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PHYSICIAN CONNECT

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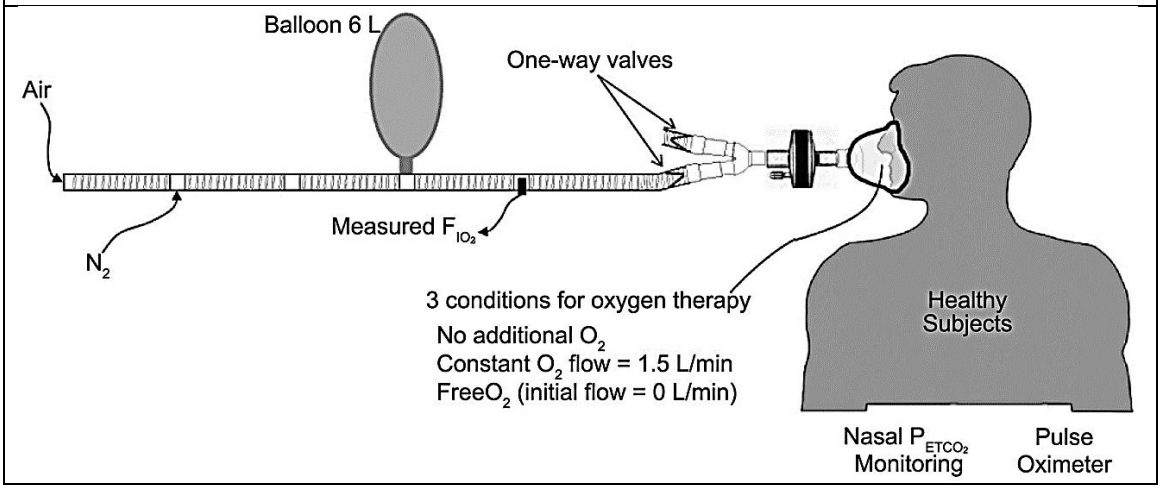
Automated Oxygen Administration Reduces Dyspnea in Patients with Acute COPD Exacerbation

Introduction:

Acute exacerbations of Chronic Obstructive Pulmonary Disease (COPD) are a major cause of hospitalisation and are typically characterised with dyspnoea, anxiety, and hypoxemia [(1)]. The primary reason that COPD patients get admitted to hospitals is because their dyspnoea is getting worse. Dyspnoea is the primary symptom of COPD. Hospitalised COPD patients frequently experience hypoxemia, thus these patients get supplementary oxygen therapy. Automated oxygen administration (AOA), a closed-loop device that titrates oxygen based on the patient's peripheral oxygen saturation SpO2 level, has been designed to prevent hypo- and hyper-oxygenation as a result of poor oxygen therapy [(2)]. Devices with Automatic Oxygen Administration (AOA) in reaction to the patient's SpO2 have been created to optimise oxygen administration and hence therapeutic impact. We aimed to see how AOA compared to standard oxygen treatment affected multiple features of dyspnea and as-needed use of opioids and benzodiazepines in hospitalised patients with Acute Exacerbation of COPD (AECOPD) [(1)].

Automated Oxygen Flow Titration to Maintain Constant Oxygenation



Methods:

A multicenter randomised controlled experiment was conducted in five respiratory wards in Denmark's Capital Region. Patients with AECOPD (n=157) were randomly assigned to either AOA (O2matic Ltd), a closed loop device that delivers oxygen based on the patient's peripheral oxygen saturation (SpO2), or traditional nurse-administered oxygen treatment. The O2matic device was used to evaluate oxygen flows and SpO2 levels in both groups, while Patient Reported Outcomes was used to assess dyspnoea, anxiety, depression, and COPD symptoms.

Results:

The intervention was completed for 127 of the 157 randomised patients. The mean duration of the intervention was 40 hours in both groups and the median length of stay across the two groups was 6.0 days, whereas the median time from randomization to discharge was 5.7 days. The fraction of time with normoxemia (SpO2 in target, either 88–92% or 90–94%), hyperoxemia (SpO2>target), hypoxemia (SpO2 between 85 and target), and severe hypoxemia (SpO2) respectively, are presented in (Figure 1), for the intervention group and the control group. The AOA considerably decreased patients' impression of overall unpleasantness on the Multidimensional Dyspnoea Profile (MDP), with a difference in medians of -3 (p=0.003) between the intervention (n=64) and control (n=63) groups. Additionally, the AOA revealed a significant between group difference for the Visual Analogue Scale - Dyspnoea (VAS-D) within the last three days (p=0.013) and all single items within the sensory domain of the MDP (all p-values 0.05). In both the MDP and VAS-D, all between group differences were more than the Minimal Clinical Important Difference. The use of as-needed opioids and/or benzodiazepines, the COPD Assessment Test, the Hospital Anxiety and Depression Scale, or the emotional response domain of the MDP did not appear to be affected by AOA (all p-values >0.05).

