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- Dizziness in the Maltese Community Setting: The Implications of Good History-Taking
- A Case Report and Overview of Carbamate Insecticide (Baygon®) Poisoning
- Meeting Prof. Lino Cutajar

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EDITORIAL

BLOCKCHAIN BRAVE NEW WORLD

Further to the previous editorial, let me attempt to highlight the main advantages of blockchain in relation to the Maltese healthcare industry…

INTEROPERABILITY - CLINICAL HEALTH DATA EXCHANGE

Health data interoperability, integrity and security, portable user-owned data … these are only a few of the buzz words surrounding blockchain-enabled health IT systems. Blockchain can enable data exchange that is cryptographically secured, allowing seamless access to historic and real-time patient data, thus eliminating the burden and cost of data reconciliation. This is exemplified by the recent collaboration between Guardtime, a security software firm, and the Estonian eHealth Foundation to secure the health records of one million Estonian citizens.

BILLING MANAGEMENT

In the US, it has been estimated that 5-10% of annual healthcare costs are fraudulent, primarily resulting from excessive billing and billing for non-performed services. In 2016 Medicare fraud was, in fact, estimated to amount to $30 million. By automating claim adjudication and payment processing activities through blockchain-based systems, one can forgo intermediaries and thus reduce administrative costs, time and obviously, the risk of fraud.

DRUG SUPPLY CHAIN INTEGRITY

Counterfeit drugs incur an estimated global annual loss of $200 billion. Indeed, approximately 30% of drugs found in developing countries is considered to be counterfeit. The pharmaceutical industry is already using technology to detect counterfeits such as scratch-off labels and radio frequency identification tags, although with some limitations. Blockchain seems to be the next evolution of these efforts, in that a blockchain-based system could ensure a chain-of-custody tracking log. This would enable manufacturers to track and trace every ingredient throughout its life-cycle as well as track and verify the authenticity of the finished product. Add-ons such as private keys and smart contracts could also help offset this plague. One such example is the Advanced Digital Ledger Technology [blockchain], being developed by iSolve LCC, a US company, aiming to ensure drug supply chain integrity.

CLINICAL TRIALS

It has been estimated that as much as 50% of clinical trials conducted worldwide go unreported, often because the results are negative. This creates knowledge gaps for healthcare stakeholders. Blockchain-enabled, time-stamped records of clinical trials, protocols and results could address important challenges such as selective reporting.

CYBER SECURITY

According to Protenus, a cloud-based analytics platform, there were a total of 450 health data breaches in the US in 2016, affecting over 27 million patients. The majority [43%] related to insider-caused breaches, followed by hacking and ransomware [27%]. One main challenge is the rise in connected health devices; an estimated 25 billion healthcare Internet of Things [IoT] connected devices will be used globally by 2020, posing challenges in the evolution of IoMT [Internet of Medical Things] ecosystems. Blockchain-enabled solutions will be the cornerstone of such ecosystems, bridging gaps of device data interoperability and at the same time, ensuring security, privacy and reliability.

Our future is exciting.

In order to conclude this short analysis, I quote Miranda's words from *The Tempest*…

'O Brave New World,
That has such [technology] in 't!'

With apologies to William Shakespeare...

Ian Ellul

Cover: Sir Paul Boffa Hospital, former King George V (KGV) Hospital, is not only a military building but it was initially opened in 1922 as a memorial to the men of the Merchant Navy who died in World War I. The building was severely damaged in 1942 during World War II, and although there had been a temporary halt due to air raid bombardment, the hospital services were never interrupted. The memorial was then rebuilt in 1948 under the premiership of Paul Boffa and renamed Sir Paul Boffa Hospital in 1976. This building has been in existence for over 50 years, and out of all the naval hospitals in Malta to date it is only Zammit Clapp Hospital and Boffa Hospital that still maintain a medical function.

Editor-in-Chief: Dr Wilfred Galea
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Sales & circulation Director: Carmen Cachia

Email: mpl@thesynapse.net
Telephone: +356 21453973/4

Publisher:
Medical Portals Ltd
The Professional Services Centre
Guzi Cutajar Street, Dingli
Malta, Europe

Production: Outlook Coop

Printing: Europrint Ltd

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Dr Theresa Agius is a Foundation Year 1 doctor currently finishing her renal medicine rotation. At the time of writing this paper she was a final year medical student. Dr Agius is at present studying cutaneous malignancy. The co-author of the article is Mr Adrian Agius.

ISSUE GUIDE

SEROXAT

The Anxiolytic Antidepressant:[1,2]

Major Depressive Disorder (MDD)[3]

Generalised Anxiety Disorder (GAD)[3]

Social Anxiety Disorder (SAD)[3]

Post -Traumatic stress Disorder (PTSD)[3]

Obsessive Compulsive Disorder (OCD)[3]

Panic Disorder[3]

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DIZZINESS IN THE MALTESE COMMUNITY SETTING: THE IMPLICATIONS OF GOOD HISTORY-TAKING

THERESE M AGIUS & ADRIAN M AGIUS

ABSTRACT

This paper presents a retrospective analysis of 100 consecutive patients presenting with dizziness to a community-based otolaryngologist. The aim of this study was to analyze the main causes of dizziness in the community, and to correlate the symptoms with the final diagnosis.

The commonest causes of dizziness in this group were benign paroxysmal positional vertigo (BPPV), labyrinthine diseases (such as Meniere's disease and acute labyrinthitis) and brainstem vascular insufficiency. The mean age of 100 patients was 55.6 (SD ± 19.1) years. Sixty six were women and 34 were men.

Clinical assessment of the main presenting symptom was very important in distinguishing between the various categories of dizziness and helped achieve a final diagnosis. Patients with vertigo were significantly more likely to have otological abnormalities compared to those patients with other forms of dizziness ($p < 0.0001$).

INTRODUCTION

Dizziness is an important, non-specific symptom affecting up to 40% of the general population and is commoner in elderly women.[1,2] In a recent Swiss study which sampled 5300 individuals aged over 68 years, 7.7% of men and 12.2% of women suffered from chronic dizziness.[3] It is not surprising that this symptom may lead to recurrent falls in the elderly causing morbidity and disability.[4]

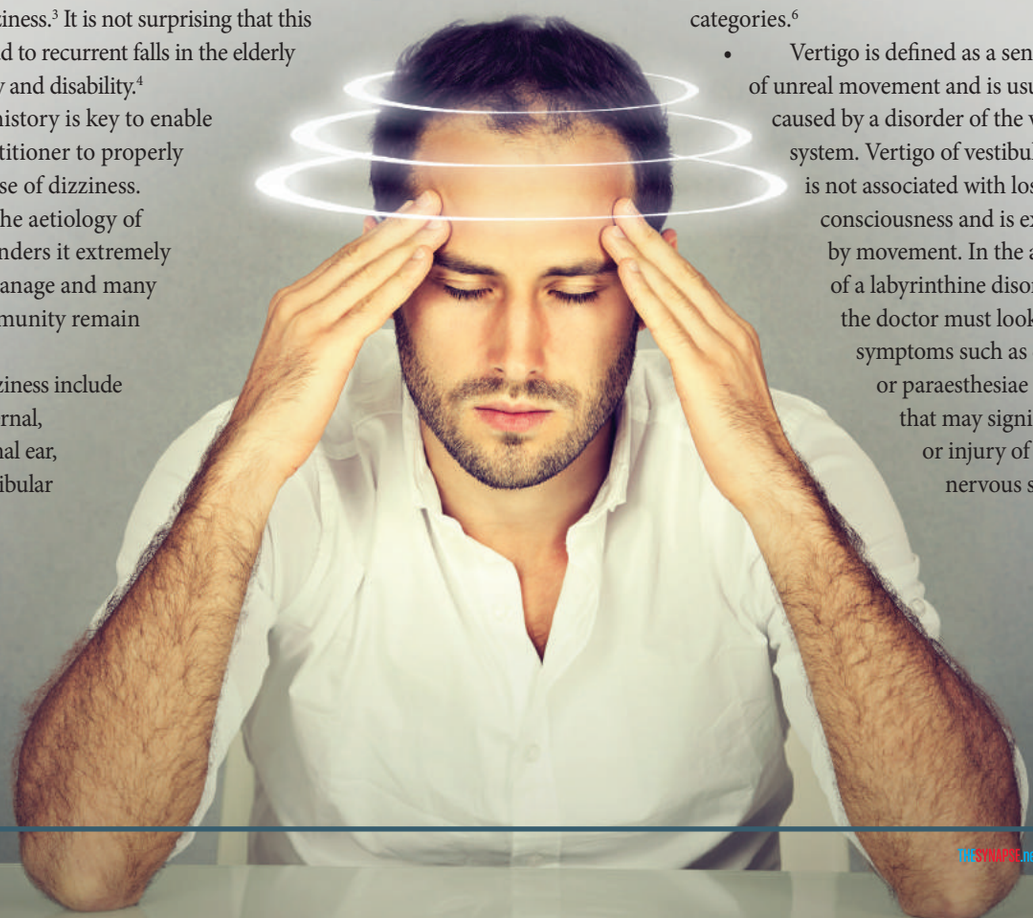
A thorough history is key to enable the medical practitioner to properly diagnose the cause of dizziness. The diversity of the aetiology of this condition renders it extremely challenging to manage and many cases in the community remain untreated.[5]

Causes of dizziness include lesions in the external, middle and internal ear, lesions in the vestibular nerve and nucleus, brainstem vascular or space-occupying lesions, other vascular or degenerative disorders of the central nervous system, metabolic disorders, ocular disorders, effects of drugs and psychological disorders.

