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PHYSICIAN CONNECT

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The level of antimony in the blood correlates with the severity of pulmonary arterial hypertension.

Pulmonary arterial hypertension (PAH) group 1 is a rare disease, with an estimated annual incidence of 2 to 5 cases per million and a prevalence of 10.6 cases per million adults, with women having a higher prevalence. [1] It is distinguished by a precapillary hemodynamic profile that includes mean pulmonary artery pressure > 20 mm Hg, pulmonary vascular resistance 3 Wood Units (WU), and pulmonary artery wedge pressure 15 mmHg as measured by right heart catheterization (RHC) during rest, after other causes of precapillary pulmonary hypertension have been ruled out. PAH can be idiopathic, hereditary, drug- or toxin-induced, or linked to HIV infection, connective tissue disease, congenital heart disease, portal hypertension, or Schistosomiasis. [2] Oxidative and endoplasmic reticulum stress are important mediators of pulmonary hypertension and right ventricular dysfunction, and there is growing evidence of the role of heavy metals and environmental pollution in cardiovascular disease, which is mediated through several pathways linked to increased oxidative stress and changes in nitric oxide homeostasis. [3]

This was a prospective, single-center pilot study that enrolled patients over a three-month period. Patients with pulmonary arterial hypertension (PAH) over the age of 18 were eligible to take part. The PAH patients who were recruited had no prior history of occupational heavy metal exposure. Antimony levels in blood, plasma, and urine were measured using an X Series II quadrupole inductively coupled plasma mass spectrometry on blood and urine samples from 20 PAH patients and 10 volunteer apparently healthy controls. The ICP-MS trace metal assay protocol included several steps, all of which were designed to avoid metal contamination. The sample digestion tubes were treated with concentrated acid/nitric acid overnight before being washed/rinsed 5 times with deionized (DI) water and dried. The metal levels in the blood, plasma, and urine were compared between groups using a two-sample t-test (or ANOVA when comparing more than two factors) for continuous variables and a chi-square (2)-test (or Fisher's exact test when the expected frequency within any cell was5 in a 2 2 table) for categorical variables. The p value and Pearson's correlation coefficient were used to assess the relationship between heavy metals and clinical biomarkers. SAS 9.5 was used for statistical analysis, and results with p values less than 0.05 were considered significant.

When compared to controls, antimony blood and plasma levels were significantly higher in PAH patients [blood: PAH mean SD (95 % Confidence interval) 1.3 0.6 (1.0-1.5) ng/ml vs. control mean SD (95 %) 0.7 0.5 (0.4-1.0) ng/ml, p = 0]. .017] [plasma: PH mean SD (95 % confidence interval) 2.6 1 (2.2-3.1) ng/ml vs. control mean SD (95 % confidence interval) 1.5 0.8 (1.0-2.0) ng/ml, p = 0.004]. In addition, when idiopathic-PAH patients and non-idiopathic PAH patients were compared to controls, antimony blood and plasma levels were significantly higher. There was a trend for higher blood and plasma antimony levels in idiopathic PAH [blood 1.6 0.6 (1.1-2.1) ng/ml and plasma 3.1 1.2 (2.2-4.1) ng/ml] when compared to non-idiopathic PAH [blood 1.1 0.6(0.8-1.4) ng/ml and plasma 2.5 0.9(2-2.9) ng/ml]. There was a significant relationship between plasma Antimony level and all prognostic hemodynamic parameters of PAH (Fig. 1), including mean right atrial pressure

(mRAP) (r = 0.47, p = 0.036), cardiac output (CO) (r = -0.50, p = 0.026), cardiac index (CI) (r = -0.54, p = 0.014), pulmonary vascular resistance (PVR) (r = 0.52, p = 0.019), (Figure:1).