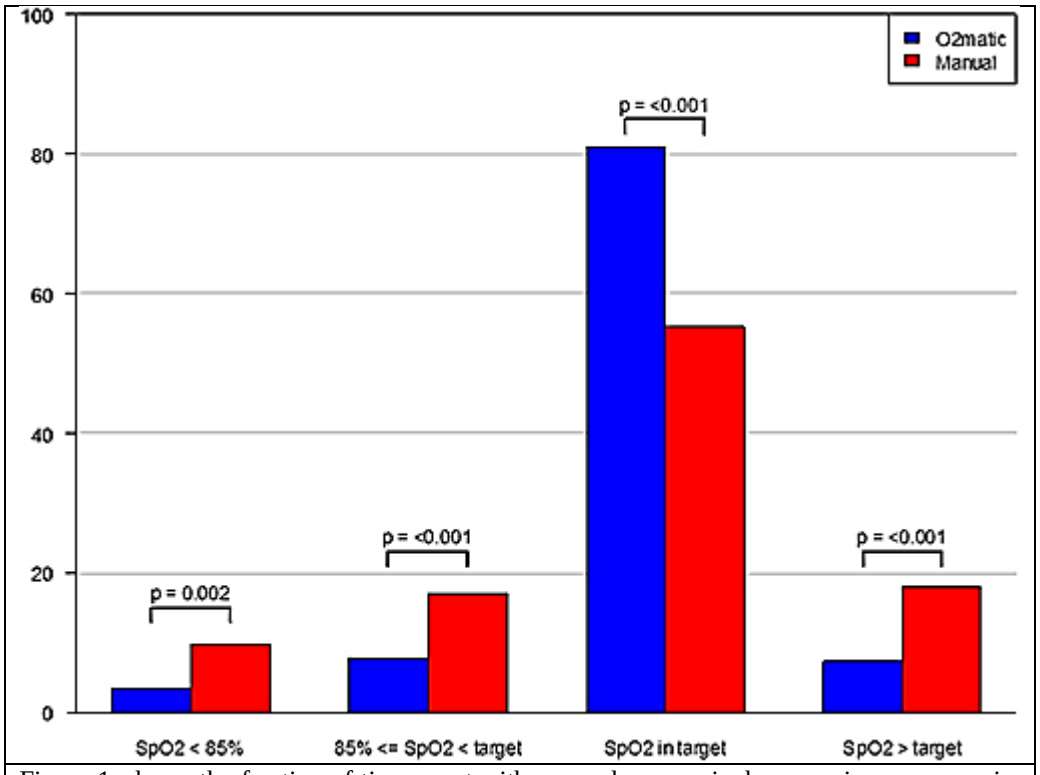


Figure 1. shows the fraction of time spent with severe hypoxemia, hypoxemia, normoxemia, and hyperoxemia for the intervention group (O2matic, blue color) and the control group (manual oxygen administration, red color) respectively, with *p*-values for the difference between the groups.

Discussion:

This study demonstrates that, when compared to traditional oxygen treatment, AOA lessens the overall severity of dyspnoea in patients hospitalised with AECOPD and hypoxemia. When

compared to traditional oxygen treatment, the AOA dramatically lessens all five sensory aspects of dyspnoea as well as dyspnoea from the previous three days [1].

According to current study, "Air hunger" best defines how individuals admitted to the hospital with AECOPD feel about their breathing at that time. This result is in line with a research by Stevens JP et al., who used MDP to examine 156 hospitalised patients with a variety of diagnoses for dyspnoea and discovered that "air hunger" was the most prominent and severe aspect of severe dyspnoea [3].

The impact of AOA on dyspnoea as evaluated by the A1 scale is clinically significant, with a within-group decrease in the intervention group that is more than eight times the MCID (The Minimal Clinical Important Difference) and a between-group difference that is more than three times the reported MCID. Similarly, a study by Stevens et al found an intensity of dyspnoea on the A1 scale to be acceptable to patients if <4 on the 0-10 rating scale, highlighting the clinical importance of AOA in current study, as dyspnoea on the A1 scale was decreased to a median of 3 in the intervention group and 4 in the control group [4].

Conclusion:

In patients hospitalised with AECOPD, AOA reduces physical sensations of dyspnoea as well as breathing pain, but it did not appear to have an effect on the patients' mental states or other COPD symptoms.

The major reason that COPD patients are admitted to hospitals is that their dyspnea is worsening. The predominant symptom of COPD is dyspnea.