In a postal questionnaire completed by 2064 patients attending general practices in London in 1998, approximately 23% of registered patients had previously experienced dizziness. Typically these were elderly patients, half of whom also experienced anxiety disorders. Elderly patients were more likely to have cardiovascular and neurological co-morbidities and were also more likely to be taking several different medications. Dizziness in younger people tended to be related to vestibular disorders, such as BPPV or psychiatric disorders such as depression or anxiety.[5] In a follow-up study on 1820 individuals carried out in 2006, again in London, the same authors confirmed their previous findings. Dizzy patients who consulted their family practitioner tended to be older, unemployed, having several medicines, and suffered from other medical conditions including depression and anxiety.[6] Four percent of all patients registered with these general practices suffered from persistent dizziness (of at least six months' duration), and were severely incapacitated by their symptoms.[6,7]

Dizziness can be divided into four main categories.[6]

- Vertigo is defined as a sensation of unreal movement and is usually caused by a disorder of the vestibular system. Vertigo of vestibular origin is not associated with loss of consciousness and is exacerbated by movement. In the absence of a labyrinthine disorder, the doctor must look for symptoms such as dysphagia, or paraesthesiae of the face that may signify disease or injury of the central nervous system.



The other categories of dizziness describe other sensations that do not involve unreal movement. These include:

- Unsteadiness (or disequilibrium) where real movement occurs, with either a tendency to fall or instability.
- Light headedness (or presyncope) is a sensation of feeling faint and it can arise spontaneously or while the patient is changing position from lying to sitting or standing.
- Giddiness, on the other hand, usually refers to bizarre sensations which the patient finds difficult to describe, or a combination of the above. Each of these sensations may be produced by an underlying organic disease.[6,8]

It is important to accurately describe and distinguish the presenting symptoms as gleaned from the patient history for reasons which shall be discussed below.

Available data on dizzy patients often reflect findings in individuals assessed at tertiary institutions.[4,9] Such patient samples may not represent the spectrum of conditions that present in the community to the family doctor.

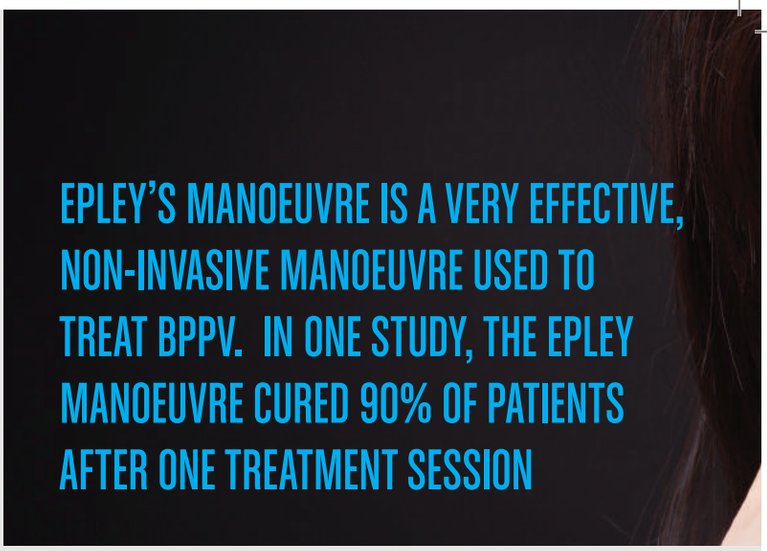
The aim of this study was to analyze the main causes of dizziness and to relate symptoms to the final diagnosis after assessment by a community–based otolaryngologist. These findings would be of practical benefit to the family practitioner as the study concentrates more on history-taking rather than investigations.

PATIENTS AND METHODS

A retrospective review of 100 patients presenting with dizziness in a community-based otolaryngology practice was conducted. Since patient data were entered on the basis of an identity number, the first 100 consecutive entries were analysed. Standardized electronic medical notes were recorded on Microsoft Access. The age of population studied ranged from 18 to 92 years. Their data were anonymised for this study so that individual patients would not be identifiable.

Cause of Dizziness	Number of patients
Labyrinthine causes (acute labyrinthitis, Meniere's disease, vestibular neuritis, seasickness)	20
Vascular causes (vascular insufficiency, carotid atherosclerosis, anaemia, hypotension, hypertension)	20
*BPPV	18
Cervical causes (spondylosis or whiplash injury)	11
Other unidentified non-otologic cause	10
Central nervous system causes (vestibular migraine, head injury, epilepsy, multiple sclerosis, jugular bulb tumour, tension type headache)	9
Sinusitis	3
Metabolic (hypoxia, metabolic)	3
Other (wax impaction, visual problems)	4
Psychological (anxiety, depression)	2
Total	100

Table 1. Main causes of dizziness. *Although BPPV is a labyrinthine cause of vertigo it is a clear cut single entity which warrants classification in its own right.



Symptoms	Diagnosis	
	Otological	Non-Otological
Vertigo (n=59)	34	25
Other (n=41)	4	37

Table 2. The relationship between symptoms and diagnosis of dizziness.

Demographic data such as age and gender were collected. Symptoms and final diagnosis were also recorded. In ten patients, the final diagnosis was not established due to normal clinical findings and investigation results, although otological causes of dizziness were excluded. Three patients had dizziness of multifactorial origin and were classified according to the main cause for the sake of clarity.

Data were retrieved, stored and analyzed using Microsoft access and this data was exported to an excel spreadsheet for mathematical analysis. For comparison between vertigo group and the group with other sensation, a chi squared test was used for statistical analysis.

RESULTS

The mean age of 100 patients was 55.6 (SD±19.1) years of which 66 were women and 34 were men. Fifty-nine patients presented with true vertigo as their main complaint, that is, with a sense of unreal movement. The rest (41 patients) presented with other symptoms such as unsteadiness, lightheadedness or giddiness as described in the introduction. The main causes of dizziness are categorized and summarized in Table 1. Ten patients had an unknown cause of dizziness where otological pathology had been excluded.

Symptoms were correlated with the final diagnosis paying attention to the presenting complaint, whether 'vertigo' or 'other' sensation (Table 2). Patients with an otologic diagnosis of dizziness included those with BPPV and labyrinthine origin. Four patients with an otologic cause had a dizzy sensation which was not vertigo.

Twenty five patients with vertigo had a non-otologic cause of their symptoms such as vascular or cervical while 37 patients with vascular, cervical, metabolic or other cause of dizziness had a sensation which was not vertigo. Patients presenting with vertigo were significantly more likely to have an otological

diagnosis whereas patients presenting with other types of dizziness were significantly more likely to have a non-otological diagnosis, such as vascular insufficiency or central nervous system disorders ($p<0.0001$).

DISCUSSION

Dizziness is a difficult complaint to analyze because it can arise from a large number of causes, or can even occur as a result from combinations of such causes. In the latter case, it may be difficult to assess the proportion that each cause contributes to the patient's symptoms. The elderly, for example, are particularly prone to cervical spondylosis, inadequate carotid and vertebrobasilar arterial blood supply, and metabolic dysfunction such as hypoglycaemia or hypoxia due to airway or chronic lung pathology.

It is important to take a detailed history of the patient's presenting symptoms. In this study, patients presenting with vertigo were significantly more likely to have an otological cause of their symptoms while other types of dizziness were more likely to be due to non-otological causes such as vascular or central nervous disorders.

