VOL 23 2024 ISSUE 01 ISSN NUMBER 2313-8084

thesynapse

THE MEDICAL PROFESSIONALS' NETWORK

MR JO-ETIENNE ABELA'S
WHEEL OF FORTUNE

cme30.eu

CONTINUING MEDICAL EDUCATION

Embark on a transformative experience with CME30.eu, where **expert-led video webinars** redefine medical education, allowing you to elevate your practice and **stay at the forefront** of healthcare innovation.

SCAN TO REGISTER

https://cme30.eu

DR IAN ELLUL

Mr Jo-Etienne Abela's Wheel of Fortune

Let us face it. In the past few weeks, the new Minister for Health has inherited a cornucopia of challenges. Challenges which many of us know and some of us know even better.

But let me go one step back and analyse the decision to appoint Dr Jo-Etienne Abela to the post of Minister for Health. You may loathe politics and strongly dissent from the current political administration, but I believe that the majority of people hailing from both sides of the political spectrum will agree that the decision by the Prime Minister to appoint Dr Abela as Minister for Health was a chess master's move. Let me explain better.

Everyone in the field knows that Dr Abela is an excellent surgeon with a distinguished career trajectory. Tick. However, in politics that does not suffice. You also need a likeable, well-meaning, and charismatic leader. Tick. Someone who can build bridges and not burn them. Tick. A person who calls a spade a spade and then, takes the bull by the horns to address any shortcomings. Tick. A visionary. Tick. Add to that the fact that he is Gozitan, and you have the ideal person. Historically, Dr Abela is the first Gozitan to occupy the post of Minister for Health (as well as Active Ageing).

I love to read. One book which has nurtured my forma mentis is *'The Politics of Persuasion'* written by the late Prof. Guido De Marco. I have that book at home personally signed by the author. It narrates an infectious sense of nationalism that inspired prominent figures in our island's history. In my opinion, our current Minister for Health fits that description. However, he will need to draw from that same nationalism the stamina to address the same gargantuan problems which Malta has been facing for the past decade.

Of course, we have an excellent level of care in many departments such as cardiology, vascular, diabetes, primary care, procurement, and so on, which are managed by dedicated and competent healthcare professionals. We take them for granted at times. This should not be the case.

For me, Malta's healthcare challenges are like a wheel of fortune with different-sized wedges. One is spoilt for choice. By fortune, I mean chance/luck of experiencing one of the following … level of care administered in our healthcare settings which may not be standardised, audits which may not be universally conducted/accepted, confusion of policies with procedures, increasing reliance on a foreign workforce as a stopgap measure, increasing waiting lists, silo mentality of specific departments, repetitive union actions, inadequate strategy to vertically increase bed space [stemming from defective concrete utilised for MDH], inadequate parking spaces at MDH and SAMOC, the fact that the renovation of Mount Carmel hospital - built 162 years ago exactly when Abraham Lincoln was made president of the US - has recently been scaled down, and list goes on. Many of those issues, and much more, have been elephants in the room for many years.

Other more pressing matters include the opening of the Regional Centre in Paola, which is overdue. I understand that this will serve as an opportunity for the career progression of younger doctors and other staff when shifting from MDH to the Regional Centre, but then again, we will lose them from MDH. Are we robbing Peter to pay Paul?

Dr Abela has managed to press on various pressure points, even though he has been in office for barely a few weeks. These include the need for a second general hospital, Public-Private Partnerships (PPPs), as well as a strategy for weight-loss surgeries to mitigate the rise in non-communicable diseases. In keeping with this, I sincerely hope that an oncology-dedicated emergency unit is set up, possibly through a PPP, to offer focused care for oncology patients who may be immunocompromised. In my opinion, this unit should be housed adjacent to the A&E at MDH, with a separate waiting room, and would address emergency care for the treatment of adverse effects or those needing procedures like paracentesis; careful triage is recommended with high-risk oncology patients referred to the general emergency department. This idea, I believe, merits a feasibility study in keeping with the fact that according to the WHO's International Agency for Research on Cancer (gco.iarc.who.int/en) the estimated number of new cancer cases in Malta in 2025 will be 3287 i.e. nine new daily cases; nine families crushed each day.

Of note is Dr Abela's track record. The services which have been introduced when he was solely responsible for Active Ageing speak for themselves. Borrowing a quote from the late US coach Vincent Lombardi, "Perfection is not attainable, but if we chase perfection, we can catch excellence." This is what we have experienced in the Ministry for Active Ageing since Dr Abela took its helm. Of two things I am sure.

1. Dr Abela's path is no yellow brick road, but
2. He will nurse our healthcare system back to health.

This, I am convinced, will be his greatest legacy, for all to see and emulate.

Issue Guide

Authors

Dr Adrienne Zerafa Simler MD is a dedicated Anaesthesia and Intensive Care trainee with an interest in advancing her medical and clinical education. Combining clinical proficiency with her interest in medical education, she strives for excellence in both fields.

Dr Anna Maria Fenech Magrin MD, MSc Public Health, MSc Aesthetic Medicine (London) is a specialist in Public Health Medicine and the Medical Director at DoctorAM Clinics. She is a Clinical Senior Lecturer and the Lead of Aesthetics Academic Program at the CCBCR Blizard Institute, Barts Queen Mary University London. Dr Fenech Magrin is a founding member of the Aesthetic Physicians Association of Malta and Director of the Mediterranean Academy of Aesthetic and Regenerative Medicine.

Dr Gabriel Galea MD MSc(Edin.) MRCP FRCR EDiR is a radiologist at Mater Dei Hospital and DaVinci Hospital. He completed his training in general radiology in Malta in 2018 and then completed a two-year fellowship in Cardiothoracic Radiology in the UK.

Dr Kirill Micallef Stafrace MD(Melit.) MSc Sports Med FRCP(Edin.) FFSEM(UK) FFSEM(Ireland) MSK Ultrasound(UEL) is a Consultant in Sports and Exercise Medicine, Deputy Chairman of the Authority for Integrity in Maltese Sports (AIMS) and Board member of the European Federation of Sports Medicine Associations (EFSMA).

Dr May Agius PhD [Man Met] is a speech and language therapist currently working in the field of AAC having played a significant role in the development of AAC services in Malta. She is also a senior visiting lecturer at the University of Malta.

Teodora Aleksic BSc [Hons][Melit.] graduated as a speech and language therapist from the University of Malta. She currently works in the field of AAC and AT in Malta. Her role includes assessment, intervention and providing training to individuals with complex communication needs. She has taken part in numerous research studies which have been presented both locally and abroad.

Editor-in-Chief: Jesmond Friggieri
Managing Editor: Dr Ian C Ellul

Sales & circulation Director:
Sarah Buttigieg
Email: info@cme30.eu
Telephone: +356 2742 2299/ 7960 3358

Publisher:
Leadership Consultancy and Training Services (LCTS Ltd)
Malta Leadership Institute (MLI)
Malta, Europe

Production: Outlook Coop
Printing: Europrint Ltd

OUR COLLABORATORS

MPSA

The magazine is distributed free of charge to all Maltese doctors, pharmacists & dentists, as well as students of the aforementioned professions, with a print run of 3500 copies.

Annual subscription rates outside Malta:
Six issues €100 or equivalent, worldwide

PUBLISHED SINCE OCTOBER 1996

DR ANNA MARIA FENECH MAGRIN

An Overview of Superficial and Medium-Depth Chemical Peels – Part I

ABSTRACT

A chemical peel is the application of a chemical agent to the skin, which causes controlled and predictable destruction of a part or entire epidermis, with or without the dermis. This ultimately results in an improved appearance of the skin, removal of superficial lesions and regeneration of new epidermal and dermal tissue.

INTRODUCTION

Objectives

The primary objective is to discuss the components like pH and composition, that affect tolerability and safety of superficial and medium-depth peels. Another objective is to explain the differences between the different major medium-depth peels and explain how one can recognize the level of injury of superficial vs medium-depth peels.

Over the past decade, facial rejuvenation procedures have become increasingly popular, especially office-based, minimally invasive procedures. This is because they can promote a youthful appearance without the downtime and risk of complications associated with surgical procedures. These procedures include chemical peels, which despite their relative simplicity and reliability, can still carry complications which can happen to the most experienced. Therefore, the goal is to perform the procedures safely and properly with the appropriate preparation and to recognize complications when they occur, to prevent long-term consequences, like scaring and pigmentary changes.

CHEMICAL PEELS

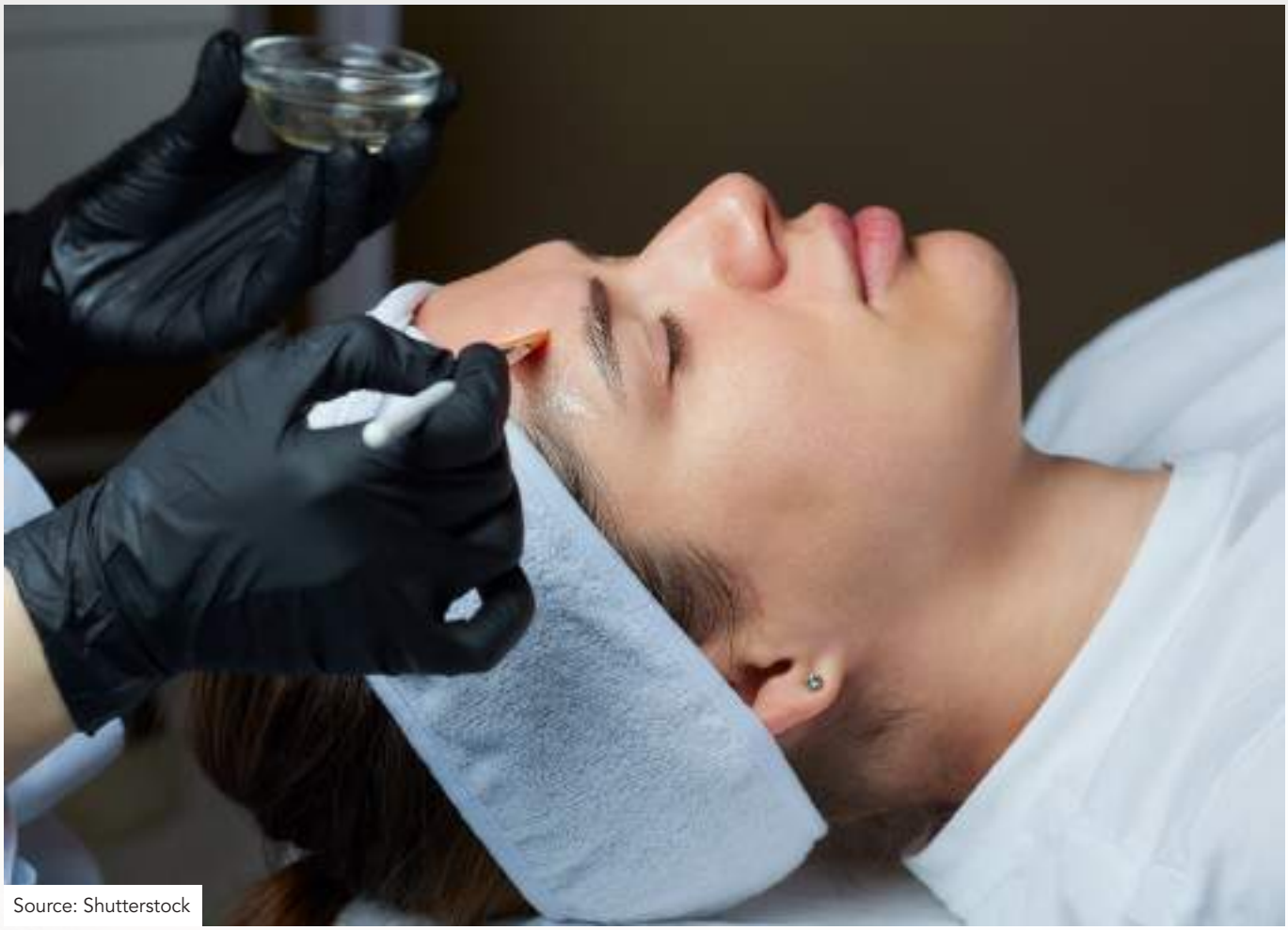
The skin comprises 3 primary layers - the epidermis, the dermis, and the subcutaneous tissue, also referred to as the hypodermis. The epidermis is the top layer of the skin and consists of five layers, i.e. Stratum corneum,