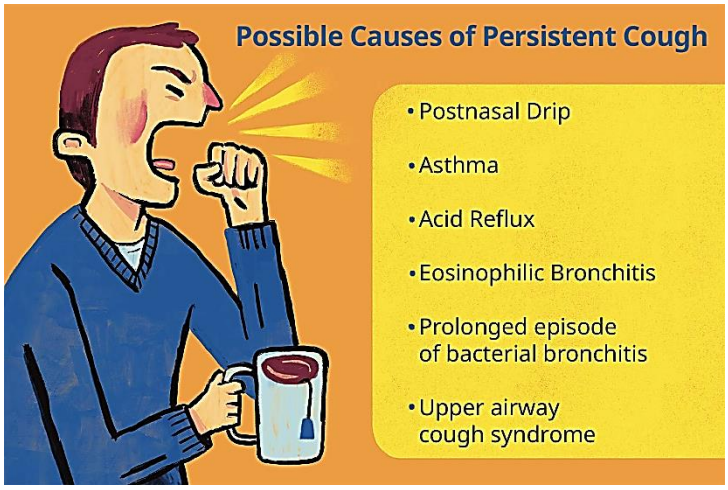
Reference:

1. Sandau C, Hansen EF, Ringbæk TJ, Kallemose T, Bove DG, Poulsen I, Nørholm V, Pedersen L, Jensen JU, Ulrik CS. Automated Oxygen Administration Alleviates Dyspnea in Patients Admitted with Acute Exacerbation of COPD: A Randomized Controlled Trial. International journal of chronic obstructive pulmonary disease. 2023 Dec 31:599-614.
2. Sandau C, Poulsen I, Nørholm V, Hansen EF, Ringbaek TJ, Suppli Ulrik C, Bove DG. Patients' Perspective on Automated Oxygen Administration during Hospitalization for Acute Exacerbation of Chronic Obstructive Pulmonary Disease: A Qualitative Study Nested in a Randomized Controlled Trial. COPD: Journal of Chronic Obstructive Pulmonary Disease. 2022 Dec 31;19(1):345-52.
3. Stevens JP, Sheridan AR, Bernstein HB, Baker K, Lansing RW, Schwartzstein RM, Banzett RB. A multidimensional profile of dyspnea in hospitalized patients. Chest. 2019 Sep 1;156(3):507-17.
4. Stevens JP, Sheridan AR, Bernstein HB, Baker K, Lansing RW, Schwartzstein RM, Banzett RB. A multidimensional profile of dyspnea in hospitalized patients. Chest. 2019 Sep 1;156(3):507-17.

Cough desensitisation therapy for individuals with persistent chronic cough

Introduction:

The term "chronic cough" (CC), which is used to describe a cough that lasts longer than eight weeks, is becoming more and more common. Significant psychological stress and quality of life difficulties are common among CC patients. 10% to 20% of CC patients are thought to have refractory chronic cough (RCC), which is defined as not responding to medical therapy despite comprehensive testing and pharmaceutical treatments [(1)]. RCC has a major financial impact on both society and people and causes serious physical and mental damage. Patients with cough hypersensitivity syndrome(CHS) who had not previously responded to Behavioural cough suppression therapy(BCST) were given successive doses of inhaled aerosolized capsaicin through a nebulizer and instruction on how to control cough in CDT Pilot 1[(2)]. The aim of this research was to gather preliminary effectiveness data on cough desensitisation therapy (CDT), a new treatment for refractory chronic cough (RCC).



Methods:

In this parallel cohort, sham-controlled, randomised controlled trial, 21 persons with RCC were allocated at random to either 12 sessions of CDT or a sham therapy. The main performance indicator was the Leicester Cough Questionnaire (LCQ). Secondary outcome measures were cough challenge testing (to gauge cough-reflex sensitivity) and perceived cough intensity using a visual analogue scale. Mixed effects linear regression and follow-up contrasts were used to analyse the data.

Results:

The research included twenty-two people. Twelve people were assigned to CDT. The CDT and sham groups had mean ages of 64 and 50, respectively. Results across all tests favoured CDT. In the CDT and sham groups, the mean change in LCQ at 3-weeks after treatment was 6.35 and 2.17, respectively, with one sham participant excluded because his baseline LCQ values were considered unreliable. The total LCQ score (p=.058), as well as the LCQ Psychological domain (p=.026) and Physical domain (p=.045) scores, all showed moderate to strong evidence of a higher improvement in the CDT group. There was significant evidence for a higher

decrease in the desire to cough during cough challenge testing(CCT) in the CDT group (p=.037) and only little evidence for a decrease in the sensitivity of the cough response to capsaicin (p=.094). Weak evidence suggested that the CDT group saw a higher reduction in cough intensity (p=.103). Mixed effects linear regression found just a slight indication of a difference between groups over time (F (3, 54) = 2.16, p = 0.103). Follow-up contrasts indicated that the CDT group improved at PT1 and PT2 (p.001 for both), whereas there was no change in the sham group at PT1 or PT2 (p = 1.00 for both). (Table 2) contains VAS results.

Figure: Change in primary and secondary outcome measures in post-treatment phase relative to baseline.