The commonest single cause of vertigo in this investigation was BPPV which affects all age groups but is more common in the elderly.[10] Concurrent vascular conditions like hypertension and metabolic conditions such as diabetes may prolong recovery time or increase the likelihood of relapse in BPPV patients.[11] Epley's manoeuvre is a very effective, non-invasive manoeuvre used to treat BPPV. In one study, the Epley manoeuvre cured 90% of patients after one treatment session.[12]

Vestibular migraine is a migraine variant characterized by spontaneous episodic vertigo lasting seconds to days and may not always be associated with headaches. It is a common cause of spontaneous episodic vertigo where the pathophysiology is thought to affect the central or peripheral vestibular system.[13] Prophylactic drugs such as beta-blockers or anticonvulsants, besides non-pharmacological treatments such as sleep and avoidance of triggers are most useful in the management of this vestibular migraine.[13]

Vascular insufficiency of the vertebrobasilar vessels, transient ischaemic attacks and migraine may all present as positional vertigo or vertigo without neurological signs. Cerebellar ischaemia, for example, may bring about symptoms of acute vertigo.[14] In a study conducted by Cloutier and Saliba,[15] up to 52% of patients with isolated vertigo of unclear aetiology had posterior circulatory anomalies. This may explain at least a proportion of the 10 patients in this study who had dizziness of unknown origin.

Epidemiological studies have shown dizziness to be commoner in women and the present study supports this finding.[10]

Dizziness is often associated with anxiety and agoraphobia, the latter being more prevalent in older patients. It is also one of the leading symptoms of panic disorder accompanied by hyperventilation. Yardley et al.[5] found that more than half of the patients with anxiety also experienced dizziness. Patients who reported dizziness when they were not suffering from anxiety were more likely to have true vertigo.[5]

This study studied the key presenting complaint in 100 patients with dizziness and compared this with the end diagnosis. It is not a large study in terms of numbers and was not prospective. However, it examined in a detailed qualitative way the symptoms, examination findings, outcome and final diagnosis in a sizable group of dizzy patients presenting in a community setting in Malta, where such data has previously not been collected.

CONCLUSION

Dizziness is very common in the community, generally affecting the elderly and is more common in women. The clinical history is important in distinguishing the actual sensation described by patients as this is significantly related to the final diagnosis. Differentiation of true vertigo as a sensation of unreal movement in contrast with other sensations of dizziness is very useful in the clinical setting but requires practice in history-taking and experience.

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† Based on 2016 ESC HF Guidelines and 2017 ACC/AHA/HFSA Guideline Update.

‡ Primary end point.

§ Secondary end point that measured the change from baseline to 8 months in the clinical summary score on the Kansas City Cardiomyopathy Questionnaire (KCCQ).

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Warnings/Precautions: Dual blockade of the renin angiotensin-aldosterone system (RAAS): Combination with an ACE inhibitor is contraindicated due to the increased risk of angioedema. Entresto must not be initiated until 36 hours after taking the last dose of ACE inhibitor therapy. If treatment with Entresto is stopped, ACE inhibitor therapy must not be initiated until 36 hours after the last dose of Entresto. Combination of Entresto with direct renin inhibitors such as aliskiren is not recommended. Entresto should not be co-administered with another ARB containing product. Hypotension: Treatment should not be initiated unless SBP is ≥100 mmHg. Patients with SBP <100 mmHg were not studied. Cases of symptomatic hypotension have been reported in patients treated with Entresto during clinical studies, especially in patients ≥65 years old, patients with renal disease and patients with low SBP (<112 mmHg). Blood pressure should be monitored routinely when initiating or during dose titration with Entresto. If hypotension occurs, temporary down-titration or discontinuation of Entresto is recommended. Impaired or worsening renal function: Limited clinical experience in patients with severe renal impairment (estimated GFR <30 ml/min/1.73m2). There is no experience in patients with end-stage renal disease and use of Entresto is not recommended. Use of Entresto may be associated with decreased renal function, and down-titration should be considered in these patients. Impaired renal function: Patients with mild-moderate renal function are more at risk of developing hypotension while patients with severe renal impairment may be at a greater risk of hypotension. Entresto is not recommended in patients with end-stage renal disease. Hyperkalaemia: Entresto should not be initiated if the serum potassium level is >5.4 mmol/l. Monitoring of serum potassium is recommended, especially in patients who have risk factors such as renal impairment, diabetes mellitus or hypoaldosteronism or who are on a high potassium diet or on mineralocorticoid antagonists. If clinically significant hyperkalaemia occurs, consider adjustment of concomitant medicinal products or temporary down-titration or discontinuation of Entresto. If serum potassium level is >5.4 mmol/l discontinuation should be considered. Angioedema: Angioedema has been reported with Entresto. If angioedema occurs, discontinue Entresto immediately and provide appropriate therapy and monitoring until complete and sustained resolution of signs and symptoms has occurred. Entresto must not be re administered. Patients with a prior history of angioedema were not studied. As they may be at higher risk for angioedema, caution is recommended if Entresto is used in these patients. Black patients have an increased susceptibility to develop angioedema. Patients with renal artery stenosis: Caution is required and monitoring of renal function is recommended. Patients with NYHA functional classification IV: Caution should be exercised due to limited clinical experience in this population. Patients with hepatic impairment: There is limited clinical experience in patients with moderate hepatic impairment (Child Pugh B classification) or with AST/ALT values more than twice the upper limit of the normal range. Caution is therefore recommended in these patients. B-type natriuretic peptide (BNP): BNP is not a suitable biomarker of heart failure in patients treated with Entresto because it is a neprilysin substrate. Interactions: Contraindicated with ACE inhibitors, 36 hours washout is required. Use with aliskiren contraindicated in patients with diabetes mellitus or in patients with renal impairment (eGFR <60 ml/min/1.73 m2). Should not be co-administered with another ARB. Use with caution when co-administering Entresto with statins or PDE5 inhibitors. No clinically relevant drug-drug interaction was observed when simvastatin and Entresto were co-administered. Monitoring serum potassium is recommended if Entresto is co-administered with potassium-sparing diuretics or substances containing potassium (such as heparin). Monitoring renal function is recommended when initiating or modifying treatment in patients on Entresto who are taking NSAIDs concomitantly. Interactions between Entresto and lithium have not been investigated. Therefore, this combination is not recommended. If the combination proves necessary, careful monitoring of serum lithium levels is recommended. Co-administration of Entresto and furosemide reduced Cmax and AUC of furosemide by 50% and 28%, respectively, with reduced urinary excretion of sodium. Co-administration of nitroglycerin and Entresto was associated with a treatment difference of 5 bpm in heart rate compared to the administration of nitroglycerine alone, no dose adjustment is required. Co administration of Entresto with inhibitors of OATP1B1, OATP1B3, OAT3 (e.g. rifampicin, ciclosporin), OAT1 (e.g. tenofovir, cidofovir) or MRP2 (e.g. ritonavir) may increase the systemic exposure of LBQ657 or valsartan. Appropriate care should be exercised. Co-administration of Entresto with metformin reduced both Cmax and AUC of metformin by 23%. When initiating therapy with Entresto in patients receiving metformin, the clinical status of the patient should be evaluated. Fertility, pregnancy and lactation: The use of Entresto is not recommended during the first trimester of pregnancy and is contraindicated during the second and third trimesters of pregnancy. It is not known whether Entresto is excreted in human milk, but components were excreted in the milk of rats. Entresto is not recommended during breastfeeding. A decision should be made whether to abstain from breast feeding or to discontinue Entresto while breast feeding, taking into account the importance of Entresto to the mother. Undesirable effects: Very common (≥ 1/10): Hyperkalaemia, hypotension, renal impairment. Common (≥ 1/100 to <1/10): Anaemia, hypokalaemia, hypoglycaemia, dizziness, headache, syncope, vertigo, orthostatic hypotension, cough, diarrhoea, nausea, gastritis, renal failure, acute renal failure, fatigue, asthenia. Uncommon (≥ 1/1,000 to <1/100): Hypersensitivity, postural dizziness, pruritis, rash, angioedema. Packs sizes: Entresto 24 mg/26 mg – x28 tablets; Entresto 49 mg/51 mg – x28 tablets; Entresto 97 mg/103 mg - x28 & x56 tablets. Legal classification: POM. Marketing Authorisation Holder: Novartis Europharm Ltd, Frimley Business Park, Camberley, GU167SR, United Kingdom. Marketing Authorisation Numbers: Entresto 24 mg/26 mg film coated tablets EU/1/15/1058/001; Entresto 49 mg/51 mg film coated tablets EU/1/15/1058/002-004; Entresto 97 mg/103 mg film coated tablets EU/1/15/1058/005-007. Please refer to the Summary of Product Characteristics (SmPC) before prescribing. Full Prescribing Information is available on request from Novartis Pharma Services Inc., Representative Office Malta, P.O. Box 4, Marsa, MRS 1000, Malta. Tel: +356 21222872. 2016-MT-ENT-16-JUN-2016

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NOVARTIS

ENT Ad1 12/17 MT

Eight key symptoms of heart failure

Even though heart failure is a **chronic condition**, it can be **effectively managed**. This means that being able to recognize and check the symptoms of chronic heart failure is especially important. Learn more about the signs and symptoms of chronic heart failure below.