Stratum lucidum, Stratum granulosum, Stratum spinosum, Stratum basale. The epidermis is avascular and contains melanocytes which form the skin pigmentation. The dermis is a connective tissue layer comprising various structures, including hair follicles, nerves, sebaceous glands, and blood vessels. It consists of the papillary layer and reticular layer. The hypodermis is the deepest layer of the skin and is composed of loose connective tissue and adipose tissue.

The effect of a chemical peel depends primarily on the depth of injury of the skin. They are classified according to the depth or wound created by the peel.

Superficial peels penetrate the epidermis only.
Medium-depth peels penetrate down and produce injury into or through the papillary dermis.
Deep peels allow for controlled injury down to the mid-reticular dermis.

A number of factors determine the depth of the peel. These include the type of chemical used, the concentration, the skin type, the mode of application and the number of "passes" on application. One layer of application with certain chemical peels allows for a more superficial peel. If multiple layers are applied, a deeper peeling results. Multiple layers of a superficial peel give different results from a single application of a medium-depth peel. The pKa represents the pH level at which 50% of the chemical is present in a free acid state. When selecting the type of peel, a lower pKa is associated with a more potent peel.



Source: Shutterstock

PATIENT SELECTION

A complete medical history and skin examination should be conducted by the physician before performing a chemical peel. This will help in the choice of the appropriate peeling agent and helps to prevent complications post-treatment.

The following factors should be considered before performing a chemical peel:

- Patient's psychological state.
- Unrealistic expectations.
- Current medications, like minocycline, and oral contraceptives (these may cause photosensitivity).
- Current or previous infections, like herpes simplex virus (HSV) and other bacterial and fungal infections. If prophylaxis is not given, HSV can be reactivated and delayed wound healing occurs.
- Immunosuppression, like HIV. These patients are at a higher risk of infection and altered wound healing with scarring.
- Recent major surgery, like facelift or brow-lift.
- Pregnancy (avoid).

The Fitzpatrick scale is a tool to classify patients based on skin colour and ability to tan.

It can also be used to evaluate the preprocedural risk of post-peel response and complications.

Patients with skin types IV-VI are at a greater risk of developing post-inflammatory hyperpigmentation. Therefore, special attention must be paid when dealing with dark-skinned patients, and deep peels should be avoided in Fitzpatrick skin types III-VI. Medium-depth peels should only be used by very experienced practitioners in skin types III-VI.[1]

Table 1. The Fitzpatrick scale.[2]

Skin type	Skin Colour	Tanning History
I	White	Always burns, never tans
II	White	Usually burns, tan with difficulty
III	White	Sometimes mild burn, tan average
IV	Moderate brown	Rarely burns, tan very easily
V	Dark brown	Very rarely burn, tan very easily
VI	Black	No burn, tan very easily

A. SUPERFICIAL PEELS

The commonly used agents include the following:

1. AHAs (Alpha Hydroxy acids)
   - Glycolic acid (30-70%)
   - Lactic acid (30%)
   - Mandelic acid (40%)

2. BHAs (Beta Hydroxy acids)
   - Salicylic acid (20-30%)

3. AKAs (Alpha Keto acids)
   - Pyruvic acid (50%)

Mechanism of Action

Superficial peels penetrate the epidermis. Dermo-epidermal junction disruption is, however, possible. During the application of superficial peels, controlled keratocoagulation and liquefaction of the cells confined to the epidermis occurs. As superficial peels produce injuries limited to the epidermis, they are indicated to treat conditions confined to the epidermis, like mild acne and epidermal and mixed melasma.[3]

The patient should be advised to avoid any cosmetic treatments with bleaching, depilation, or exfoliation for one week before the treatment. Shaving must also be avoided 24 hours before a peel.

1. AHAs

Glycolic Acid (GA)

This alpha-hydroxy acid is derived from sugar cane. GA is the most common alpha-hydroxy acid peel and has the smallest molecular weight amongst all the alpha-hydroxy acids.[4] It is a highly hydrophilic molecule.

It is used at concentrations of 30 to 50% applied for 1-2 minutes for very superficial exfoliation, at concentrations of 50–70% applied for 2-5 minutes for superficial peels, and 70% GA, applied for 3-15 minutes, is used as a medium-depth peel.[5]

Indication

GA peels are indicated for the treatment of acne, acne scars, melasma, hyperpigmentation, photoaging, and seborrhoea.

Mechanism of Action

GA peels have anti-inflammatory, keratolytic, and antioxidant effects. In low concentrations, GA facilitates the weakening of cohesion of intercellular material of the stratum corneum, causing desquamation.[6]

The depth of the GA peel depends on the concentration of the acid used and also, on the number of coats applied and the time for which it is applied.[7]

GA peels are not self-neutralizing, which means that keratocoagulation continues to occur, as long as it remains on the skin. The acid is neutralised by using water or an alkaline neutralising agent, like 10% sodium bicarbonate, ammonium salts or sodium hydroxide.[8] Neutralization is an exothermic process, therefore it can cause increase in warmth, burning or stinging sensation. When the clinical endpoint is reached, in this case erythema, the acid has to be neutralised. GA peels are frequently combined with other peels and treatments, to give better results.

Application Method

The skin is cleansed and degreased. The degreasing agent, like chlorhexidine, removes surface grease and allows better penetration. The hair is pulled back with a hair band or cap. The patient lies down with the head elevated to 45 degrees and with the eyes closed. The required strength of the peeling agent is poured into a glass beaker. The neutralizing agent is also kept ready. Sensitive areas like the inner canthus of the eyes, and the corners of the nose and lips are protected with petroleum jelly such as Vaseline®. The peeling agent is then applied either with a brush, cotton-tipped applicator or saturated gauze pad. The chemical is applied quickly on the entire face, which is divided into cosmetic units, beginning from the forehead, then the right cheek, nose, left cheek and chin.

[GA] IS USED AT CONCENTRATIONS OF 30 TO 50% APPLIED FOR 1-2 MINUTES FOR VERY SUPERFICIAL EXFOLIATION, AT CONCENTRATIONS OF 50–70% APPLIED FOR 2-5 MINUTES FOR SUPERFICIAL PEELS, AND 70% GA, APPLIED FOR 3-15 MINUTES, IS USED AS A MEDIUM-DEPTH PEEL.

For GA peels, the peel is neutralized after the predetermined duration of time (usually 3-5 minutes). However, if erythema or epidermolysis occurs, seen as a greyish-white appearance of the epidermis or small blisters, the peel must be neutralized immediately irrespective of the duration. Neutralization is done with 10-15% sodium bicarbonate solution or lotion and then washed off with water.

The erythema can rapidly progress to light frosting (level 1), which indicates epidermolysis with separation of the epidermis from the underlying dermis. Care must be taken as this transition to frosting can result in scarring or post- inflammatory hyperpigmentation (PIH).

It is always better to start with a low concentration (20% GA) and increase the concentration and application time during subsequent sessions. Peeling is repeated once every 15 days for 4-6 months until the desired result is achieved.[7]

Side Effects

Good results and minimal side effects should result if patients are selected properly. The timing of the peel and timely neutralization are also very important. Minor side effects include erythema, burning sensation and transient PIH. In rare cases, blistering and scarring can occur.

Lactic Acid and Mandelic Acid

Lactic acid is also an alpha hydroxy acid having activities similar to GA. It has a lower pH than glycolic acid at the same concentrations. This allows for efficient peeling at lower concentrations than glycolic acid, with fewer side effects and faster recovery time. It is indicated to reduce fine wrinkling, uneven pigmentation and to improve the texture of sun-damaged skin.[9]

Mandelic acid, a simple phenolic alpha-hydroxy acid is soluble in both water and polar organic solutions, and therefore results in more uniform penetration through lipid-rich areas of the skin. The results are more subtle than those with GA peels. Side effects and downtime are also less. This peel is effective in the treatment of superficial erythema and dyspigmentation, as well as in the reduction of cutaneous sebum production.[10]

2. BHAs

Salicylic Acid

Salicylic Acid is a beta-hydroxy acid and a phenolic compound. It is poorly soluble in water but highly lipophilic, which combined with its low pKa and small molecular size makes it ideal for rapid penetration through the lipid barriers of the epidermis.

It has anti-inflammatory, antimicrobial and depigmenting properties. It is very effective in the treatment of cutaneous disorders involving excess sebum production.[1]

30% salicylic acid is considered the "gold-standard" superficial peel for the treatment of acne.

Salicylic acid is self-neutralizing or self-limiting. However, care must be taken as overpenetration may still occur due to a cumulative dose effect. Multiple layers or long application time may cause rapid and excessive keratocoagulation beyond the epidermis into the papillary dermis. Overpenetration may thus result in PIH.

3. AKAs

Pyruvic Acid

Pyruvic acid is the simplest alpha-keto acid and is partially lipophilic and partially hydrophilic, giving it properties similar to both salicylic and glycolic acid. Pyruvic acid is not self-neutralizing, and has to be neutralised with an alkaline solution when the endpoint is reached. Although pyruvic acid has demonstrated clinical efficacy for the treatment of disorders associated with excess sebum production, it is not as efficacious as salicylic acid which is more lipophilic.[10] At 50% concentration it is also indicated for mild to moderate acne and fine wrinkles.

[to be continued ...]