	Mean Change at PT1				Mean Change at PT2						
	CDT		Sham		CDT		Sham				
	Mean	95% CI	Mean	95% CI	mean	95% CI	Mean	95% CI	N^2_p	F-Value	P=Value
LCQ_{Total}	5.32	3.04 to 7.60	2.34	-.33 to 5.02	6.35	4.07 to 8.63	2.17	-.50 to 4.85	.14	2.657	.058
LCQ_{Phys}	1.46	.74 to 2.18	.60	-.24 to 1.42	1.64	.92 to 2.36	.47	-.36 to 1.30	.17	2.49	.097
LCQ_{Psych}	1.95	.92 to 2.99	.80	-.42 to 2.01	2.43	1.39 to 3.46	.50	-.72 to 1.71	.19	3.36	.046
LCQ_{Soc}	1.91	1.00 to 2.81	.96	-.09 to 2.00	2.30	1.39 to 3.20	.96	-.09 to 2.00	.14	1.84	.174
VAS	-32.3	-52.1 to -12.4	-5.11	-27.0 to 16.8	-33.4	-53.2 to -13.5	-4.2	-26.1 to 17.1	.11	2.16	.103
logC5	1.12	.53 to 1.78	.41	-.41 to .97	–	–	–	–	.13	2.54	.094
UTC	-5.00	-7.47 to -2.53	-1.61	-4.34 to 1.11	–	–	–	–	.15	5.08	.037

PT1 = 1-week post-treatment; PT2 = 3 weeks post-treatment; CI = confidence interval; LCQ = Leicester Cough Questionnaire; LCQPhys = LCQ Physical Domain; LCQPsych = LCQ Psychological Domain; LCQSoc = LCQ Social Domain; VAS = visual analogue scale of cough severity; logC5 = log form of dose that causes 5 or more coughs during cough challenge testing; N 2 p = partial eta squared effect size; UTC = urge-to-cough.

Discussion:

A larger improvement was shown with more CDT sessions 6.35 vs. 3.20 change in the overall LCQ. The sham group improved more in pilot 2 (2.73 vs. 1.75), as well. In a parallel study by Bowen AJ et al and Smith JA et al, in pharmaceutical studies, the change in total LCQ with a sham or placebo treatment has not been greater than 1.2 (range: 0.8 to 1.2) [3,4], and Chamberlain Mitchell SA et al found 1.66 in the one BCST study that included a placebo and the LCQ [5].

Conclusion:

A phase 2 clinical trial is strongly supported by the fact that CDT improved cough-related quality of life more than both medical and behavioural therapies, as reported in the literature.

A phase 2 clinical trial is significantly supported by the finding that CDT improved cough-related quality of life more than both medicinal and behavioural interventions.

Reference:

1. Slovarp L, Reynolds JE, Bozarth-Dailey E, Popp S, Campbell S, Morkrid P. Cough desensitization treatment: A randomized, sham-controlled pilot trial for patients with refractory chronic cough. Respiratory Medicine. 2022 Mar 1;193:106739.
2. Slovarp LJ, Reynolds JE, Tolbert S, Campbell S, Welby S, Morkrid P. Cough Desensitization Treatment for Patients with Refractory Chronic Cough: Results of a Second Pilot Randomized Control Trial.
3. Bowen AJ, Nowacki AS, Contrera K, Trask D, Kaltenbach J, Milstein CF, Adessa M, Benninger MS, Taliercio R, Bryson PC. Short-and long-term effects of neuromodulators for unexplained chronic cough. Otolaryngology–Head and Neck Surgery. 2018 Sep;159(3):508-15.
4. Smith JA, Kitt MM, Butera P, Smith SA, Li Y, Xu ZJ, Holt K, Sen S, Sher MR, Ford AP. Gefapixant in two randomised dose-escalation studies in chronic cough. European Respiratory Journal. 2020 Mar 1;55(3).
5. Mitchell SA, Garrod R, Clark L, Douiri A, Parker SM, Ellis J, Fowler SJ, Ludlow S, Hull JH, Chung KF, Lee KK. Physiotherapy, and speech and language therapy intervention for patients with refractory chronic cough: a multicentre randomised control trial. Thorax. 2017 Feb 1;72(2):129-36.