Shortness of breath

Heart failure can cause the fluid in your body to gather in your lungs which may cause you to feel short of breath during everyday activities.

Shortness of breath when lying down

Lying flat may also make you feel short of breath so that you need to sleep sat up or with multiple pillows.

Rapid heartbeat

The heart sometimes starts to speed up to compensate for its reduced ability to pump blood around the body.

Loss of appetite

A build-up of fluid around the gut can affect digestion and might cause a loss of appetite, or make you feel sick when eating.

Tiredness

Heart failure means less oxygen-rich blood is circulating the body. Because your muscles and tissues need oxygen for energy, this means people with heart failure can feel tired very easily.

Swelling in the ankles, legs and abdomen

Clothes or shoes might feel tighter as fluid in the body builds up in the legs, ankles or abdomen causing them to swell up.

Sudden weight increase

Worsening heart failure may cause an increase in weight of more than two kilograms (about six pounds) in one week because fluid builds up in your body.

Frequency of urination

A reduced amount of blood reaches your kidneys when you have heart failure, causing you to urinate less frequently. Conversely, if you take diuretics (eg. water pills), you might urinate **more** frequently, when the excess fluid in your body is eliminated.

To learn more about the symptoms of heart failure, and how you can check and manage them, explore **www.KeepItPumping.com**.

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Keep It Pumping™

www.KeepItPumping.com

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CASE REPORT

A CASE REPORT AND OVERVIEW OF CARBAMATE INSECTICIDE (BAYGON®) POISONING

PETER MUSCAT & MARLENE ATTARD

ABSTRACT

The following is a case report of a 55-year-old gentleman who experienced excessive exposure to Baygon®, a local carbamate insecticide spray. He developed symptoms that were compatible with cholinesterase inhibition, namely headache, light-headedness, confusion, bad taste, nausea and fatigue. This report demonstrates that the misapplication of insecticides commonly used in residences can cause acute poisoning.

Keywords: insecticides, carbamate poisoning, Baygon®

INTRODUCTION

Poisoning refers to the damaging physiological effects of inhalation, ingestion, skin contact or other exposure to pharmaceuticals, drugs and chemicals, including pesticides, heavy metals, gases/vapours and common household cleaning substances.[1] Poisoning is a real health problem in every country of the world.

Carbamate pesticides are derived from carbamic acid and kill insects in a similar fashion as organophosphate insecticides. They are widely used in homes, gardens and agriculture. Their mode of action is by inhibition of cholinesterase enzymes, affecting nerve impulse transmission. The first carbamate, carbaryl, was introduced in 1956 and has been most widely used throughout the world, more than all other carbamates combined.[2] Most of the carbamates are extremely toxic to foraging parasitic wasps, ants and bees.

CASE REPORT

A 55-year-old gentleman was admitted to the emergency department of Gozo General hospital suffering from sudden onset of headache accompanied by very bad taste, severe dizziness, bouts of confusion and light-headedness. He had no difficulty with breathing. He did not cough or vomit. His past medical history was unremarkable with no history of head injury, nose trauma, diabetes or asthma. According to his wife the episode started 45 minutes before admission while he was helping her prepare dinner. On further questioning he admitted that he had sprayed half a bottle of baygon insecticide in the cellar to which he remained exposed for 20 minutes.

On examination, the patient, who was a well-built man, was conscious but not oriented. Oxygen saturation was 98%. His pulse was regular at 78 beats/min and his blood pressure was 139/88 mmHg. Neurological examination was unremarkable. Laboratory results showed a leucocytosis of 15.3x10(9)/L (range from 4.3 to 9.8x10(3)/L). Liver function and kidney function tests were normal. Arterial blood gas analysis showed metabolic acidosis with pH 7.31, pCO2 of 31 mmHg, pO2 of 96 mmHg and bicarbonate of 16 mmol/L. An urgent CT scan of the brain and paranasal sinuses was done immediately on admission and was reported as normal. The chest X-ray showed no abnormalities. An electrocardiogram showed no conduction abnormalities. A preliminary diagnosis of poisoning due to inhalation of Baygon® insecticide was made. The patient was admitted to the intensive therapy unit. Continuous cardiac monitoring as well as continuous oxygen support and chest physiotherapy were done. Fortunately the clinical signs and symptoms did not require anticholinergic treatment. After two days at the intensive care unit the patient was stable and was transfered to the general ward and eventually discharged home after four days.

DISCUSSION

MECHANISM OF TOXICITY

The signs and symptoms of carbamate poisoning are similar to those caused by organophosphate pesticides. The carbamate's principal route of entry is by inhalation or ingestion or secondarily through dermal route. Dermal exposure tends to be less toxic than inhalation or ingestion.[2] The carbamates are hydrolysed enzymatically by the liver and the metabolites are excreted by the kidneys and the liver. As with organophosphates, the signs and symptoms are based on excessive cholinergic stimulation. Unlike organophosphate poisoning, carbamate poisoning tends to be of shorter duration. This is because the inhibition of nervous tissue acetylcholinesterase (AChE) is more easily reversible. The carbamylation of the enzyme is unstable, and the regeneration of AChE is relatively rapid compared with that from a phosphorylated enzyme. Because of this, carbamate pesticides are less dangerous with regard to human exposure than organophosphorus pesticides. In keeping with this, the ratio between the dose required to produce death and the dose required to produce minimum symptoms of poisoning is substantially larger for carbamate compounds than for organophosphorus compounds.[2]

CLINICAL PRESENTATION

The most commonly reported early symptoms are muscle weakness, dizziness, sweating and slight body discomfort. Higher levels of exposure present with headache, salivation, nausea, vomiting, abdominal pain and diarrhoea. Contraction of pupils with blurred vision, incoordination, confusion, muscle twitching and slurred

speech have also been reported. Respiratory depression together with pulmonary oedema is the usual cause of death from poisoning by carbamate compounds. Because of their chemical structure, carbamates do not cause delayed neuropathy.[3]

FIRST AID MEASURES

***First aid for the eyes*:** The eyes should be rinsed gently with water, ideally running tap water for 15-20 minutes. Contact lenses should be removed if present.

First aid for the skin: Contaminated clothing should be removed and the skin rinsed immediately with plenty of water for 15-20 minutes. The patient's clothes should be discarded since they absorb carbamate agents, and re-exposure may occur even after washing.

***First aid for inhalation*:** The person should be moved to an uncontaminated area so that the patient can breath fresh air. If the person is not breathing one should call for an ambulance then start artificial respiration, preferably mouth–to-mouth, if possible.

TREATMENT OF ACUTE TOXICITY

In our case the patient only needed continuous 100% oxygen via facemask and cardiac support at the intensive therapy unit with spontaneous recovery. In moderate to severe cases the following treatment may be needed.

ATROPINE

Atropine should be given beginning with 2mg IV repeated at 15 to 30-minute intervals. Once atropinized, a maintenance dose at 1-3mg half hourly is usually sufficient. The dose and the frequency of atropine treatment varies from case to case, but should maintain the patient fully atropinized (dilated pupils, dry mouth, skin flushing, normal pulse rate and good mental state). The total dose and duration of atropine use depends on the type and amount of carbamate compound consumed.[4]

VENTILATORY SUPPORT

Intubation is strongly considered in moderate to severe poisoning. Patients who appear mildly poisoned may rapidly develop respiratory failure due to a combination of CNS depression, niconitic receptor-mediated diaphragmatic weakness, bronchospasm and copious secretions.[4]

OXIME REACTIVATORS

There is no rational basis for using these drugs. Furthermore, some unconfirmed reports suggest an increased toxicity of carbamates when oximes are administered.[4]

BENZODIAZEPINE THERAPY

Diazepam 0.1-0.2 mg/kg IV can be given and repeated as necessary if seizures occur. The early use of diazepam may reduce morbidity and mortality.