REFERENCES

1. Castillo DE, Keri JE. Chemical peels in the treatment of acne: patient selection and perspectives. Clin Cosmet Investig Dermatol 2018;11:365-372.
2. Roberts WE. Skin type classification systems old and new. Dermatol Clin 2009;27(4):529–533.
3. Lee KC, Wambier CG, Soon SL, et al. Basic chemical peeling: superficial and medium-depth peels. Journal of the American Academy of Dermatology 2019;81(2):313-24.
4. Landau M. Chemical peels. Clinics in dermatology 2008;26(2):200-8.
5. Fabbrocini G, De Padova MP, Tosti A. Chemical peels: what's new and what isn't new but still works well. Facial plastic surgery 2009;25(05):329-36.
6. Fartasch M, Teal J, Menon GK. Mode of action of glycolic acid on human stratum corneum: ultrastructural and functional evaluation of the epidermal barrier. Archives of dermatological research 1997;289:404-9.
7. Sharad J. Glycolic acid peel therapy–a current review. Clinical, cosmetic and investigational dermatology 2013;6:281-8.
8. Dayal S, Sahu P, Sangal B, et al. Role of chemical peels in postinflammatory hyperpigmentation: a comprehensive review. Pigment International 2019;6(2):59-66.
9. Sachdeva S. Lactic acid peeling in superficial acne scarring in Indian skin. Journal of Cosmetic Dermatology 2010;9(3):246-8.
10. Soleymani T, Lanoue J, Rahman Z. A practical approach to chemical peels: a review of fundamentals and step-by-step algorithmic protocol for treatment. The Journal of clinical and aesthetic dermatology 2018;11(8):21.

DR GABRIEL GALEA

Lung Cancer Screening: An Overview

DA VINCI HEALTH

INTRODUCTION

Lung cancer is a heterogeneous and often aggressive disease. Despite advances in lung cancer treatment and an overall reduction in the rate of heavy smoking over the last half century, lung cancer remains the most common cancer and the leading cause of cancer death worldwide and in Malta.[1,2]

The theoretical benefits of lung cancer screening are clear; it is a common disease with a significant prognostic benefit when detected early.[3] A cheap, non-invasive and widely available screening tool was the major obstacle to large-scale, successful lung cancer screening. Historical attempts at lung cancer screening using chest radiographs with or without cytological analysis of sputum specimens were largely unsuccessful, mainly due to the limitations of chest radiographs in the detection of early-stage lung cancer. The advent of low-dose chest CT (LDCT) around the turn of the millennium led to the establishment of several large-scale trials determining whether screening with LDCT would reduce mortality from lung cancer among high-risk persons.[4-6]

WHAT IS LOW-DOSE CHEST CT?

Due to an inherently high contrast resolution between air and lung nodules, chest CT can be performed using a low radiation dose while maintaining good diagnostic quality. There is no consensus on what level of radiation is considered 'low dose' and the techniques that affect dose in CT will vary from centre to centre. In general, diagnostic quality CT screening can be accomplished at an overall average effective dose of 2 mSv or less.[7] For comparison, an average lumbar spine x-ray series (lateral and anteroposterior) is approximately 1.5-2 mSv.[8]

RESULTS OF LARGE TRIALS IN LUNG CANCER SCREENING

The National Lung Screening Trial (NLST) conducted in the United States on 53,454 current or former heavy smokers is the largest randomized controlled trial to date. It was launched in 2002 with initial findings reported in 2010 and the most recent follow-up data published in 2019. Participants were randomly assigned to either receive a LDCT scan each year for three years or one chest X-ray each year for three years. LDCT screening showed a higher sensitivity in detecting early-stage lung cancer, a 20% reduction in lung cancer mortality and no increase in radiation-induced lung cancer after 13 years of follow-up.

In Europe, the Dutch-Belgian Randomised Lung Cancer Screening Trial (NELSON) on 15,792 patients is the largest trial performed to date, beginning in 2003 with the final round of follow-up in 2015. Participants aged 50-74 who were current or heavy smokers were randomly assigned to either a screening group that underwent CT screening at baseline, year 1, year 3, and year 5.5 or a control group that received no screening. The results showed that LDCT screening led to a 24% reduction in lung cancer deaths in men and a 33% reduction in lung cancer deaths in women after ten years of follow-up.[5]

Smaller trials including those performed in Italy and the United Kingdom showed similar results with regard to reduction in lung cancer mortality in patients undergoing lung cancer screening.[9,10]

SCREENING GUIDELINES

Screening guidelines vary slightly between countries and will continue to be refined over the coming years as data pools grow and follow-up periods are lengthened. The main parameters to be defined are patient age and smoking history as both are intrinsically linked to lung cancer risk. Any slight variations in inclusion criteria become significant when applied across the numbers of large-scale population screening. Participants at greatest risk for lung cancer mortality are older and usually have more comorbid conditions; this needs to be balanced against the higher screening costs (mainly due to increased detection of incidental findings of questionable significance that nevertheless require further workup) for these patients and their often-reduced life-expectancy due to age and other comorbidities.

The US current screening program recommends annual screening for lung cancer with LDCT in adults aged 50 to 80 years who have a 20 pack-year smoking history and currently smoke or have quit within the past 15 years. A pack year is calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked.

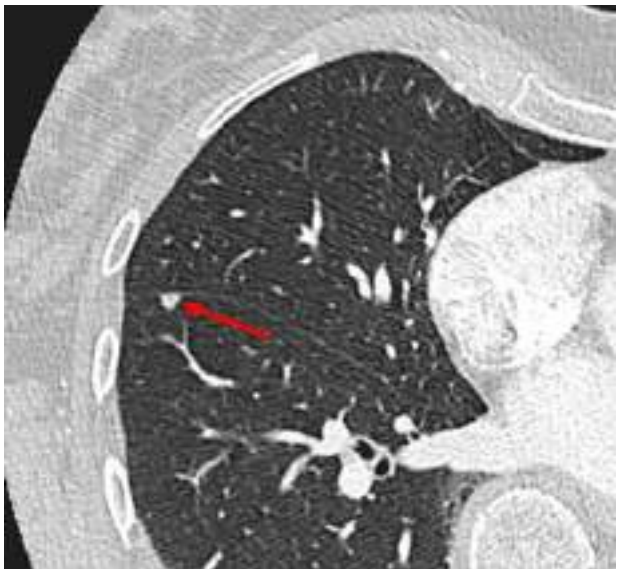
For example, 20 pack years implies one pack of cigarettes per day for 20 years, two packs per day for 10 years, and so on. Screening should be discontinued once a person has not smoked for 15 years, or develops a health problem that substantially limits life expectancy. This is the official recommendation of the U.S. Preventive Services Task Force which is an independent panel of national experts in disease prevention and evidence-based medicine.

Other guidelines are more conservative and less costly. Canadian guidelines now recommend screening using LDCT every year for up to three consecutive years in individuals aged 55 to 74 who have at least a 30 pack-year history of smoking.[11,12] The Netherlands have also begun rolling out a national lung cancer screening program targeting individuals who are between 50 and 75 years old and who have smoked at least 15 cigarettes per day for at least 25 years or at least 10 cigarettes per day for at least 30 years. Participants in this program will receive a LDCT scan every year for three years and then every two years for the subsequent four years if the initial scans are negative.[13]

In 2023, the UK began rolling out a lung 'health check' program which involves identifying and inviting 'ever' smokers aged 55-74 from GP records, then assessing eligibility and frequency interval for LDCT screening using a risk assessment algorithm.[14]

Definitive European Union-wide lung cancer screening recommendations have not been implemented yet, however in 2022 the European Commission recommended that as part of the *Europe's Beating Cancer Plan*, screening of patients aged 50-75 years who are current or ex-smokers who have quit smoking within the previous 15 years should be introduced. More detailed and concrete recommendations are awaited.

Figure 1: Triangular perifissural/subpleural nodule (red arrow) in right lower lobe typical of an intrapulmonary lymph node. Lung-RADS 2.



LUNG RADS CLASSIFICATION

The Lung-RADS (Lung Imaging Reporting and Data System) is a classification proposed to aid with reporting of lung nodules detected on LDCT and standardise follow-up and management decisions. The details of the Lung-RADS classification are beyond the scope of this article however it is broadly as follows. An inexhaustive explanation as well as recommendations in italics are given below. The figures below illustrate a few examples of common lung nodules and their Lung-RADS categories.

Lung-RADS 0 (incomplete)

- Prior CTs not available for comparison, lungs incompletely imaged or incidental infection in the lungs

Lung-RADS 1 (negative, <1% chance of malignancy); *continue annual screening*

- No lung nodules or benign lung nodules including calcified nodules (benign granulomas) or fat-containing nodules (hamartomas)

Lung-RADS 2 (benign appearances or behaviour, <1% chance of malignancy); *continue annual screening*

- Nodules in a subpleural location with oval, lentiform or triangular appearances in keeping with physiological intrapulmonary lymph nodes
- Solid nodules <6 mm at baseline or new nodule <4 mm or ground glass nodules <30 mm at baseline

Lung-RADS 3 (probably benign, 1-2% chance of malignancy); *6-month follow-up*

- Solid nodules 6-8 mm at baseline or new nodules measuring 4-6 mm
- Part solid nodule ≥6 mm total diameter at baseline or ground glass nodule ≥30 mm at baseline
- Thick-walled lung cyst with enlarging cystic component

Lung-RADS 4A (suspicious, 5-15% chance of lung cancer); *3-month follow-up or PET-CT in certain circumstances*

- Nodules measuring ≥8 mm to <15 mm at baseline or nodules <8 mm but showing growth
- New nodules 6 mm to <8 mm

UNITED FOR LONG-LASTING† LDL-C CONTROL[1]

LEQVIO is administered every 6 months* and provides effective LDL-C control, supported by 6+ years of data[1,2]

*Two doses a year after the two initial doses. Single subcutaneous injection at the start of treatment, again at 3 months, and thereafter every 6 months.[1]
†LDL-C reduction was maintained during each 6-month dosing interval after 2 initial doses of inclisiran.