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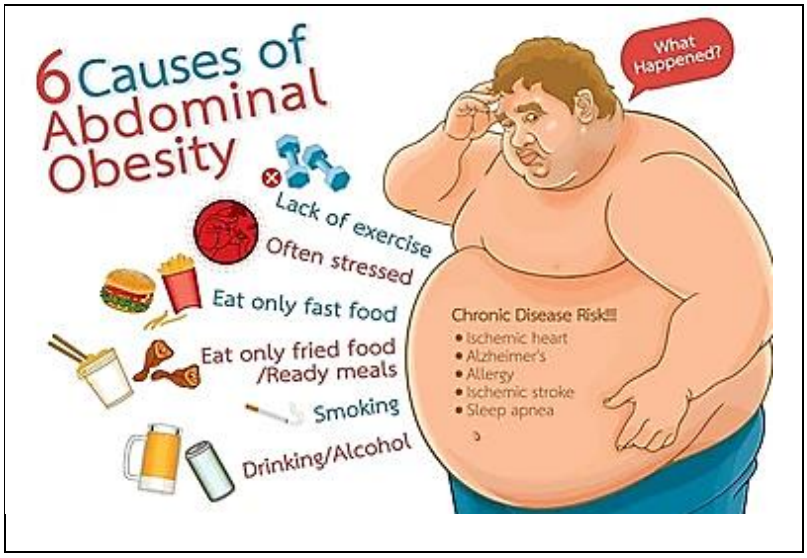
Patients with Diabetes Mellitus Type 2 and Abdominal Obesity Are More Prone to Osteodysfunction

Introduction:

Obesity, osteoporosis, and type 2 diabetes mellitus (T2DM) were all on the rise globally. Due to their widespread occurrence and serious metabolic consequences, they have been classified as public health problems that are associated with an increase in mortality [(1)]. A number of genetic and/or environmental variables can cause diabetes mellitus (DM), a chronic health disorder with several contributing elements. According to the World Health Organization's (WHO) Global Report on Diabetes, there are currently 422 million individuals worldwide who have diabetes, with that figure anticipated to rise to 693 million by 2045. High blood sugar levels are a characteristic of the condition as a result of insulin, the pancreatic hormone responsible for controlling glycemia, being insufficient in concentration or activity [(2)]. One of the main public health issues of nowadays is obesity, which has become much more prevalent over the previous two decades both locally and worldwide. A large number of persons have a significant risk of developing metabolic syndrome, which favours the onset of type 2 diabetes mellitus (T2DM) and atherosclerotic vascular diseases (AVD), mostly as a result of abdominal obesity [(3)]. Obesity has been identified as a risk factor for diabetes complications, particularly abdominal obesity.Public interest in the relationship between diabetes, obesity, and bone metabolism was growing. However, the entire extent of the osteometabolic modifications in diabetes mellitus type 2 (T2DM) individuals with abdominal obesity is yet unknown. The purpose of this study is to examine the relationship between bone turnover indicators and abdominal obesity indices in T2DM subjects [(1)].



Methods:

The METAL research includes 4351 participants. The data was collected by skilled personnel. A well-designed questionnaire was used to collect information on demographic parameters, primary complaints, past personal and family medical history, and risk factors in everyday life. The neck, waist, and hip circumferences, the visceral adiposity index (VAI), the lipid accumulation product (LAP), the waist-to-hip ratio (WHR), and the Chinese visceral adiposity index (CVAI) are all indicators of abdominal obesity. They were used to explain the relationship between intact N-terminal propeptide of type I collagen (P1NP), osteocalcin (OC), and -C-terminal telopeptide (-CTX).

Results:

The analysis included 2004 males and 2347 females with diabetes in total. Males had an average age of 67.64 ±8:73 years while females had an average age of 67.15± 8:62 years. (Table 2) shows the relationships between abdominal obesity indices and BTMs in T2DM groups. Age, TC, TG, HDL, LDL, FPG, HbA1c, glucagon, C-peptide, insulin, Vit D, current smoking, and hypertension were all factored into the model. The indicators of abdominal obesity were highly inversely related to OC and -CTX. In men, five indices (BMI, WC, LAP, WHR, and CVAI) and OC (BMI, NC, WC, WHR, and CVAI) had negative correlations with -CTX and OC. With P1NP, there were no significant connections. All eight indicators had a poor correlation with -CTX in females. Seven indices (BMI, NC, WC, HC, LAP, WHR, and CVAI) were inversely associated to OC. The VAI and P1NP have a negative correlation.

Table 1:Multiple linear regression analysis of abdominal obesity index and BTMs (male)

	Index					
BTMs	β-CTX	*P*	OC	*P*	P1NP	*P*
	B (95% CI)		*B* (95% CI)		*B* (95% CI)	
BMI	-.008 (-.014, -.002)	0.006	-.009 (-.014, -.004)	<0.001	0.001 (-.004, 0.006)	0.643
NC	-.004 (-.010, 0.003)	0.258	-.007 (-.012, -.002)	0.005	0.003 (-.002, 0.009)	0.252
WC	-.004 (-.006, -.002)	<0.001	-.005 (-.007, -.003)	<0.001	0.001 (-.001, 0.003)	0.265
HC	-.001 (-.004, 0.002)	0.522	-.002 (-.004, 0.000)	0.111	0.001 (-.001, 0.004)	0.396
VAI	-.004 (-0.09, 0.002)	0.172	-.001 (-.005, 0.003)	0.686	-.002 (-.006, 0.003)	0.492
LAP	0.000 (-0.001, 0.000)	0.025	0.000 (0.000, 0.000)	0.188	0.000 (0.000, 0.000)	0.723
WHR	-1.055 (-1.430, -.681)	<0.001	-1.137 (-1.442, -.832)	<0.001	0.126 (-.204, 0.457)	0.454
CVAI	-.001 (-.001, 0.000)	0.001	-.001 (-.001, -.001)	<0.001	0.000 (0.000, 0.001)	0.183

Discussion:

The current study came to the conclusion that abdominal obesity had a clear negative connection with bone metabolism in diabetes populations through epidemiological analysis of a large sample of individuals. Both skeletal development and degradation were linked to the abdominal obesity indices.