FLUID AND ELECTROLYTE BALANCE

Patients may require extra fluids and electrolytes to compensate for the loss due to vomiting, high fever, diarrhoea and for decreased intake. Other diseases like diabetes, hypertension, heart failure or complications like aspiration pneumonia have to be dealt with and treated.[5]

SYMPATHETIC AND CARING DISCUSSION WITH PATIENTS

Self-poisoning using carbamates is very common in developing countries. Since first described as a problem in India over 40 years ago, this problem has increased with India and Sri Lanka being the countries with the highest number of cases. Death from pesticide poisoning was the 6th most common cause of death in Sri Lanka in 2003.[4] Patients who survive self-poisoning require an empathic and caring approach by the treating doctors and nurses. These patients may require referral for psychiatric treatment.[5]

GASTRIC LAVAGE AND ACTIVATED CHARCOAL

Emptying the stomach by gastric lavage is most useful if attempted within 1 to 2 hours after ingestion of the poison. If the patient is unconscious, the time elapsed since ingestion may be less relevant since the gastrointestinal stasis which often accompanies coma can delay gastric emptying. It is therefore recommended that gastric lavage is carried out in every unconscious poisoned patient.[6]

CONCLUSION

Pesticides are widely used in buildings and agriculture throughout the Maltese islands. This case report is intended to draw the attention of clinicians to the potential exposure of carbamates and organophosphate pesticides which may cause poisoning.

Manufacturers need to be persuaded to compulsary market their pesticides in tamper-proof packages along with labels mentioning their poisonous nature, contents and antidote in the unfortunate instances of accidental exposure, in conformity with international standards. Legislation and law enforcement against substandard packaging is warranted.[7]

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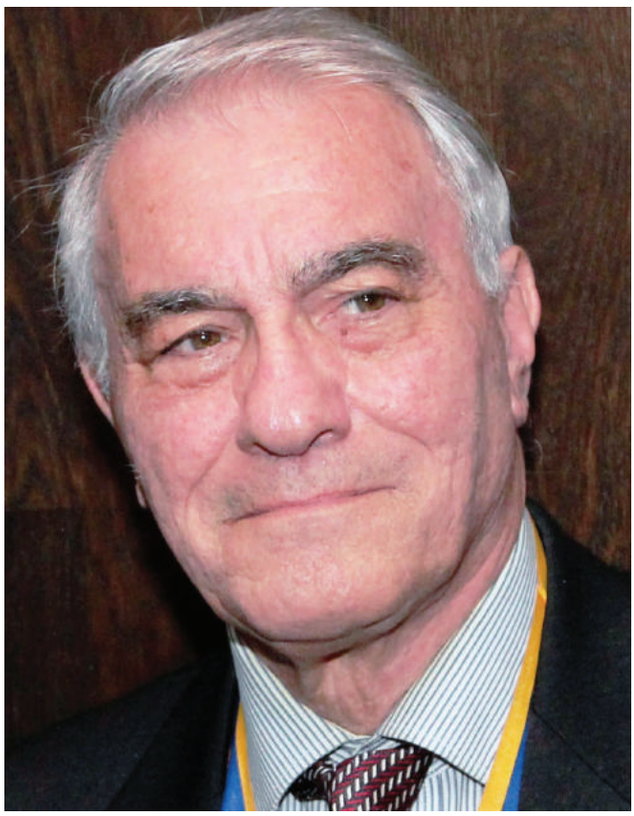
FIFTY YEARS A SURGEON

PROFESSOR CARMEL LINO CUTAJAR SHARES SOME MEMORIES WITH MARIKA AZZOPARDI



TS: WHAT WERE THE KEY EVENTS THAT MARKED YOUR FIRST YEARS AS A DOCTOR AND SURGEON?

I graduated in Malta in 1964. I married my wife Irene in early 1966. We immediately proceeded to London for me to commence my surgical training. I was lucky to be attached to one of the best teaching hospitals, the Middlesex Hospital. My main objective was to train in general surgery which meant a lot of hard work. After I was admitted by examination to Fellowship of the Royal Colleges of Surgeons of England and Edinburgh I decided to pursue further specialisation. I was lucky to be offered a Commonwealth Scholarship which allowed me to do two years full-time research in vascular surgery. I presented my research work in haemodynamics, which at the time was ground-breaking, to the Surgical Research Society and to various reputable journals. Just when I thought that my training was complete an opportunity unexpectedly arose to join the urology unit of Sir Eric Riches, the doyen of urology at the time. I was hooked and spent two further years of training in that speciality. Therefore when we finally did return to Malta (now with two young children) I was able to practice general, vascular and urological surgery. The latter would eventually be my dominant interest.

Saudi Arabia, 1980. Lavish Bedouin hospitality. Prof. Cutajar is 2nd from left dressed in Arab garb as requested by his host (on my right). His wife, Irene, and the two children, David and Louise, are on the right

TS: YOU AGAIN LEFT MALTA IN 1977. WHY WAS THAT?

Unfortunately in 1977 there was a head-on clash between the Government of the time and the medical profession. I will not go into this dispute except to mention that as a result the vast majority of Maltese specialists in all departments left country, myself included. With my family I went to Saudi Arabia where I was engaged in an American-run hospital with the main objective of opening a specialised urology department. We initially spent over four years in Saudi Arabia before the same American organisation asked me to move to Abu Dhabi (Emirates) for another 30 months. On the one hand these seven years in the Middle East were very exciting as we were exposed to new cultures and experiences. The negative side, however, was that due to lack of adequate education facilities we had to send our two very young children to boarding schools in England.

TS: BACK IN MALTA IN 1984, YOU WERE APPOINTED CONSULTANT SURGEON AND UROLOGIST AT ST LUKE'S HOSPITAL. CAN YOU ELABORATE?

Certainly. My appointment at St Luke's Hospital was primarily as urologist but also undertaking general surgical work. Opening the Department of Urology was a big challenge as one needed to focus and absorb the spectacular advances which urology was experiencing. I was also entrusted with the opening of the

Receiving the diploma of Fellow of the Royal college of Surgeons of England from the President, Sir Thomas Holmes Sellors in London, 1971

Endoscopy Unit. This was the first-ever dedicated day-care surgery department, a novelty at the time. In 1986 we started the technique of percutaneous nephro-lithotripsy (key-hole surgery for the removal of kidney stones), a very sophisticated and advanced technology. We were among the first in Europe to do so which greatly enhanced the reputation of our urology department still in its infancy.

TS: IN 1991 YOU WERE APPOINTED DIRECTOR OF SURGERY. CAN YOU COMMENT ON THIS NEW DEVELOPMENT?

My appointment as Director of Surgery within the Department of Health and Professor and Head of the academic department of surgery in the medical school ushered in a period of intense activity which would last a full ten years. One should remember that at that time we were emerging from the trauma of the events I mentioned before. I was lucky to have a young team of excellent surgeons, mainly trained in the UK. Thanks to their support we managed to push surgery to new heights. New sub-specialities (such as urology, cardiac, plastic, vascular) were introduced to strengthen the ones already available. I pressed for the opening of a day-care unit. We managed to start the service in a rather restricted space area. Nevertheless it was an immediate success liberating hundreds of in-patient beds. Having already established keyhole surgery for the removal of kidney stones we then turned to abdominal keyhole surgery, pioneered by our famous compatriot, and my great friend, Professor Sir Alfred Cuschieri working in Scotland. On the academic side we introduced structured tuition and encouraged surgical trainees to do at least part of their surgical training in Malta. I am impressed by the great progress made in this respect in the last few years. During this period I was also involved in expanding my own speciality of urology. I became member of several European urological societies and also took part in research programmes as a member of the European Organisation for Cancer Research. I was also Founder Member and President of the Association of Surgeons of Malta as well as Founder Member of the European Society of Surgery of which I was President in 2005 and still on its board.

Investiture in the Sovereign Military Order of St John, 2008

TS: IS THERE TIME FOR ANYTHING OTHER THAN MEDICINE IN YOUR LIFE?

I lead a very active social life. I have been President of the Malta Red Cross and of Rotary Club Malta. I am also a Knight of the Order of St John. I like to read especially biographies, history of art and, of course, medical journals. I enjoy classical music and opera. My other hobby is medical philately. I love watching football. I support Arsenal in the English Premier League. I have travelled extensively with my wife.