LEQVIO®
inclisiran

▼ LEQVIO™ **Important note: Before prescribing, consult full prescribing information. Presentation:** Solution for injection: Each pre-filled syringe contains inclisiran sodium equivalent to 284 mg inclisiran in 1.5 ml solution. **Indications:** Indicated in adults with primary hypercholesterolaemia (heterozygous familial and nonfamilial) or mixed dyslipidaemia, as an adjunct to diet: • in combination with a statin or statin with other lipidlowering therapies in patients unable to reach LDLC goals with the maximum tolerated dose of a statin, or • alone or in combination with other lipidlowering therapies in patients who are statinintolerant, or for whom a statin is contraindicated. **Dosage and administration:** Recommended dose: 284 mg administered as a single subcutaneous injection: initially, again at 3 months, followed by every 6 months. **Missed dose:** • If a planned dose is missed by less than 3 months, inclisiran should be administered and dosing continued according to the patient's original schedule.• If a planned dose is missed by more than 3 months, a new dosing schedule should be started – inclisiran should be administered initially, again at 3 months, followed by every 6 months. Treatment Transition from PCSK9 Inhibitor: Inclisiran can be administered immediately after the last dose of a monoclonal antibody PCSK9 inhibitor. To maintain LDL-C lowering, it is recommended that inclisiran is administered within 2 weeks after the last dose of a monoclonal antibody PCSK9 inhibitor. **Special populations:** *Renal impairment:* No dose adjustment is necessary for patients with renal impairment (mild, moderate or severe), or end-stage renal disease. *Hepatic impairment:* No dose adjustment is necessary for patients with mild or moderate hepatic impairment. Inclisiran should be used with caution in patients with severe hepatic impairment. *Pediatric patients (below 18 years):* The safety and efficacy of inclisiran have not been established. *Geriatric patients (65 years of age or above):* No dose adjustment is necessary. **Method of administration:** Intended for administration by a healthcare professional. For subcutaneous injection into the abdomen. Leqvio should be inspected visually for particulate matter prior to administration. Each pre-filled syringe is for single use only. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Warnings and precautions:** *Hemodialysis:* Hemodialysis should not be performed for at least 72 hours after inclisiran dosing. **Pregnancy, lactation, fertility** *Pregnancy:* No available human data. Animal reproduction studies have not shown risk of increased fetal abnormalities. *Lactation:* Not known if transferred into human milk. A risk to newborns/infants cannot be excluded. A decision must be made whether to discontinue breastfeeding or to discontinue/abstain from inclisiran therapy, taking into account the benefit of breastfeeding for the child and the benefit of therapy for the woman. *Fertility:* No human data. No effects on animal fertility. **Adverse drug reactions:** *Common (≥1/100 to <1/10):* Adverse events at the injection site (includes injection site reaction, injection site pain, injection site erythema, and injection site rash). **Interactions:** Not a substrate, inhibitor or inducer of CYP450 enzymes or common drug transporters. Not expected to have clinically significant interactions with other medications. Based on the limited data available, clinically meaningful interactions with atorvastatin, rosuvastatin or other statins are not expected. **Packs and prices:** LEQVIO solution for injection 284mg/1.5ml in pre-filled syringe: €2408,73, LEQVIO solution for injection 284mg/1.5ml in pre-filled syringe (with needle guard): €2393,01.

LEQ01/2021

Reporting of suspected adverse reactions: Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions to: Novartis Pharma Services Inc., Methonis Tower, 73 Makarios Avenue, 1070 Nicosia, Tel: +357 22 690 690 (Pharmacovigilance Department), Fax: +357 22 315032 or to Pharmaceutical Services, Ministry of Health, CY-1475, www.moh.gov.cy/phs, Tel: +357 22 608 632/661, Fax: +357 22 608 649, by completing the Yellow Card which is available to the public pharmacies or electronically in the website www.kitrinikarta.gov.cy.

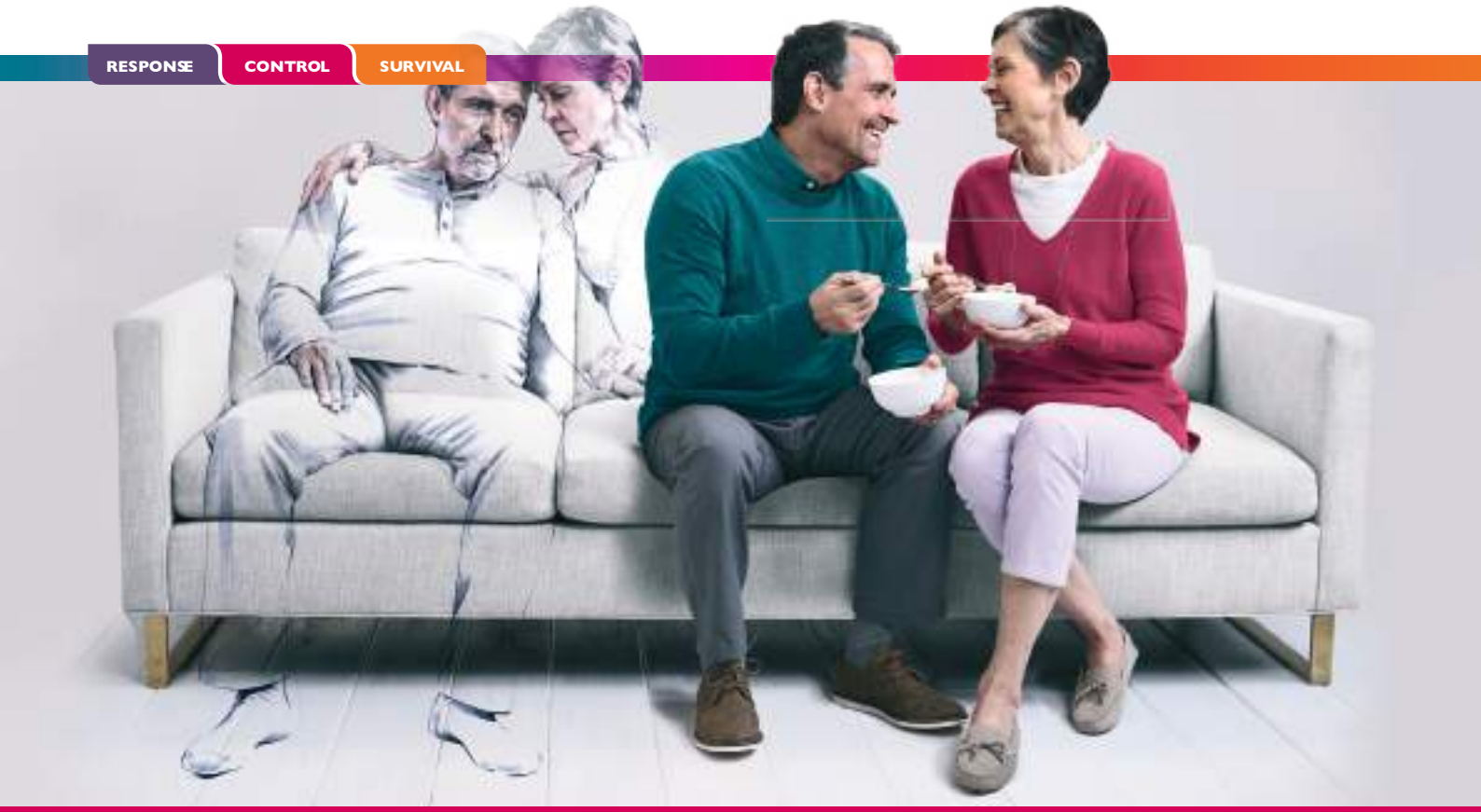
References: 1. Leqvio Summary of product characteristics. Novartis. August 2022. **2.** RS Wright, FJ Raal, W Koenig, U Landmesser, LA Leiter, GG Schwartz, A Lesogor, P Maheux, Z Talloczy, S Vikarunnessa, X Zang, KK Ray. ORION-8: Long-term efficacy and safety of twice-yearly inclisiran in high cardiovascular risk patients. Data presented at the ESC Congress on August 28, 2023.

NOVARTIS
Novartis Pharma Services Inc.
Methonis Tower, 73 Makarios Avenue, 1070 Nicosia, Tel: +357 22 690 690, Fax: +357 22 496 798

LEQAD 02.24 CY

The day you choose **JAKAVI®** (ruxolitinib) **is the day you could change their life**[1-5]

RESPONSE CONTROL SURVIVAL



JAKAVI is indicated for the treatment of adult patients with myelofibrosis (MF), including primary myelofibrosis, post-polycythemia vera myelofibrosis or post-essential thrombocythemia myelofibrosis.[1]

