an automatic rate-drop sensing dual chamber pacemaker would be the best choice in this case [8], this device was unavailable. A BIOTRONIK PHILOS DR device was then implanted with bipolar BIOTRONIK SELOX ST 53 and SELOX 60 passive fixation leads positioned in right auriculum and in the right ventricle apex. (fig 1). The device was programmed in DDD mode at 90 beats/min with 60 beats/min hysteresis and long AV delay of 300 msec. Programmed output were 2.5 V with 0.4msec for both atrial and ventricular leads (both unipolar mode) and the programmed sensing was 0.5mV and 2mV for atrial and ventricular lead respectively, both in bipolar mode.

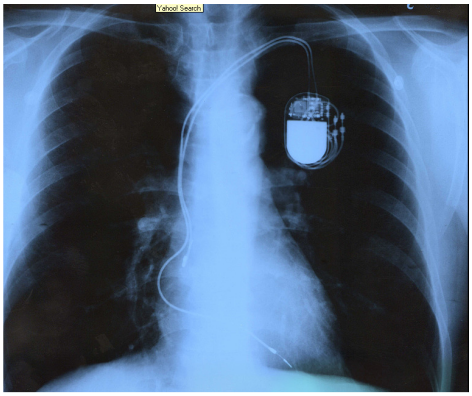

Fig. 2 *Biotronic Philos DR implantation*

After the implant the patient became asymptomatic. A treadmill test (Bruce protocol) was also performed, the patient attained step 3 on the protocol and no symptoms was revealed. The patient underwent monthly controls. He remained free of symptoms for the next two months. After this interval, he had few lightness which become more serious and then syncope-like episodes reappeared.

He was readmitted into the hospital and syncopal episodes were recorded. We obviated the fact that, despite an appropriate pacemaker function (see fig 2.) during the syncopal episodes, the patient still presented an important decrease of SBP until 70 mmHg.

Although it is known that patient with heart rate of 15-20 b/m will experience low BP, as was the case before the pacemaker implantation, we found that a less severe hypotension still exist after the implanttantion, thus favour the hypothesis that a vaso-depressor component may also be involved.

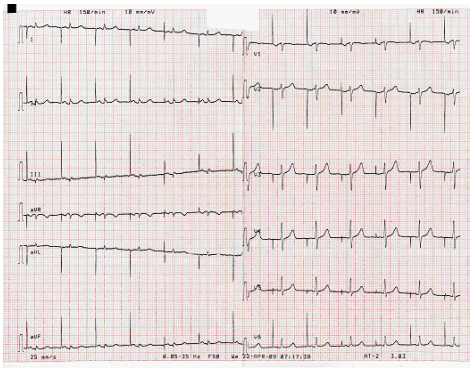

Fig. 3. *ECG tracing during a syncope-like episode.*

Clinical examination this time showed irregular mass at the left side of the neck, with hard consistency and apparently fixed to the skin and underlying tissue. The entire area was painful. Swalowness was not affected. Fever, weight loss, malaise or other accompanying symptoms were absent. Chest radiography showed no leads dislodgment.

Repeated CT exam (head and neck native and with contrast substance) showed tumour mass at the left neck level, infiltrating the local tissues and surrounding the neck vessels and carotid sinus (see fig 4).

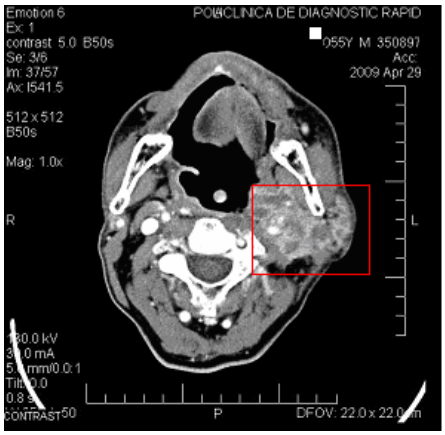

Fig. 4. *CT scan showing left sided tumour mass*

The patient was referred to an Oncology Department for further treatment. He was re-operated. The analysis of the removed tissue reconfirmed the diagnosis of spinocelular epidermoid carcinoma. The patient eventually died in September 2009. Few episodes of lightness and no significant syncope were recorded during this time. The presence of pacemaker was also allowed a much safer surgery around sinus carotis region.