TS: YOU RECENTLY PUBLISHED YOUR OWN AUTOBIOGRAPHY. WHY?

My book entitled *'In at the Deep End'* was published by Progress Press and launched by Her Excellency the President Marie Louise Coleiro Preca. I decided to write it to explain, perhaps also to myself, the developments in my life and career as a doctor and surgeon in Malta and abroad, as well as to provide an overview of the social and historical events which marked Malta in those years and of the medical developments which concerned my profession. I dedicated the book to my wife, Irene, who was a beacon of strength for me but who unfortunately passed away before she could read our shared experiences.

The Urology team on Prof. Cutajar's retirement, 2001. From left Surgeons P. Zammit, S. Mattocks, Prof. Cutajar, K. German, R. Jovanovic, and two head nurses Stephen Balzan [2nd from left, partially hidden] & Maria Pace [far right]

I READ THE SYNAPSE BECAUSE...

It is like a breath of fresh air keeping me abreast with various aspects of medicine beyond my speciality.

AZITHRIN

Azithromycin, 500mg film-coated tablets

Abridged Prescribing Information: *Please refer to Full Summary of Product Characteristics before prescribing.* **Name of Medicinal Product:** Azithrin. **Composition:** Azithromycin dihydrate equivalent to 500 mg azithromycin per tablet. **Pharmaceutical form:** Film-coated tablets. **Indications:** The treatment of respiratory tract infections of mild to moderate severity caused by susceptible strains of microorganisms, such as: *Lower respiratory tract infections (when a susceptible pathogen sensitive to azithromycin in vitro has been isolated or is suspected)*;Acute bacterial exacerbations of chronic bronchitis due to *Haemophilus influenzae*, *Moraxella catarrhalis*, *Haemophilus parainffuenzae* or *Streptococcus pneumonia*; Community-acquired pneumonia due to *Chlamydophila pneumonia*, *Haemophilus influenzae*, *Haemophilus parainfluenzae*, *Moraxella catarrhalis*, *Mycoplasma pneumoniae* or *Streptococcus pneumonia* (for pneumonia see note at the end of section); *Upper respiratory tract infections:* Acute bacterial sinusitis due to *Haemophilus influenzae*, *Moraxella catarrhalis*, *Streptococcus pneumoiae*. Tonsillopharyngitis due to *Streptococcus pyogenes*. **Posology and method of administration:** Azithromycin should be administered once daily. Azithromycin tablets may be taken with food. Use in adults and adolescents (› 12 years old): For the treatment of sexually transmitted diseases due to *Chlamydia trachomatis*, *Haemophilus ducreyi* or susceptible strains of *Neisseria gonorrhoeae*, the dose of the drug is 1000 mg taken as a single oral dose. For all the other indications the total dose is 1500 mg administered as daily doses of 500 mg for three days. As an alternative treatment, the same total dose of the drug may be administered within a 5-day period: 500 mg are administered on the first day of treatment and then a daily dose of 250 mg is administered from the 2nd up to and including the 5th day. Elderly patients: No dose adjustment is required for elderly patients that require therapy with azithromycin. Patients with renal failure: No dose adjustment is recommended in patients with mild to moderate renal failure (GFR 10 - 80 ml/min). Caution should be exercised when azithromycin is administered to patients with severe renal impairment (GFR ‹ 10 ml/min). Patients with hepatic impairment: No dose adjustment is recommended in patients with mild to moderate hepatic failure. Azithromycin can be administered to people with severe hepatic impairment. **Contraindications:** The use of the product is contraindicated in patients with hypersensitivity to azithromycin, erythromycin, any macrolide or ketolide antibiotic, or to any of the excipients. (Refer to full Summary of Product characteristics for further information). Concomitant administration of macrolides with cisapride is contraindicated. **Special warnings and precautions for use:** As with erythromycin and other macrolides, rare serious allergic reactions including angioneurotic oedema and anaphylaxis (rarely fatal), have been reported. Some of these reactions occurring after azithromycin administration have resulted in recurring symptoms that required a longer period of observation and treatment. Since the liver is the primary route of elimination for azithromycin, it should be used with caution in patients with severe hepatic disease. Cases of fulminant hepatitis potentially leading to life-threatening hepatic failure have been reported with the use of azithromycin. In patients presenting signs and symptoms of hepatic impairment such as asthenia, jaundice, dark urine, bleeding tendency or hepatic encephalopathy, the relevant liver function tests should be performed. In patients receiving ergot derivatives, ergotism has occurred following administration of certain macrolide antibiotics. As with any other antibiotic, monitoring of patients for signs of superinfection by non-susceptible micro-organisms, including fungi, is recommended. *Clostridium difficile* associated diarrhoea (CDAD) has been reported during use of nearly all antibacterial agents, including azithromycin, which may range in severity from mild diarrhoea to fatal colitis. (Refer to full Summary of Product characteristics for further information). In patients with severe renal impairment (GFR ‹ 10 ml/min) a 33% increase in systemic exposure to azithromycin has been observed. Prolonged cardiac repolarisation and QT interval, entailing a risk of developing cardiac arrhythmia and torsades de pointes, have been observed during treatment with other macrolides. A similar effect with azithromycin cannot be completely excluded in patients with increased risk of prolonged cardiac repolarisation, therefore caution should be exercised in treating patients: With congenital or demonstrated QT prolongation ; Currently receiving treatment with other active substances known to prolong QT interval such as antiarrhythmics of Class IA and III, cisapride and terfenadine; With electrolyte disturbances, particularly in cases of hypokalaemia and hypomagnesaemia; With clinically relevant bradycardia, cardiac arrhythmia or severe cardiac failure. Exacerbations of the symptoms of myasthenia gravis and new onset of myasthenia syndrome have been reported in patients receiving azithromycin therapy. With respect to treatment of pneumonia, azithromycin has only been proven to be safe and effective in the treatment of mild community-acquired pneumonia due to *Streptococcus pneumoniae* or *Haemophilus influenzae* in patients considered eligible for outpatient oral treatment. Azithromycin should not be used in patients with pneumonia, who are considered ineligible for outpatient oral treatment due to moderate or severe infection or due to any of the following risk factors: patients affected by hospital-acquired pathogens; patients with known or suspected bacteraemia; patients requiring hospitalization; elderly or debilitated patients or patients with co-existing significant health problems that may compromise their ability to respond to their illness (including immunodeficiency or functional asplenia). **Interactions:** antacids; digoxin; ergot alkaloids; Coumarin – type oral anticoagulants; cyclosporin; terfenadine; theophylline; cisapride. **Pregnancy and lactation:** There are no data on the excretion of azithromycin in human milk. Similar to many drugs excreted in human milk, azithromycin should not be used for the treatment of breast-feeding women, unless the doctor believes that the potential benefits justify the potential risks to the infant. **Effects on the ability to drive and use machines:** There is no evidence to demonstrate that azithromycin may have an effect on a patient's ability to drive or operate machines. **Undesirable effects:** Common (≥1/100 to ‹1/10):Anorexia; visual impairment; deafness; pruritus; rash; arthralgia; fatigue; lymphocyte count decreased, eosinophil count increased, blood bicarbonate decreased. **Market Authorisation Holder:** Alet Pharmaceuticals S.A., 31-33, Athinon Ave.,104 47, Athens – Greece. **Marketing Authorisation Number:** 44313/11 – 20/04/2012. **Legal Category:** POM. Date of revision of text: March 2011. Adverse effects should be reported to the Malta Medicines Authority via the ADR Reporting Website: www.medifinesauthority.gov.mt/adrportal. Adverse events should also be reported to the phv@actavis.com.mt

Always read the package insert. For further information please refer the Full Summary of Product Characteristics on **www.actavis.com.mt**