JAKAVI® ruxolitinib

PRESENTATION: Each tablet contains 5mg, 10mg, 15mg or 20mg Ruxolitinib **INDICATIONS:** Jakavi is indicated for the treatment of disease-related splenomegaly or symptoms in adult patients with primary myelofibrosis (also known as chronic idiopathic myelofibrosis), post polycythaemia vera myelofibrosis or post essential thrombocythaemia myelofibrosis. Jakavi is indicated for the treatment of adult patients with polycythaemia vera who are resistant to or intolerant to hydroxyurea. Jakavi is indicated for the treatment of graft host versus host disease or chronic graft versus host disease in patients over the age of 12 who have inadequate response to corticosteroids or other systemic therapies. **DOSAGE:** Jakavi treatment should only be initiated by a physician experienced in the administration of anti cancer medicinal products. A complete blood cell count, including a white blood cell count differential, must be performed before initiating therapy with Jakavi. The recommended starting dose in MF is of 5mg ruxolitinib twice daily for patients with platelet count between 50,000/mm^3 and 75,000/mm^3, 10mg twice daily for patients with platelet count from 75,000/mm^2 to 100,000/mm^2, 15 mg ruxolitinib twice daily for patients with a platelet count between 100,000/mm^3 and 200,000/mm^3 and 20 mg twice daily for patients with a platelet count of >200,000/mm^3. Treatment may be continued as long as the benefit-risk remains positive. However the treatment should be discontinued after 6 months if there has been no reduction in spleen size or improvement in symptoms since initiation of therapy. Jakavi is to be taken orally, with or without food. If a dose is missed, the patient should not take an additional dose, but should take the next usual prescribed dose. The recommended dose for PV is 10mg given orally twice daily. Treatment should also be interrupted when haemoglobin is below 8g/dl. After recovery of blood counts above these levels, dosing may be restarted at 5mg twice daily and gradually increased based on careful monitoring of complete blood count. Dose reduction should also be considered if haemoglobin decreases below 12g/dl and is recommended if it decreases below 10g/dl. If efficacy is considered insufficient and blood counts are adequate, doses may be increased by a maximum of 5mg twice daily, up to a maximum dose of 25 mg twice daily. The recommended dose in GvHD is 10mg given orally twice daily. It can be added to continued corticosteroid use and/or calcineurin inhibitors (CNIs). Dose reductions and temporary interruptions of treatment may be needed in GvHD-patients with thrombocytopenia, neutropenia, or elevated total bilirubin after standard supportive therapy including growth-factors, anti-infective therapies and transfusions. In GvHD, tapering of Jakavi may be considered in patients with a response and after having discontinued corticosteroids. A 50% dose reduction of Jakavi every two months is recommended. **CONTRAINDICATIONS:** Hypersensitivity to the active substance or to any of the excipients. Pregnancy and lactation. **WARNINGS/PRECAUTIONS:** Treatment with Jakavi can cause haematological adverse drug reactions, including thrombocytopenia, anaemia and neutropenia. A complete blood count, including a white blood cell count differential, must be performed before initiating therapy with Jakavi. Patients should be assessed for the risk of developing serious bacterial, mycobacterial, fungal and viral infections. Tuberculosis has been reported in patients receiving Jakavi. Before starting treatment, patients should be evaluated for active and inactive ("latent") tuberculosis, as per local recommendations. Hepatitis B viral load increases have been reported in patients with chronic HBV infections taking Jakavi. Therefore such patients should be treated and monitored according to clinical guidelines. It is recommended to screen for HBV prior to commencing treatment with Jakavi. Physicians should educate patients about early signs and symptoms of herpes zoster, advising that treatment should be sought as early as possible. Progressive multifocal leukoencephalopathy (PML) has been reported with Jakavi treatment for myelofibrosis. Non-melanoma skin cancers have been reported in patinets treated with ruxolitinib. Periodic skin examination is recommended for patients who are at risk for skin cancer. Treatment with Jakavi has been associated with increased lipid parameters and therefrore lipid monitoring and treatment of dyslipidemia according to clinical guidelines is recommended. The starting dose of Jakavi should be reduced in patients with severe renal impairment. For patients with end-stage renal disease on haemodialysis the starting dose should be based on platelet counts. The starting dose of Jakavi should be reduced by approximately 50% in patients with hepatic impairment. Following interruption or discontinuation of Jakavi, symptoms of myelofibrosis may return over a period of approximately one week. There have been cases of patients discontinuing Jakavi who sustained more severe events, particularly in the presence of acute intercurrent illness. Jakavi contains lactose. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicinal product. **INTERACTIONS:** Interaction studies have only been performed in adults. When administering Jakavi with strong CYP3A4 inhibitors the unit dose of Jakavi should be reduced by approximately 50%, to be administered twice daily. Patients should be closely monitored (e.g. twice weekly) for cytopenias and dose titrated based on safety and efficacy. Patients should be closely monitored and the dose titrated based on safety and efficacy. In healthy subjects given ruxolitinib (50 mg single dose) following the potent CYP3A4 inducer rifampicin (600 mg daily dose for 10 days), ruxolitinib AUC was 70% lower than after administration of Jakavi alone. No dose adjustment is recommended when ruxolitinib is co-administered with mild or moderate CYP3A4 inhibitors (e.g. erythromycin). There is no interaction study with oral contraceptives. It cannot be excluded that ruxolitinib inhibits CYP3A4 in the intestine. Ruxolitinib may inhibit P-glycoprotein and breast cancer resistance protein (BCRP) in the intestine. This may result in increased sytemic exposure of substrates of these transporters, such as dabigatran etexilate, ciclosporin, rosuvastatin and potentially digoxin. The concurrent use of haematopoietic growth factors and Jakavi has not been studied. The concomitant use of cytoreductive therapies and Jakavi has not been studied. The safety and efficacy of this co-administration is not known. **ADVERSE REACTIONS:** MF patients – Very common: Urinary tract infections, Herpes zoster, Pneumonia, Anaemia, Thrombocytopenia (any CTCAE grade and CTCAE grade 3), Neutropenia (any CTCAE grade), Bleeding (any bleeding including intracranial bleeding, gastrointestinal bleeding other bleeding), Bruising, Gastrointestinal bleeding, Other bleeding (including epistaxis, post-procedural haemorrhage and haematuria), Hypercholesterolaemia (any CTCAE grade), Hypertriglyceridaemia (any CTACAE grade), Weight gain, Dizziness, Headache, Elevated lipase (any CTCAE grade), Constipation, Raised alanine aminotransferase (any CTCAE grade), Raised aspartate aminotransferase (any CTCAE grade), Hypertension. Common: Sepsis, Thrombocytopenia (CTCAE grade 4), Neutropenia (CTCAE grades 3&4), pancytopenia, Intracranial bleeding, Flatulence, Raised alanine aminotransferase (CTCAE grade 3). PV patients – Very common: Urinary tract infections, Herpes zoster, Anaemia (any CTCAE grade), Thrombocytopenia (any CTCAE grade), , Bleeding (any bleeding including intracranial, and gastrointestinal bleeding, bruising and other bleeding), Bruising, Other bleeding (including epistaxis, post-procedural haemorrhage and haematuria), Hypercholesterolaemia (any CTACAE grade), Hypertriglyceridaemia (any CTCAE grade), Weight gain, Dizziness, Headache, Elevated lipase (any CTCAE grade), Constipation, Raised alanine aminotransferase (any CTCAE grade), Raised aspartate aminotransferase (any CTCAE grade), Hypertension; Common; Pneumonia, Anaemia (CTCAE grade 3), Thrombocytopenia (CTCAE grade 3), Neutropenia (any CTCAE grade), pancytoenia, Gastrointestinal bleeding, Flatulence. For a full list of adverse reaction please refer to the Summary of Product Characteristics. **LEGAL CATEGORY:** POM **PACK SIZE:** 56 Tablets **MARKETING AUTHORISATION HOLDER:** Novartis Europharm Limited, Vista Building, Elm Park, Merrion Road, Dublin 4, Ireland. **MARKETING AUTHORISATION NUMBER:** EU/1/12/773/005, 008, 011, 015.

Please refer to 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 21222872.2022-MT-JAK-29-APRIL-2022. To report adverse events electronically please use the following link: www.novartis.com/report or by e-mail at drug_safety.malta@novartis.com.

References: 1. JAKAVI® (ruxolitinib) Summary of Product Characteristics, April 2022. **2.** Verstovsek S, et al. N Engl J Med. 2012;366:799 807., **3.** Harrison C, et al. N Engl J Med. 2012;366:787–798. **4.** Verstovsek S et al. J Hematol Oncol. 2017; 10(1): 55. **5.** Verstovsek S et al. J Hematol Oncol. 2017; 10(1): 156.

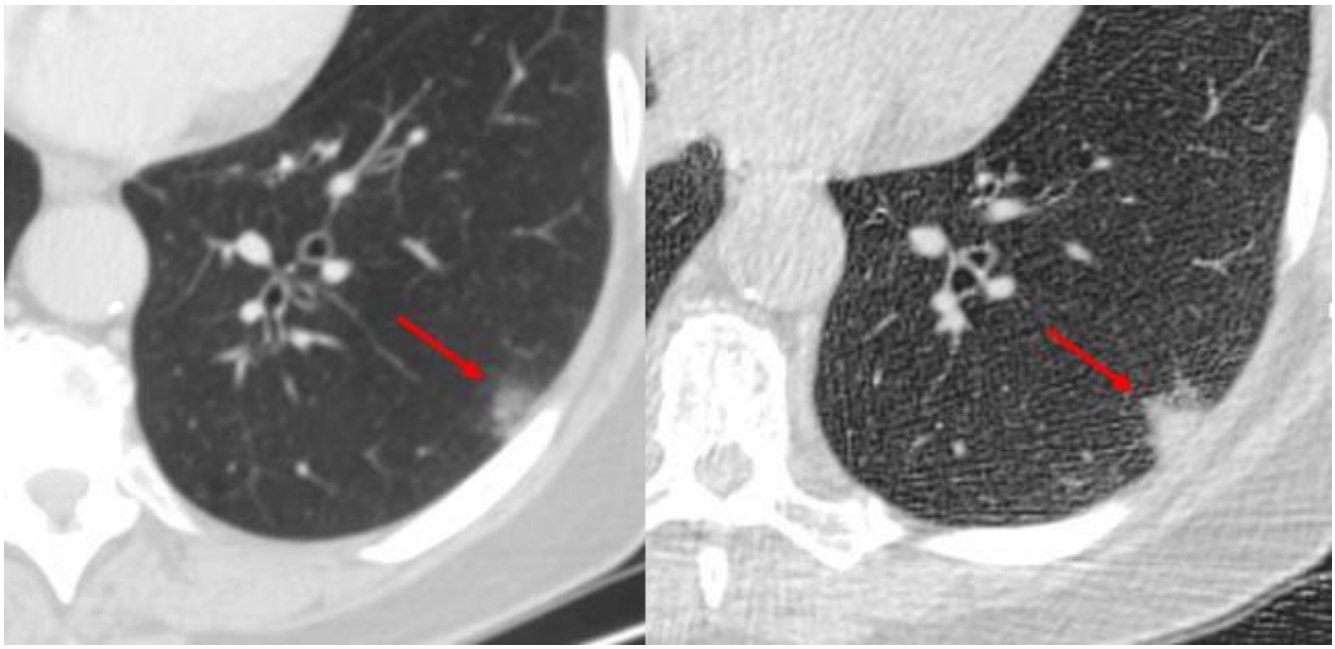
NOVARTIS

Novartis Pharma Services Inc. Malta.
Ewropa Business Centre, Level 4, The Penthouse, Suite 27, Triq Dun Karm Birkirkara BKR9034 +35621222872, novartis.malta@novartis.com

JAK AD 02 24 MT

... DIAGNOSTIC QUALITY CT SCREENING CAN BE ACCOMPLISHED AT AN OVERALL AVERAGE EFFECTIVE DOSE OF 2 MSV OR LESS. FOR COMPARISON, AN AVERAGE LUMBAR SPINE X-RAY SERIES (LATERAL AND ANTEROPOSTERIOR) IS APPROXIMATELY 1.5-2 MSV

Figure 2: Left image shows a part-solid nodule (red arrow) measuring 12 mm in total diameter with a small solid component (solid nodule with ground glass 'halo'). Lung-RADS 3. Follow-up CT 1 year later shows a part-solid nodule with a growing solid component, reclassified as Lung-RADS 4.



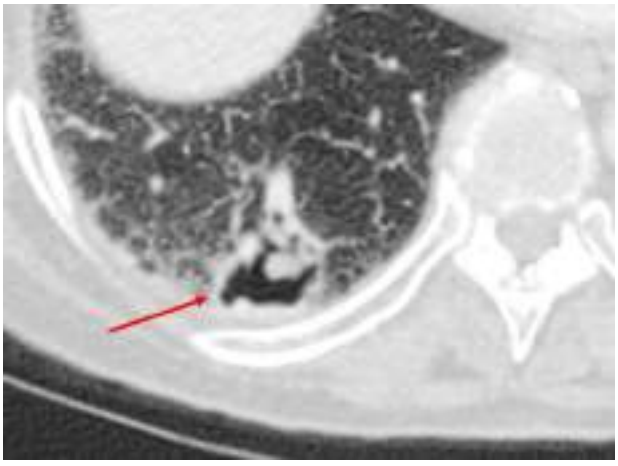
Lung-RADS 4B (very suspicious, >15% chance of malignancy); *Multidisciplinary team management with options including follow-up, chest CT with contrast, FDG/PET-CT and/or tissue diagnosis*

- Solid nodule ≥15 mm at baseline or new/growing nodules ≥8 mm
- Thick-walled cyst with increasing wall thickness/nodularity
- Nodule growing slowly over multiple screening exams

Lung-RADS 4X (very suspicious, >15% chance of malignancy); *Multidisciplinary team management with options including follow-up, chest CT with contrast, FDG/PET-CT and/or tissue diagnosis*

- Category 3 or 4 nodules with additional imaging features that increase the suspicion of malignancy

Figure 3: Thick-walled cyst (red arrow) classified as Lung-RADS 4A which showed increased nodularity on a follow-up CT, upgraded to Lung-RADS 4B.



CHALLENGES OF LUNG CANCER SCREENING

The benefits of lung cancer screening are clear. Detection of lung cancer at an earlier stage leads to a better outcome with historical data reporting a 52% 5-year survival at stage I compared with a 5% 5-year survival at stage IV. Lung cancer screening aims to detect lung cancer at an earlier stage.