Waist circumference (WC), the first and most stable assessment tool, has been widely utilised in estimating and even diagnosing clinical disorders such as metabolic syndrome. Several investigations have shown a link between it and metabolic problems, although the link with bone metabolism has been rarely described. A study that enrolled 64 participants with abdominal obesity discovered a negative relationship between insulin resistance, WC, and

osteocalcin (OC) [4]. Another research of 382 postmenopausal women found that decreased OC levels were significantly associated with high blood glucose and WC [5]. Similarly, in our study, WC was strongly negatively linked with -CTX (C-terminal telopeptide) and OC in both the male and female groups, although there was no discernible association between WC and N-terminal propeptide of type I collagen (P1NP) in either group.

Conclusion:

The current investigation found that abdominal obesity showed a clearly unfavourable link with bone metabolism in T2DM patients. The abdominal obesity indices were substantially adversely linked with skeletal degradation (-CTX) and formation (OC) in both males and females, whereas the VAI was the only predictor relevant to P1NP in females. These easily acquired indices might be utilised as a preliminary screening approach and important indicators for osteodysfunction incidence risk in normal clinical practise at no additional expense, and may be of particular relevance for postmenopausal women in T2DM populations.

Both men and females' skeletal formation (-CTX) and degradation (OC) were significantly negatively correlated with the abdominal obesity indices, however the VAI was the only predictor important for N-terminal propeptide of type I collagen(P1NP) in females.

Reference:

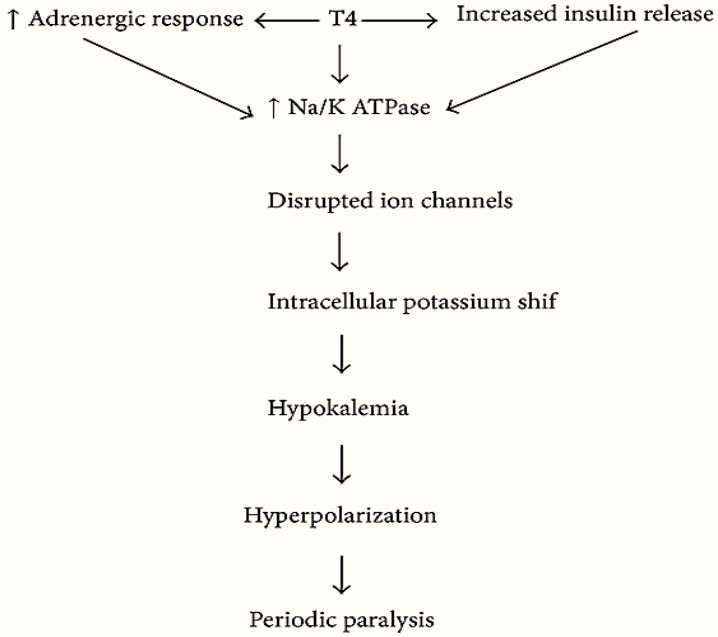
1. Guo H, An Z, Wang N, Ge S, Cai J, Yu S, Zhou Y, Ying R, Zha K, Gu T, Zhao Y. Diabetes Mellitus Type 2 Patients with Abdominal Obesity Are Prone to Osteodysfunction: A Cross-Sectional Study. Journal of Diabetes Research. 2023 Apr 17;2023.
2. Artasensi A, Pedretti A, Vistoli G, Fumagalli L. Type 2 diabetes mellitus: a review of multi-target drugs. Molecules. 2020 Apr 23;25(8):1987.
3. Lukács A, Horváth E, Máté Z, Szabó A, Virág K, Papp M, Sándor J, Ádány R, Paulik E. Abdominal obesity increases metabolic risk factors in non-obese adults: a Hungarian cross-sectional study. BMC Public Health. 2019 Dec;19:1-8.
4. Fuglsang-Nielsen R, Rakvaag E, Vestergaard P, Hartmann B, Holst JJ, Hermansen K, Gregersen S, Starup-Linde J. Consumption of nutrients and insulin resistance suppress markers of bone turnover in subjects with abdominal obesity. Bone. 2020 Apr 1;133:115230.
5. Movahed A, Larijani B, Nabipour I, Kalantarhormozi M, Asadipooya K, Vahdat K, Akbarzadeh S, Farrokhnia M, Assadi M, Amirinejad R, Bargahi A. Reduced serum osteocalcin concentrations are associated with type 2 diabetes mellitus and the metabolic syndrome components in postmenopausal women: the crosstalk between bone and energy metabolism. Journal of bone and mineral metabolism. 2012 Nov;30:683-91.