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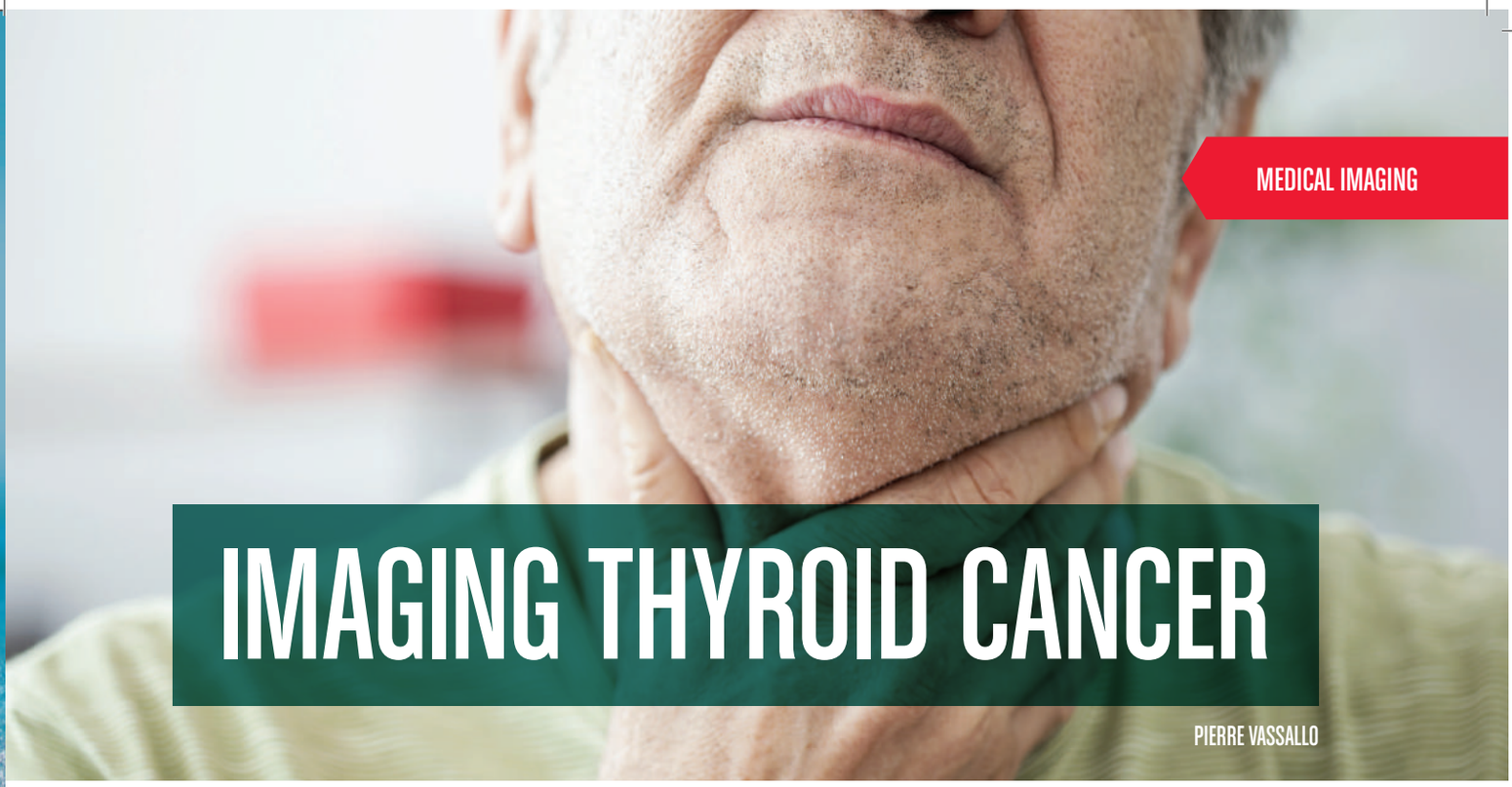

IMAGING THYROID CANCER

PIERRE VASSALLO

There are four main types of thyroid cancer: papillary, follicular, medullary and anaplastic types. Papillary and follicular cancers are well-differentiated cancers that originate from the thyroid follicle. They are the more common types of thyroid cancer, accounting for 80% and 10% of cases, respectively.[1]

Most clinically and ultrasound-detected thyroid nodules are benign, many representing purely cystic lesions known as colloid cysts. Solid or partly solid nodules however, should be further investigated since approximately one in four will be malignant.

Although differentiated thyroid cancers are generally slow growing, lymph node metastases are present in 30-80% of cases at the time of first diagnosis.[2] The outcome of treatment is good with a 98% 5-year survival,[3] but these results depend on proper surgical treatment.

The presence of lymph node metastases is the most common cause of thyroid cancer recurrence. Since most recurrences occur within the first five post-operative years, they may likely result from inadequate detection and resection at the time of initial management.[4] Physical examination has shown poor accuracy in detection of lymph node metastases. Accurate staging of thyroid cancer depends on meticulous ultrasound evaluation and detailed reporting, which are crucial for accurate surgical planning.

NECK ANATOMY

To ensure a clear communication between the radiologist and the surgeon, we share a common nomenclature for indicating the location of abnormal ultrasound findings. The neck is divided into seven levels as shown in figure 1. The aim is to standardise terminology related to surgical dissections for head and neck cancer.

Level VI is considered the central compartment and it contains the thyroid gland; it is bordered by the hyoid bone superiorly, the carotid arteries and sternocleidomastoid muscles laterally and the sternal notch inferiorly. This is the most common site for lymph node metastases and cancer recurrence.

The lateral compartment is mainly composed of levels II-VI with levels I and VI being less frequently involved; metastatic disease in this compartment is associated with a worse prognosis.

SURGICAL TECHNIQUE

There are multiple surgical procedures that can be performed to treat thyroid cancer; the aim however is to excise all locations of disease, as surgical excision is the most effective mode of therapy.

All solid or partly solid nodules require biopsy. This consists of a fine needle aspiration (FNA) that is performed under ultrasound guidance. Biopsy-confirmed malignant nodules are excised and the surgical approach depends on the location and number of malignant nodules found.

Unilateral nodules ≤1cm in diameter are treated with thyroid lobectomy, while nodules >4cm in diameter require a total thyroidectomy. For solitary nodules measuring 1-4cm in diameter with no lymph node metastases, a thyroid lobectomy is usually performed although some surgeons prefer to perform a total thyroidectomy. For bilateral malignant nodules and in the presence of regional or distant metastases a total thyroidectomy

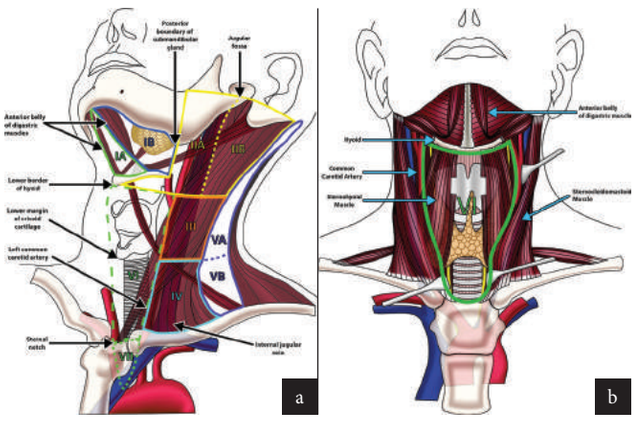

Figure 1a: Diagram shows the boundaries of the different levels of the neck. (b) An anterior view shows the boundaries between the central and lateral compartments.

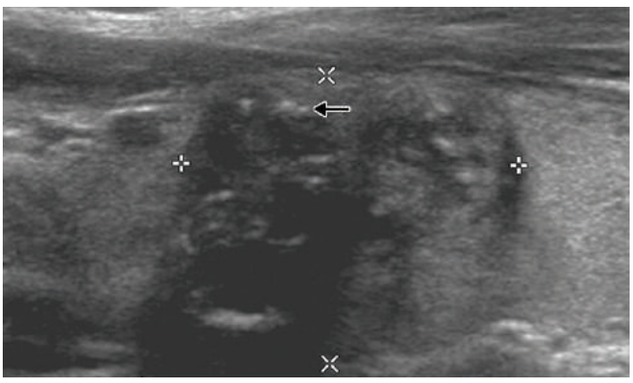

Figure 2. Sagittal scan through the left thyroid lobe shows a papillary thyroid cancer seen as an ill-defined nodule (between callipers) containing microcalcifications (black arrow).

is required along with resection of any lymph node metastases. In the case of extracapsular invasion by thyroid cancer into adjacent structures (e.g. strap muscles of the neck), the involved structures must also be excised.

The risks of total thyroidectomy include bilateral recurrent laryngeal nerve paralysis and permanent hypocalcaemia due to parathyroid gland damage.

Proper lymph node dissection is key for reducing the risk of recurrence. The nomenclature used for lymph node dissection depends on the extent of dissection performed. A radical lymph node dissection involves resection of level I-V lymph nodes, the sternocleidomastoid muscle, the jugular vein and the superficial accessory nerve, while an extended radical dissection includes excision of any further involved structures. If any structures are retained, such as the jugular vein, the procedure is called a modified (or selective) radical dissection.