The risks and disadvantages of lung cancer screening are not insignificant, however. There is a radiation risk from the yearly exposure needed for lung cancer screening although studies performed so far did not show an increased risk of lung cancer at long-term follow-up. There is also a small but material risk, as well as a cost, from invasive investigations (FDG/PET-CT, bronchoscopy and CT-guided biopsy) performed for false-positive nodules i.e. the flagging of benign nodules as suspicious. [10,15]

One of the major challenges in lung cancer screening and screening programs in general, is effective recruitment and retention of patients. Patient recruitment can be done by public information campaigns or by targeted recruitment through a patient's general practitioner; the latter is a more challenging method in countries where there is no named GP assigned to individual patients. It is important to explain to the participant the importance of screening, the different examination steps and the time each step will take as well as the difference between a screening CT and a standard CT with regard to radiation exposure and diagnostic quality. Lung cancer is unique as a cancer in that smokers may feel stigmatised or even guilty about smoking. There is a perception among smokers that lung cancer is a punishment[16] and it is important to present screening in a positive way to increase acceptance. The Manchester arm of the UK Lung Cancer screening trial chose to name their programme as 'lung health check' which feels more positive in an attempt to increase participation.

Perhaps the biggest challenge facing lung cancer screening around the world is a shortage of health care professionals. In particular, if lung cancer screening were to become widespread, there would be a dramatic shortage of expert thoracic radiologists to read the LDCT scans, and a double reading by experts, as carried out in European studies, does not seem realistic for large-scale screening. The two most viable options to tackle this are to train general radiologists in lung cancer screening or to fully embrace computer-aided diagnosis (CAD) in reading LDCT. CAD has been shown to be at least as good as radiologists at detecting nodules and is already an important part of most screening programs. CAD's sensitivity for nodule detection comes at a cost of increased false-positives. It is hoped that the development of deep-learning-based algorithms with particular emphasis on correct Lung-RADS category categorisation of nodules will lead to higher automation with results on par with radiologists.

CONCLUSION

LDCT lung cancer screening will almost certainly see widespread implementation in Europe in the medium- to long-term, with current evidence suggesting a clear benefit to patients. The medical community must overcome some important challenges before this widespread implementation. It is essential that the screening parameters and patient inclusion criteria are refined. Patient recruitment will be a particular challenge, unique to each country's healthcare system. A solid plan for training and enlargement of the current workforce is one of the most important and challenging hurdles to surmount, particularly for radiologists. Artificial intelligence will undoubtedly play an important role although the exact role remains unclear and will need to be prospectively validated. Finally, quality assurance needs to be implemented and a European or worldwide registry for collection of lung cancer CT screening data should ideally be developed, with regular assessment of the continued utility of lung cancer screening.

REFERENCES

1. OECD/European Observatory on Health Systems and Policies. Malta: Country Health Profile 2021, State of Health in the EU. Brussels; 2021.
2. Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin 2021;71(3):209–49.
3. De Angelis R, Sant M, Coleman MP, et al. Cancer survival in Europe 1999–2007 by country and age: results of EUROCARE-5—a population-based study. Lancet Oncol 2014;15(1):23–34.
4. The National Lung Screening Trial Research Team. Lung Cancer Incidence and Mortality with Extended Follow-up in the National Lung Screening Trial. Journal of Thoracic Oncology 2019;14(10):1732–42.
5. de Koning HJ, van der Aalst CM, de Jong PA, et al. Reduced Lung-Cancer Mortality with Volume CT Screening in a Randomized Trial. New England Journal of Medicine 2020;382(6):503–13.
6. Field JK, Vulkan D, Davies MPA, et al. Lung cancer mortality reduction by LDCT screening: UKLS randomised trial results and international meta-analysis. The Lancet Regional Health - Europe 2021;10:100179.
7. Larke FJ, Kruger RL, Cagnon CH, et al. Estimated Radiation Dose Associated With Low-Dose Chest CT of Average-Size Participants in the National Lung Screening Trial. American Journal of Roentgenology 2011;197(5):1165–9.
8. Mellor FE, Thomas P, Breen A. Moving back: The radiation dose received from lumbar spine quantitative fluoroscopy compared to lumbar spine radiographs with suggestions for dose reduction. Radiography 2014;20(3):251–7.
9. Pastorino U, Sverzellati N, Sestini S, et al. Ten-year results of the Multicentric Italian Lung Detection trial demonstrate the safety and efficacy of biennial lung cancer screening. Eur J Cancer 2019;118:142–8.
10. Field JK, Vulkan D, Davies MPA, et al. Lung cancer mortality reduction by LDCT screening: UKLS randomised trial results and international meta-analysis. The Lancet Regional Health - Europe 2021;10:100179.
11. Krist AH, Davidson KW, Mangione CM, et al. Screening for Lung Cancer. JAMA 2021;325(10):962.
12. Canadian Task Force on Preventive Health Care. Recommendations on screening for lung cancer. Can Med Assoc J 2016;188(6):425–32.
13. Amicizia D, Piazza MF, Marchini F, et al. Systematic Review of Lung Cancer Screening: Advancements and Strategies for Implementation. Healthcare 2023;11(14):2085.
14. UK National Screening Committee. Targeted screening for lung cancer in adults with a history of smoking [Internet]. 2022 [cited 2024 Jan 2]. Available from: https://view-health-screening-recommendations.service.gov.uk/document/585/download
15. Jonas DE, Reuland DS, Reddy SM, et al. Screening for Lung Cancer With Low-Dose Computed Tomography. JAMA 2021;325(10):971.
16. Mazières J, Pujol JL, Kalampalikis N, et al. Perception of Lung Cancer among the General Population and Comparison with Other Cancers. Journal of Thoracic Oncology 2015;10(3):420–5.

NOW
AVAILABLE

DulcoEase® Hydrate

*suitable for children from 6 months & pregnant women

ORAL SOLUTION

Gentle & effective relief of constipation and irregularity

250ml

GENTLE RELIEF

WORKS WITHIN 24 to 72hrs

GLUTEN LACTOSE & SUGAR FREE

Available in pharmacies across Malta & Gozo.

SAN-DUL-MT-0224-3

DR MARIE ADRIENNE ZERAFA SIMLER, DR KIRILL MICALLEF STAFRACE

Osgood-Schlatter Disease in Adulthood: A Case Report

ABSTRACT

An athletic 29-year-old gentleman presented with severe pain over the proximal aspect of the anterior tibia. The onset of symptoms was during heavy aerobic exercise involving running and jumping, following a long period of inactivity. The patient had no further comorbidities, did not smoke or drink alcohol. The pain had lasted several months with no relief despite conservative treatment with rest, physiotherapy, and oral analgesia. Symptoms were relieved on starting external shockwave therapy. This case report describes the etiopathology, symptomatology, diagnosis, and treatment of Osgood-Schlatter disease, a rare condition in adulthood.

KEYWORDS

Osgood-Schlatter, Orthopaedics, Sports medicine, Diagnosis, Management

INTRODUCTION

An athletic 29-year-old gentleman presented to the Orthopaedic Outpatients at Mater Dei Hospital complaining of severe pain over the proximal aspect of the left anterior tibia, just below and lateral to the patella. The onset occurred when the patient began heavy physical aerobic exercise involving running and jumping, following a long period of inactivity. The pain had been ongoing for 11 months with no relief despite conservative treatment with rest, physiotherapy and oral analgesia. Results for routine haematology and biochemistry were unremarkable. Medical history was not significant for any comorbidities, and this was his first presentation with such a complaint. He was completely fit and independent and did not smoke or drink alcohol.

CASE PRESENTATION

Several months following failed attempts at conservative management, this gentleman was referred to the Sports Clinic at the Orthopaedic Outpatients. On examination, his left knee was swollen with a minimal effusion and an area of erythema and deformity marking the proximal anterior aspect of the tibia over the tibial tuberosity. The knee was otherwise stable for its anatomical components. The patient had decreased range of motion secondary to severe pain on flexion of the knee to more than ninety degrees. The pain was specific to the area of erythema and deformity over the left tibial tuberosity.

Figure 1. X-Rays revealing extensive calcification and soft tissue swelling over the insertion of the patellar tendon into the tibial tuberosity.

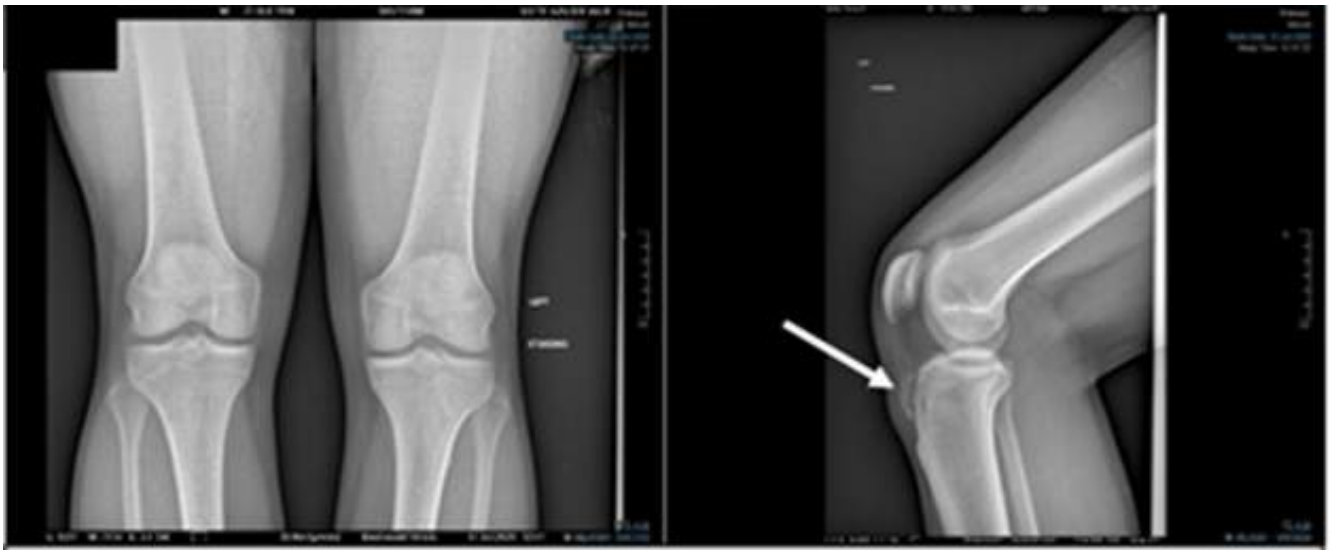
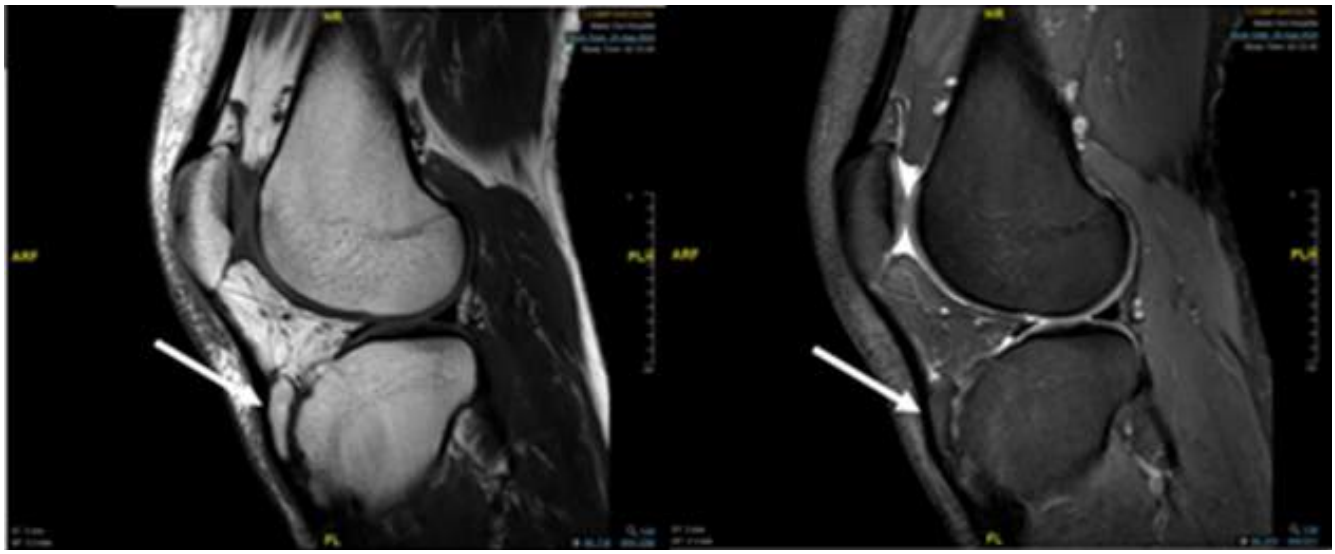


Figure 2. MRI demonstrating infrapatellar bursitis and severe thickening and calcification of the distal patellar tendon at its insertion into the tibial tuberosity.