A case report of thyrotoxic periodic paralysis with no identified genetic predisposition

Introduction:

TPP (Thyrotoxic periodic paralysis) is a rare endocrine condition characterised by thyrotoxicosis, hypokalaemia, and sudden muscular weakness that most commonly occurs at night. Although girls are nine times more likely than males to get thyrotoxicosis, TPP is more prevalent in men (male to female ratios ranging from 17:1 to 70:1) and occurs [(1)]. It causes paralysis of both the proximal and distal muscles in the upper and lower limbs, as well as the respiratory and cardiac conduction systems [(2)]. This is a case report of a 19-year-old Caucasian man who was admitted to the hospital with paralysis in both his upper and lower limbs and tachycardia.

Mechanism of paralysis in TPP.



Case presentation:

A 19-year-old Caucasian male was admitted to the hospital with upper and lower limb paralysis and tachycardia. He had been experiencing anxiety, sweating more than normal, everyday palpitations, shortness of breath on exercise, and loose stools for several months, and had dropped 21 kg in the previous year. His vital signs at the time of admission were as follows: heart rate 120bpm, blood pressure 104/45mmHg, respiration rate 16/min, temperature 36.6°C, and oxygen saturation 99%. The electrocardiogram (ECG) revealed atrial flutter with 1:2 and 1:3 block (Fig. 1, panel A). Two hours after admission, his potassium levels had returned to normal, his potassium supplements had been discontinued, and he had regained strength in both his upper and lower limbs. The ECG went into sinus rhythm at a frequency of 100-120bpm (Fig. 1, panel B). An initial blood gas revealed very low potassium levels of 1.4mM, and blood tests revealed reduced Thyroid-stimulating hormone (TSH) levels of $<0.01\times 10^{-3}$ IU/L, increased free thyroxine (fT4) levels of 63.5 pM (reference interval (RI): 12.0-22.0 pM), and elevated total triiodothyronine (T3) levels of 8.2nM (RI: 1.0-2.6nM). He was diagnosed with TPP and was given liquid oral potassium chloride (30mmol every 30 minutes) and

propylthiouracil. TSH-receptor antibodies (TRAB) and thyroid-peroxidase antibodies (TPO-ab) levels were extremely high. Thyroid ultrasound revealed a normal-sized gland, and colour Doppler sonography revealed enhanced vascularity throughout the gland, both of which are consistent with Graves' disease. On day 4, he was discharged with a normal potassium level and was followed in the outpatient clinic, where he got conventional Graves' disease therapy. Whole-genome sequencing revealed no genetic variations in genes previously related with TPP.