The term central compartment neck dissection refers to removal of all involved structures and lymph nodes in level V, while a lateral compartment neck dissection includes dissection of involved structures and lymph nodes in levels II-V.

ULTRASOUND TECHNIQUE

During the ultrasound scan, the whole thyroid gland and all potential areas of metastatic disease must be evaluated. All abnormalities must be described in detail based on their morphology and size, and their locations mapped according to the levels described above. Accurate description of the morphology and location of all foci of disease is crucial for guiding completed excision to reduce the risk of recurrence.

There are some features that increase the level of suspicion when assessing thyroid nodules. A purely cystic lesion with a thin wall and no solid component is benign and does not require any follow-up. All solid or partly solid nodules with suspicious features and measuring ≥1cm in diameter require FNA evaluation. Ultrasound must also identify bilateral location of nodules, extracapsular extension (e.g. strap muscles) and retrosternal extension. Retrosternal extension should be further evaluated with computed tomography (CT) to detect extent of direct extension and the presence of mediastinal lymph node metastases.

Suspicious features in a thyroid nodule include a size ≥1cm, solid components, a shape that is taller than wide, irregular or lobulated margins, microcalcifications, interrupted rim calcifications, extra-thyroid extension into adjacent structures and presence both peripheral and central vascularity.

The presence of microcalcifications is highly suggestive of malignant disease having a sensitivity of 89%, a specificity of 95% and an accuracy of 94% (Figure 2). Other features such as size, shape, irregularity of margins, extra-thyroid extension (Figure 3 and 4) and central vascularity are all important when planning FNA procedures. Table 1 shows how suspicious features relate to the risk of malignancy as taken from the American Thyroid

Ultrasound Findings	Size (cm)	Malignancy Risk (%)
Nodule with microcalcifications	≥1	>70-90
Solid nodule		
Hypoechoic with suspicious features	≥1	>70-90
Hypoechoic without suspicious features	≥1	10-20
Isoechoic or hyperechoic	≥1.5	5-10
Mixed cystic and solid nodule		
Suspicious features in solid component	≥1	>70-90
Without suspicious features	≥1.5	5-10
Spongiform without suspicious features	≥2	<3

Table 1. Relation between suspicious features and malignancy risk

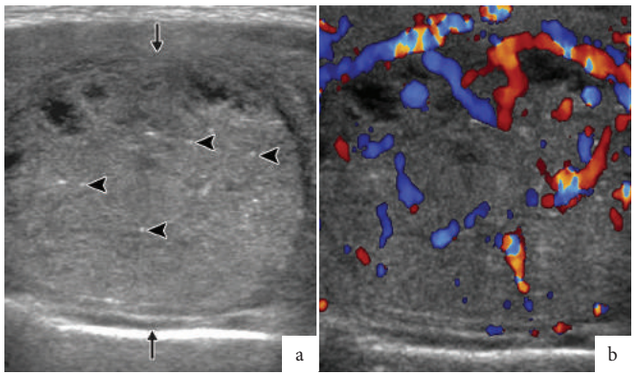

Figure 3. (a) Sagittal image through the left thyroid lobe containing a papillary thyroid cancer with well-defined margins (arrows), microcalcifications (arrowheads) and (b) central + peripheral vascularity.

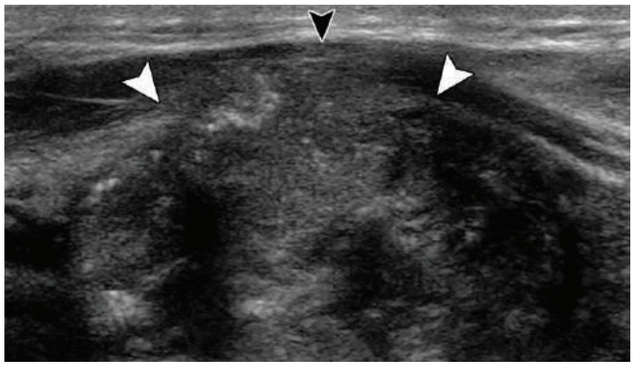

Figure 4. Sagittal image of the right thyroid lobe showing extracapsular extension (between white arrowheads) of cancer into the anterior strap muscle (black arrowhead).

Association consensus report of 2015.[5] Detailed evaluation of lymph nodes at all neck levels is required. Size is not a reliable criterion for excluding metastatic disease; normal cut-off size is <0.8cm in level II and <0.5cm in levels III-VII. Microcalcifications and cystic degeneration within a lymph node are strongly suggestive of metastatic disease with a high degree of accuracy (Figure 5). Increased peripheral vascularity is also indicative of metastatic disease. Additional suspicious features include echogenicity greater than adjacent muscle, a rounded shape (long-to-short-axis ratio <2) and loss of the central fatty hilum.

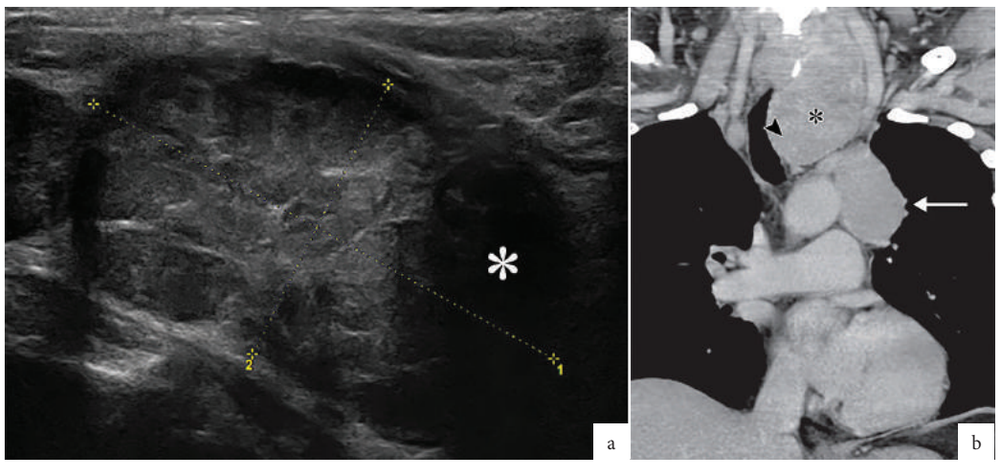

Figure 5. (a) Sagittal image of a large left thyroid nodule (callipers) showing intrathoracic extension (*). (b) CT image shows intrathoracic extension (*) with tracheal compression (arrowhead) and mediastinal lymph node metastases (arrow).

During thyroid surgery, the central compartment is always exposed, while the lateral compartment is only exposed if FNA-proven lymph node metastases are present. Thus, accurate pre-operative mapping of all ultrasound detected sites of disease is required to guide the surgical approach.

POST-OPERATIVE ULTRASOUND EVALUATION

In the immediate post-operative period, numerous nodules may be seen in the thyroid bed that are benign. In fact, 90% of nodules measuring up to 11mm in diameter seen in the immediate post-operative period are benign.[6] Post-operative assessment with ultrasound should not be performed earlier than three months after surgery.

Residual thyroid tissue may be noted on post-operative scans as iso- or hyperechoic nodules. Hypoechoic nodules should raise suspicion for malignant disease. An increasing serum thyroglobulin level (repeated 6-12 months after surgery) is also suggestive of recurrence.

CONCLUSION

Complete surgical excision is the most effective treatment for thyroid cancer. Detailed analysis and mapping of all sites of disease and clear communication with the surgeon are crucial for preventing recurrence.

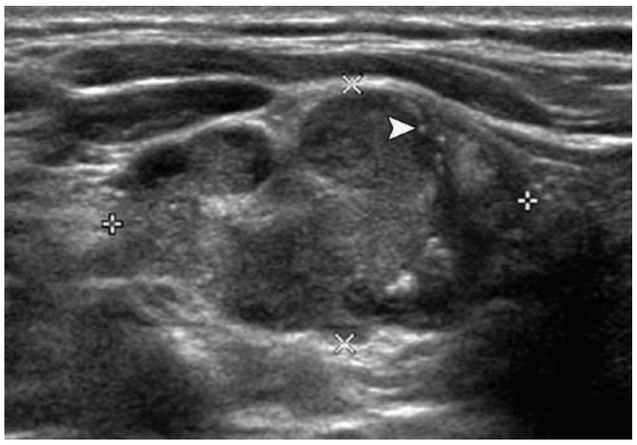

Figure 6. Transverse image through level III showing an enlarged lymph node disease (callipers) with no central fatty hilum and microcalcifications (arrowhead).

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