Laboratory investigations revealed that relevant blood analysis was within normal parameters for pathologies such as infection, gout and inflammatory musculoskeletal and rheumatological disease. Plain radiographs of both knees revealed extensive calcification and soft tissue swelling over the insertion of the patellar tendon into the tibial tuberosity (Figure 1). An MRI was more sensitive and specific to these radiological findings and demonstrated further soft tissue swelling and bone marrow oedema. Infrapatellar bursitis and severe thickening and calcification of the distal patellar tendon at its insertion into the tibial tuberosity was also noted (Figure 2).

Finally, an US once again indicated ossified cartilage with surrounding oedema of the surrounding soft tissues and thickening of the patellar tendon. Doppler investigation of the ossified component of the patellar tendon showed neovascularization around the significant ossification at the insertion of the patellar tendon into the tibial tuberosity.

DISCUSSION

Osgood-Schlatter disease was first described in the early 1900s when the two physicians Dr Robert Osgood, a US orthopaedic surgeon, and Dr Carl Schlatter, a Swiss emergency surgeon, reported an increased incidence of adolescents complaining of pain over the tibial tuberosity in their lower limbs on increased physical exercise. This pathological process, also known as tibial osteochondrosis, has in fact become one of the most common traction apophysitis and overuse injury in the knee of adolescent athletes.[1]

Although well described, the origin of this condition and its pathological process remains very controversial. Its pathophysiology involves loss of continuity of the patellar tendon-cartilage-bone junction of the developing tibial tuberosity through an inflammatory process secondary to chronic tendinitis and calcification. The most accepted theory for this process is repetitive knee extension mechanism contraction causing microavulsions at the insertion of the patellar tendon into the tibial tuberosity.[2,3] Anatomical variants also play a role as they may predispose to increased tension over the patellar tendon. The most significant variant noted was the position of the tibial tuberosity as this dictated the tension and extension forces over the patellar tendon during quadriceps contraction.[4]

Osgood-Schlatter disease has been explored in great detail in developing adolescents and children between eight and fifteen years of age. It is more common in males and is known to resolve once closure of the epiphyseal growth plates has occurred. Nonetheless, although rare, it is known to occasionally persist into adulthood in active individuals as in the case demonstrated in this report.[5] In fact, 10% of children and adolescents who develop Osgood-Schlatter disease continue to experience symptoms into adulthood. This is a rare but recognised condition in young adults which is an ongoing orthopaedics issue faced by physicians and surgeons alike.[5]

The treatment offered to both adults and adolescents is initially conservative. It involves restriction of aerobic activities such as running which causes excess force over the patellar tendon and instead increase exercises

CORTICOSTEROID INJECTION INTO THE PATELLAR TENDON IS NOT RECOMMENDED AS A TREATMENT FOR OSGOOD-SCHLATTER DISEASE. THIS IS MOSTLY SECONDARY TO ITS HIGH INCIDENCE OF SUBCUTANEOUS ATROPHY AND RUPTURE OF THE PATELLAR TENDON

such as stretching, swimming, and cycling. These activities increase hamstring and quadriceps strength and flexibility and are known to accelerate recovery.[6,7] Protective pads, ice and elevation are also part of the usually prescribed conservative management. Such a regime is frequently accompanied by a short prescription of nonsteroidal anti-inflammatory drugs for bouts of increased pain.[6,7] Such management has reported good response with only refractory cases requiring further intervention.[7] Further intervention such as external shockwave therapy for the duration of over a month have proved to be successful when conservative management alone fails. External shockwave therapy is effective in reducing pain, enhancing patient-reported functional recovery, and improving performance-based functional outcomes in adults with Osgood-Schlatter disease.[8]

Corticosteroid injection into the patellar tendon is not recommended as a treatment for Osgood-Schlatter disease. This is mostly secondary to its high incidence of subcutaneous atrophy and rupture of the patellar tendon. Other injections such as hyperosmolar dextrose and autologous conditioned plasma are also available, however their efficacy alone is limited and are used as adjuncts to the conservative management options mentioned above.[9,10]

When all conservative management options fail, operative treatment is considered. Multiple procedures have been documented for the treatment of this disease, especially in adults. These include drilling of the tubercle, removal of loose fragments, autogenous bone peg insertion through the tubercle, tibial tuberosity excision and sequestration.[11]

Less invasive modalities such as arthroscopic surgery and bursoscopic excision have also shown promising results. Arthroscopic surgery was noted to be less invasive and spared the patellar tendon from incision and surgical trauma. This allegedly saves the patient from pain on kneeling unlike more invasive interventions.[12] Bursoscopic excision is even less invasive as such a procedure does not violate the infrapatellar fat pad and avoids meniscal and ligamentous iatrogenic injury. However, this approach does have a limited working space which inhibits adequate reduction of the described ossifications and abnormalities.[13,14]

CONCLUSION

In conclusion, this report notes that Osgood-Schlatter disease is a common pathology in children and adolescents. It resolves as the adolescent develops into adulthood but may persist and cause ongoing unwanted symptoms as Osgood-Schlatter disease of the adult. As a result, adults who do not respond to conservative management may need to opt for surgical intervention for cure of this disease.

REFERENCES

1. Demirag B, Ozturk C, Yazici Z, et al. The pathophysiology of Osgood-Schlatter disease: a magnetic resonance investigation. J Pediatr Orthop B 2004;13(6):379-82.
2. Gholve PA, Scher DM, Khakharia S, et al. Osgood Schlatter syndrome. Curr Opin Pediatr 2007;19(1):44-50.
3. Maffulli N, Grewal R. Avulsion of the tibial tuberosity: muscles too strong for a growth plate. Clinical Journal of Sport Medicine: Official Journal of the Canadian Academy of Sport Medicine 1997;7(2):129-32.
4. Kaufer H. Mechanical function of the patella. JBJS-AM 1971;53(8):1551-1560.
5. Høgh J, Lund B. The sequelae of Osgood-Schlatter's disease in adults. International Orthopaedics 1988;12(3):213-215.
6. Circi E, Atalay Y, Beyzadeoglu T. Treatment of Osgood-Schlatter disease: review of the literature. Musculoskelet Surg 2017;101(3):195-200.
7. Bloom OJ, Mackler L, Barbee J. Clinical inquiries. What is the best treatment for Osgood-Schlatter disease? J Fam Pract 2004;53(2):153-6.
8. Liao CD, Xie GM, Tsauo JY, et al. Efficacy of extracorporeal shock wave therapy for knee tendinopathies and other soft tissue disorders: a meta-analysis of randomized controlled trials. BMC Musculoskelet Disord 2018;19(1):278.
9. Danneberg DJ. Successful Treatment of Osgood-Schlatter Disease with Autologous-Conditioned Plasma in Two Patients. Joints 2017;5(3):191-194.
10. Nakase J, Numata H, Oshima T, et al. A Novel Injective Approach For Osgood-schlatter Disease: A Prospective Randomized Double-blind Study. Orthop J Sports Med 2016;4(7 suppl4):2325967116S00159.
11. Rostron PKM, Calver RF. Subcutaneous atrophy following methylprednisolone injection in Osgood-Schlatter Epiphysitis JBJS 1979;61(4):627-628.
12. Beyzadeoglu T, Inan M, Bekler H, et al. Arthroscopic excision of an ununited ossicle due to Osgood-Schlatter disease. Arthroscopy: the Journal of Arthroscopic & Related Surgery: Official Publication of the Arthroscopy Association of North America and the International Arthroscopy Association 2008;24(9):1081-1083.
13. DeBerardino TM, Branstetter JG, Owens BD. Arthroscopic treatment of unresolved Osgood-Schlatter lesions. Arthroscopy 2007;23(10):1127.e1-3.
14. Eun SS, Lee SA, Kumar R, et al. Direct bursoscopic ossicle resection in young and active patients with unresolved Osgood-Schlatter disease. Arthroscopy 2015;31(3):416-21.

DR MAY AGIUS, TEODORA ALEKSIC

Empowering Communication:

The Crucial Role of Speech and Language Therapists in Augmentative and Alternative Communication Practices

Key words: Augmentative and Alternative Communication, Speech and Language Therapy, Complex Communication Needs, Service Delivery, Case Studies.

ABSTRACT

Traditionally, the profession of Speech and Language Therapy has been associated with the assessment and intervention of children and adults with speech, language, communication, and swallowing difficulties. A lesser-known area of speech and language therapy relates to the use of augmentative and alternative communication. In this article, we explore the role of Speech and Language Therapy in the field of augmentative and alternative communication as well as provide the reader with basic information on how this can support communication. Four case studies are presented to demonstrate how augmentative and alternative communication can support individuals to achieve communicative competence.

INTRODUCTION

Speech and language therapy (SLT) is an area of practice that aims to provide support and treatment for children and adults who have speech, language, and communication needs (SLCN), as well as providing services to individuals who have difficulties with eating, drinking, and swallowing.

One area of practice requiring specific SLT expertise is augmentative and alternative communication (AAC). Speech and language therapists (SLTs) working in this field are found in community settings. In many countries, however, SLTs also work within specialist teams that specialize in the area of AAC and assistive technology which may include other allied health professionals such as occupational therapists. In this article, we explore the role of the SLT in the field of AAC as a means of supporting individuals with severe speech, language and communication difficulties to participate in society. The first part of the article deals with the basic concepts of AAC, who can benefit from it, and the role of the SLT within this field of practice. In the second part of this article, we will provide four case vignettes to demonstrate how AAC can be used to successfully support children and adults to communicate and be more independent.