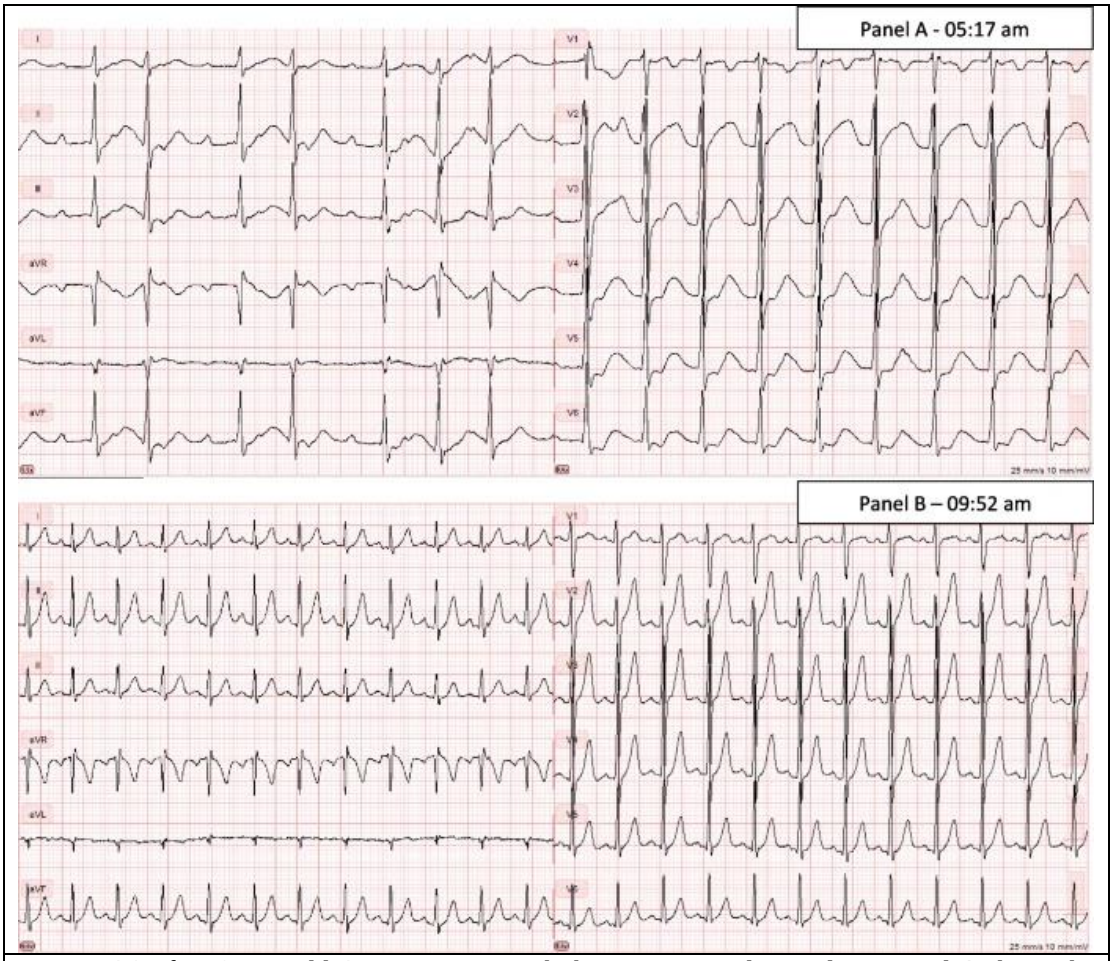


Fig. 1 ECG of a 19-year-old Caucasian man with thyrotoxic periodic paralysis. Panel A shows the ECG at arrival to the Emergency Department with a potassium of 1.4mM. Panel B (same day) shows the ECG after treatment with liquid oral potassium chloride (30mmol every 30minutes).

Discussion:

Thyrotoxic periodic paralysis (TPP), hypernatremic hypokalaemia paralysis (HHP), familial periodic paralysis (FPP), and idiopathic/sporadic periodic paralysis (SPP) are the aetiologies of hypokalemic periodic paralysis (HPP). SPP patients had no hyperthyroidism, hypernatremia, or a family history of HPP. According to Noso S et al, the majority of HPP cases in Caucasians are caused by FPP, whereas the majority of HPP cases in Asians are caused by TPP [(3)].

CACNA1S pathogenic variations account for up to 70% of all HPP cases, whereas SCN4A pathogenic variants account for roughly 10%. Fialho D, et al discovered that HPP produced by SCN4A or CACNA1S variants is inherited in an autosomal dominant manner [(4)].

The inwardly rectifying potassium channel, Kir2.1, is encoded by the KCNJ2 gene (potassium inwardly rectifying channel subfamily J Member 2). Park S, et al discovered that variants in the KCNJ2 gene are strongly linked with TPP [(5)].

Conclusion:

TPP is extremely rare in Caucasians, although it is more common in East Asian young males. Genetic testing of CACNA1S, KCNJ18, SCN4A, KCNJ2, KCNE3, and ABCC8 in a Caucasian male with TPP reveals no pathogenic mutations in genes previously related with TPP.

TPP (hypokalemic thyrotoxic periodic paralysis) is an uncommon endocrine disorder marked by thyrotoxicosis, hypokalaemia, and sudden muscle weakness that usually occurs at night.

References:

1. Patel M, Ladak K. Thyrotoxic periodic paralysis: a case report and literature review. Clinical Medicine & Research. 2021 Sep 1;19(3):148-51.

2. Heydorn A, Bertelsen B, Nolsöe RL, Eiken P, Kristensen PL. Thyrotoxic periodic paralysis in a Caucasian man without identifiable genetic predisposition: a case report. Thyroid Research. 2023 Dec;16(1):1-5.

3. Noso S, Babaya N, Hiromine Y, Ito H, Taketomo Y, Yoshida S, Niwano F, Monobe K, Minohara T, Okada T, Tsugawa M. Contribution of Asian haplotype of KCNJ18 to susceptibility to and ethnic differences in thyrotoxic periodic paralysis. The Journal of Clinical Endocrinology & Metabolism. 2019 Dec;104(12):6338-44.

4. Fialho D, Griggs RC, Matthews E. Periodic paralysis. Handbook of clinical neurology. 2018 Jan 1; 148:505-20.

5. Park S, Kim TY, Sim S, Oh HS, Song E, Kim M, Kwon H, Choi YM, Jeon MJ, Kim WG, Shong YK. Association of KCNJ2 Genetic Variants with Susceptibility to Thyrotoxic Periodic Paralysis in Patients with Graves' Disease. Experimental and Clinical Endocrinology & Diabetes. 2017 Feb;125(02):75-8.

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