Discussions

About 60% of supraclavicular triangle masses are metastases from distant primary sites, elsewhere in the neck, 80% of cancerous cervical adenopathy originates in the upper respiratory or alimentary tract. [5] Likely sites of origin are the posterior-lateral border of the tongue and the floor of the mouth followed by the nasopharynx, palatine tonsil, laryngeal surface of the epiglottis, and hypo pharynx, including the pyriform sinuses.

In this particular case, the metastasis appeared as clinically significant 6 months after surgery.

Regarding the mechanism of syncope, we have initially supposed a dysfunction of carotid sinus receptor after surgery and/or a mechanical factor involving the neck scar which was able to trigger vaso-vagal reflexes. In evolution, the direct impact of the left neck tumour mass (metastasis) added its contribution to the worsening of symptoms. fig 3.

The relative efficacy of a pacemaker implantation is mostly described for cardio-inhibitory induced vaso-vagal syncope [3, 7] but this therapy seems to be efficient in hypersensitive carotic syndrome which responses to DR pacemaker implantation [3]. However, the usefulness of cardiac pacing in patients with combined response (vasodepressor and cardio-inhibitor) remains controversial

because of the dominant role of the vasodepressor component during the episode. An upright tilt test (UTT) would be beneficial for further clarification of the syncope mechanism, but it was not available.

Although this patient was known as having a poor prognosis due to his oncological illness, we decided pacemaker implantation that provided him a significant improvement in quality of life, even if it was not a drop-response rate device.

Repeated episodes of hypotension in this patient were in fact impossible to be avoided, but maintaining an appropriate heart rate by implanting a pacemaker we have actually converted the syncope episode in syncope–like episodes or just lightness which was much easier to support and manage. This is an important aspect while those patients with recurrent syncope have a high level of quality of life impairment. [2].

Conclusion

Pacemaker DDDR therapy in patients with vagal syncope triggered by post-surgical acquired carotis sinus hypersensitivity and manifest with both cardio-inhibitor and vasodepressor mechanism may be useful in regard to significant improvement in quality of life by decreasing the severity of syncope episodes.

References

1. Ammirati F., Colivicchi F., Santini M., Vasovagal Syncope: A Multicenter, Randomized, Controlled Trial. *Circulation* 2001; 104; 52-57.
2. Calkin's H., Zipes D. in Braunwald's Heart Disease: *A Textbook of Cardiovascular Medicine*, 8th ed. Chapter 37 *Hypotension and Syncope*, p. 977-978.
3. Connoly S.J., Sheldon R., Roberts R.S., et al. The North American Vasovagal Pacemaker Study (VPS) A Randomized Trial of Permanent Cardiac Pacing for the Prevention of Vasovagal Syncope JACC 1999. 33, 1:203.
4. Fenton A. M., Hamill S.C, Rea R.F. et al. Vasovagal syncope. *Ann Intern Med*. 2000; 133:714-725. 2.
5. Gleeson M., Herbert A., Richards A. Management of lateral neck masses in adults. *BMJ.* 2000 June 3; 320(7248): 1521–1524
6. Linzer M., Pontinen M., Gold D.T., Divine G.W., Felder A., Brooks W.B. Impairment of physical and psychosocial function in recurrent syncope. *J Clin Epidemiol* 1991; 44: 1037–43.
7. Petersen M.E.V., Chamberlain – Webber R., Fitzpatrick A.P., et al. Permanent pacing for cardioinhibitory malignant vasovagal syndrome. *Br Heart J*. 1994;71:274-281
8. Sheldon R.S., Koshman M.L., Wilson W., Kieser T., Rose S. Effect of dual-chamber pacing with automatic rate-drop sensing on recurrent neurally mediated syncope. *Am J Cardiol* 1998; 81:158–162.
9. Sutton R., Petersen M., Brignole M., et al. Proposed classification for tilt induced vasovagal syncope. *Eur J Cardiac Pacing Electrophysiol.* 1992; 2: 180 –183.