The term AAC refers to systems and/or strategies that can be used to augment an individual's communication skills, either temporarily or in the longer term.[1] AAC also includes the interventions required for the individual to attain communicative competence. The recommendation for AAC systems and/or strategies and interventions is consistent with Article 19 of the Universal Declaration of Human Rights which declares the right to communicate for all individuals including those with communication disabilities.[2] This is further supported by Article 21 of the Convention on the Rights of Persons with Disabilities.[3] SLTs working in this area are therefore uniquely placed to support individuals with communication disabilities to realize their rights and their full potential.

AAC includes unaided and aided systems and strategies. Unaided strategies make use of the body and include manual signing, the use of gestures, and body language to support communication. Aided systems are classified into low-tech and high-tech systems. Low-tech systems are often paper-based and not battery-powered. Examples of low-tech AAC systems include communication books as well as the use of pen and paper to write down messages to support communication expression. High-tech AAC systems include devices designed specifically to support communication, referred to as dedicated communication aids. In the last 15 years, mainstream technology, including tablets with special AAC software or applications, has also been utilized as communication aids. High-tech AAC systems can provide voice output which may be synthesized or digitized as well as provide access to robust language expression.

AAC is recommended for a small percentage of individuals with communication disability who are referred to as having complex communication needs (CCN). The term CCN refers to individuals who have difficulty communicating in everyday situations using speech. They may have difficulty producing or understanding speech.

At present, no local data is available on the number of people who could benefit from AAC in Malta. It has, however, been suggested that this need could account for 0.5% of the population in the UK and that 97.5% of these have nine specific medical conditions as follows: dementia, Parkinson's disease, autism, intellectual disability, stroke, cerebral palsy, head injury, multiple sclerosis, and motor neuron disease.[4] Furthermore, it is estimated that 97 million people worldwide may benefit from AAC.[5]

The goal of AAC is to provide individuals with effective ways to express themselves, participate in social interactions, engage in meaningful communication with others, and improve their overall quality of life. The selection of AAC depends on the individual's communication abilities, motor skills, cognitive abilities, and personal preferences. Often, a combination of both aided and unaided AAC methods may be used to provide the most effective and versatile communication support for an individual. The goal is to tailor the AAC system to meet the specific needs and goals of the user.

The feature-matching process in AAC assessment involves systematically matching the features of AAC systems to the specific needs and abilities of an individual. SLTs play a crucial role in the process of feature-matching considering the following:

- Assessment of communication skills - the SLT conducts a comprehensive assessment of the individual's current communication abilities including their receptive and expressive skills, speech intelligibility, and cognitive skills.
- Identifying expectations of individual and family - the SLT explores what expectations the individual and family have for the use of the AAC system and/or strategy, and which contexts it will be used in, for example, in school, work, and/or leisure. The level of support the individual has is also taken into consideration.
- Determining access method - the individual's physical and motor capabilities are taken into account to determine the most suitable access method for the communication system or strategy. This can be direct access through the use of hands, body parts, or eye pointing, or indirect access such as using a switch to control the AAC system. Switches can be activated using multiple parts of the body. Access assessments are usually carried out in collaboration with occupational therapists to determine the most consistent and efficient access method.

SARAH, 45 YEARS OLD, WAS DIAGNOSED WITH AMYOTROPHIC LATERAL SCLEROSIS AND FACED THE GRADUAL LOSS OF HER ABILITY TO SPEAK ... USING VOICE BANKING WITH AN APPROPRIATE AAC SYSTEM, SARAH WAS ABLE TO MAINTAIN MEANINGFUL CONNECTIONS WITH LOVED ONES, EXPRESSING HERSELF AUTONOMOUSLY THROUGH A VOICE OUTPUT COMMUNICATION AID

- Determining vocabulary representation and size - Vocabulary can be picture-based, symbol-based, phrase-based, or text-based. The selection is tailored to the individual's comprehension level and cognitive skills. Vocabulary size consists of determining the range of words and topics that are made available to the individual, depending on the contexts the individual will need to communicate in.

Based on assessment findings, the SLT's role is to make recommendations for the appropriate AAC systems and/or strategies when appropriate. If high-tech AAC is recommended this would include the appropriate hardware and software solution for the individual. Part of the SLT's role is to set up the AAC system, particularly if it is aided, as well as to support the appropriate selection of vocabulary. Training to the individual, family, members, caregivers, and relevant professionals on how to use and support the AAC systems and/or strategy is also provided. Goal setting is an important component of the AAC process to ensure the successful implementation of the AAC system. Throughout the feature-matching process, the SLT collaborates closely with the individual, their support network, and other professionals to ensure that the chosen AAC system and/or strategy aligns with the user's abilities, preferences, and communication goals.

The following is a set of four case studies demonstrating how AAC is used in practice. The names of the clients have been changed.

CASE STUDY 1. SUPPORTING PARTICIPATION BEYOND COMMUNICATION.

Fourteen-year-old Emily, diagnosed with cerebral palsy, faced communication challenges due to limited speech abilities. A SLT recommended and implemented a dedicated communication aid with a touch screen and customized software which Emily could access using a switch. The switch was activated using her right hand. The vocabulary was represented on the software using symbols and a keyboard with word-prediction was also available for her to spell words. Through comprehensive intervention, Emily and her family learned to use the communication aid, empowering her to express her thoughts, participate in class, and build social connections. Ongoing support from the therapist ensured the AAC system adapted to Emily's evolving needs; for example, by using the communication aid, Emily was given access to social media such as Facebook, Messenger and Instagram. She learned how to use YouTube and the Internet to search for information. This case demonstrates the transformative impact of AAC in enhancing communication for children with cerebral palsy.

BASED ON ASSESSMENT FINDINGS, THE SLT'S ROLE IS TO MAKE RECOMMENDATIONS FOR THE APPROPRIATE AAC SYSTEMS AND/OR STRATEGIES WHEN APPROPRIATE

CASE STUDY 2.
DEVELOPING COMMUNICATIVE FUNCTIONS.

Alan, an autistic 3-year-old child, communicated mainly by taking adults to things that he wanted. When he did not want to do something, he screamed and threw himself on the floor. Occasionally he uttered some words, but these were not used functionally. The regular SLT introduced picture exchange communication in which Alan was taught how to exchange a picture symbol of a motivating object/activity with an adult to request what he wanted. The SLT also focused on teaching him to point to request desired objects and to wave to greet adults. As Alan began to progress, the therapist referred him to a specialist AAC team to consider high-tech AAC. The team recommended a dedicated communication aid for Alan and specific intervention strategies to support him in developing communication beyond requesting and greeting. At ten years of age, Alan was using the communication aid to make phrases to request his needs, express opinions, greet others, and express negation. He also began to use speech more frequently in the appropriate contexts, demonstrating how AAC can also support the development of speech, language and communication.

CASE STUDY 3.
PRESERVING IDENTITY THROUGH VOICE BANKING.

Sarah, 45 years old, was diagnosed with amyotrophic lateral sclerosis (ALS) and faced the gradual loss of her ability to speak. Collaborating with her SLT, she recorded a variety of phrases and expressions using voice banking technology. This preserved her natural voice as her ALS progressed. Following the AAC assessment, the banked voice was matched to compatible hardware and software to allow Sarah to use her banked messages. Using voice banking with an appropriate AAC system, Sarah was able to maintain meaningful connections with loved ones, expressing herself autonomously through a voice output communication aid. As Sarah's ALS progressed and her physical abilities diminished, the mode of access to the device was changed from access using touch on the screen to eye-gaze technology. Sarah was able to use her AAC system to communicate but also to surf the internet, use social media apps, read the newspapers, and watch videos/movies.

CASE STUDY 4:
AUGMENTING SPEECH RECOVERY POST-STROKE.

Mark, aged 60 years, experienced aphasia following a stroke, impairing his communication abilities including speech clarity. His SLT introduced AAC strategies to supplement his communication recovery, starting with low-tech tools including communication boards to aid in comprehension and expression. Mark gradually transitioned to a voice output communication aid to help him in situations in which he was not understood. With consistent therapy and AAC support, Mark regained confidence in expressing his thoughts and needs, enhancing his quality of life post-stroke.

CONCLUSION

The SLT plays a seminal role in the assessment and intervention of individuals with speech, language and communication needs. SLTs who work in the field of AAC, play a crucial role in ensuring that individuals meet their rights to communicate, to be independent and to increase their overall quality of life. SLTs are therefore essential guides in navigating the evolving landscape of AAC systems and/or strategies in a fast-changing technological world. As advocates for client-centered care, SLTs are capable of bridging the gap between medical diagnosis and practical solutions. This collaboration between healthcare professionals and SLTs can support adopting a more client-centered approach. If you, as family doctors, think you know someone who could benefit from AAC, please reach out to a Speech and Language Therapist.

ADDITIONAL READING RESOURCES

- Communication Matters UK: www.communicationmatters.org.uk/
- American Speech Hearing Association: www.asha.org/practice-portal/professional-issues/augmentative-and-alternative-communication/#collapse_1

REFERENCES

1. Light J, McNaughton D. Communicative competence for individuals who require augmentative and alternative communication: A new definition for a new era of communication?. Augmentative and Alternative Communication 2014;30(1):1-8.
2. United Nations General Assembly. The Universal Declaration of Human Rights (UDHR). New York: United Nations General Assembly, 1948. Available from: https://www.un.org/en/about-us/universal-declaration-of-human-rights
3. United Nations. Article 21 - Freedom of expression and opinion, and access to information. [web page]. Available at: https://www.un.org/development/desa/disabilities/convention-on-the-rights-of-persons-with-disabilities/article-21-freedom-of-expression-and-opinion-and-access-to-information.html
4. Creer S, Enderby P, Judge S, et al. Prevalence of people who could benefit from augmentative and alternative communication (AAC) in the UK: determining the need. International Journal of Language & Communication Disorders 2016;51(6):639-53.
5. Light J, McNaughton D, Caron J. New and emerging AAC technology supports for children with complex communication needs and their communication partners: State of the science and future research directions. Augmentative and Alternative Communication 2019;35(1):26-41.

DA VINCI HEALTH

Your health is our business... Make it your business, use our services!

State-of-the-art private hospital

KAN KARMENU PIROTTA ROAD,
BIRKIRKARA BKR 1111, MALTA.
(+356) 21 491 200
INFO@DAVINCIHEALTH.COM
WWW.DAVINCIHEALTH.COM
DAVINCIHEALTHMALTA

DaVinci Health is a medical center, which focuses on the promotion of health in its patients and not simply the treatment of injury or illness. We believe in maintaining the overall health of our patients by combining the experience and expertise of all our specialists to offer a holistic service and prevent medical conditions from developing. DaVinci Health offers a range of services to support our clients in every aspect of their physical and mental wellbeing.

SPECIAL OFFER:

Get a full medical checkup for **ONLY €35.**

Offer valid until end of April

- Full physical examination
- Height, Weight and Body Mass Index
- Blood pressure test
- Resting ECG
- Report of the findings

Redeem coupon at reception desk. Call now on **21 491200** for an appointment.