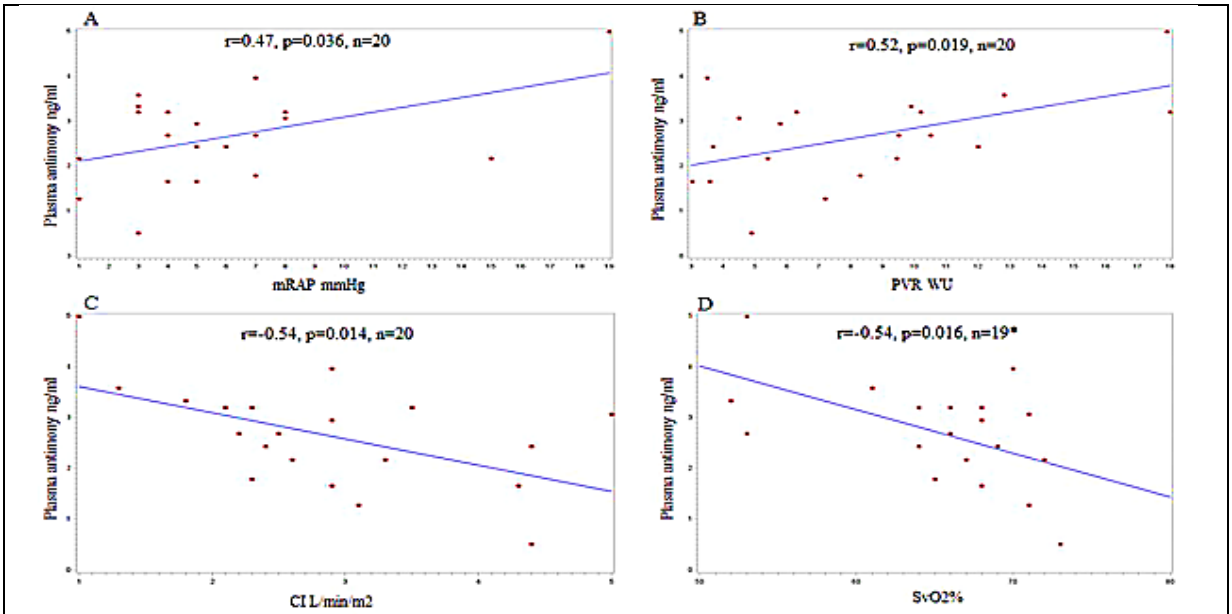


Fig. 1. Scatter plots of plasma antimony (Sb) and mRAP (A), PVR (B), CI (C), and SvO2% (D). *Svo2% was missing in one patient.

When compared to controls, PAH patients had significantly higher blood and plasma antimony levels in this pilot study. There was also a significant correlation between plasma antimony levels in PAH patients and all of the prognostic hemodynamic markers of PAH, such as mRAP, CO, CI, PVR, and SvO2. Patients with group 1 PAH are currently classified into different risk groups based on their estimated one-year mortality. The risk stratification strategy is a multiparametric evaluation that incorporates some RHC hemodynamic parameters. mRAP, CI, SvO2, and PVR are prognostic RHC parameters that are currently used in risk assessment. [4] This study focused on group 1 PAH and correlated antimony levels to prognostic hemodynamic markers that reflect right ventricular function, which are currently used in PAH risk assessment. Given the significant correlation between plasma antimony levels and PAH prognostic hemodynamic markers that reflect right ventricular function, it is possible that PAH patients with higher levels of environmental antimony levels are predisposed to right ventricular dysfunction, which is one of the main determinants of outcomes in PAH.

Previous studies aimed at better understanding antimony cardiac toxicity can be used to extrapolate the hypothesis of the involved mechanism of right ventricular dysfunction. Several studies have found that antimony exposure has negative effects on the heart. Antimony exposure caused cardiac induced apoptosis in mice via endoplasmic reticulum stress, which is linked to calcium homeostasis disruption, with cardiac myocytes showing granular degeneration, mitochondrial swelling, and endoplasmic reticulum dilation. [5] Another study with 3022 participants found a positive dose-response relationship between urinary antimony and urinary 8-iso-PGF2 level, which is a common biomarker for lipid peroxidation, as well as a negative association between urinary 8-iso-PGF2 and one or more heart rate variability parameters. [6]

When compared to controls, PAH patients had significantly higher antimony levels in their blood and plasma in this pilot study. The study also showed a link between plasma antimony levels and all of the prognostic hemodynamic parameters of PAH. These findings merit further investigation and confirmation in larger studies.

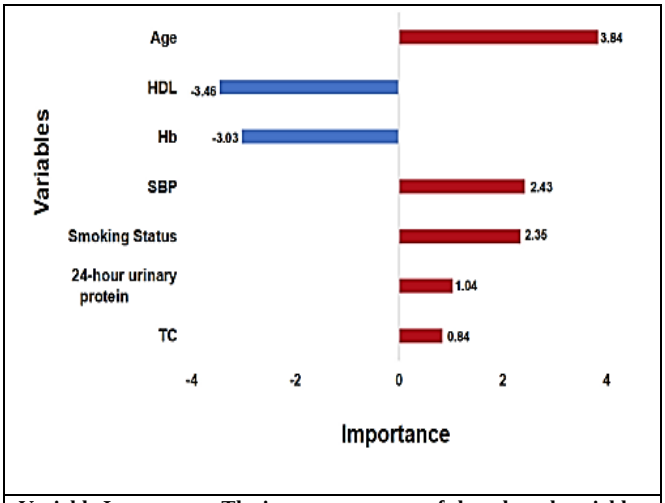
Reference:

1. El-Kersh K, Hopkins CD, Wu X, Rai SN, Cai L, Huang J. Plasma level of antimony correlates with pulmonary arterial hypertension severity. Current Research in Toxicology. 2022 Jun 27:100080.
2. Simonneau G, Montani D, Celermajer DS, Denton CP, Gatzoulis MA, Krowka M, Williams PG, Souza R. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. European Respiratory Journal. 2019 Jan 1;53(1).
3. Lahm T, Douglas IS, Archer SL, Bogaard HJ, Chesler NC, Haddad F, Hemnes AR, Kawut SM, Kline JA, Kolb TM, Mathai SC. Assessment of right ventricular function in the research setting: knowledge gaps and pathways forward. An Official American Thoracic Society Research Statement. American journal of respiratory and critical care medicine. 2018 Aug 15;198(4):e15-43.
4. Benza RL, Gomberg-Maitland M, Elliott CG, Farber HW, Foreman AJ, Frost AE, McGoon MD, Pasta DJ, Selej M, Burger CD, Frantz RP. Predicting survival in patients with pulmonary arterial hypertension: the REVEAL risk score calculator 2.0 and comparison with ESC/ERS-based risk assessment strategies. Chest. 2019 Aug 1;156(2):323-37.
5. Jiang X, Yu W, Wu S, Tang L, Zhong G, Wan F, Lan J, Zhang H, Pan J, Tang Z, Zhang X. Arsenic (III) and/or Antimony (III) induced disruption of calcium homeostasis and endoplasmic reticulum stress resulting in apoptosis in mice heart. Ecotoxicology and Environmental Safety. 2021 Sep 1;220:112394.
6. Tan Q, Ma J, Zhou M, Wang D, Wang B, Nie X, Mu G, Zhang X, Chen W. Heavy metals exposure, lipid peroxidation and heart rate variability alteration: association and mediation analyses in urban adults. Ecotoxicology and Environmental Safety. 2020 Dec 1;205:111149.

Cardiovascular Disease Prediction and Risk Stratification in Diabetic Kidney Disease Patients

Diabetes kidney disease (DKD) is one of the most serious diabetic microvascular complications, affecting up to 50% of diabetic patients and becoming the leading cause of end-stage renal disease (ESRD) worldwide (1-3). Patients with DKD are at high risk of developing cardiovascular disease (CVD), putting a strain on the public health system. [1] Renal function decline is regarded as an independent risk factor and predictor of CVD. [2] There are numerous risk factors that contribute to the high prevalence of CVD in DKD patients. Risk factor studies and CVD predictive tools, such as the Framingham, QRISK, and China-PAR models, are common in the general population. [3] The objective of this study was to create a predictive model based on deep learning to predict the progression of CVD in DKD patients. It can also help us investigate risk factors, make treatment recommendations for better cardiovascular outcomes, and support personalised medicine. Using the model's output risk values, patients were stratified into different risk subgroups. In addition, based on the predictive model, an easy-to-use online tool for calculating the incidence rate of CVD in DKD patients was developed.

A retrospective cohort study was carried out in DKD patients to assess the risk of developing composite cardiovascular disease, which includes coronary heart disease, cerebrovascular diseases, congestive heart failure, and peripheral artery disease. The variable selection was carried out using least absolute shrinkage and selection operator (LASSO) regression. DeepSurv, a deep learning-based survival model based on a feed-forward neural network, was developed to predict CVD risk in DKD patients. The concordance index (C-index), area under the curve (AUC), and integrated Brier scores were used to compare the model performance with the traditional Cox proportional hazards (CPH) model and the Random survival forest (RSF) model (IBS). The cumulative incidence curves for the two risk subgroups were plotted in the study. Figure 1 shows how the DeepSurv model-based risk stratification can successfully stratify patients into different risk groups with significant differences.



Variable Importance. The importance score of the selected variables is calculated by their weights in the DeepSurv model. SBP, systolic blood pressure; TC, total cholesterol; Hb, hemoglobin; HDL, high density lipoprotein.

In this retrospective study, 890 patients with DKD were recruited. During a median follow-up of 10.4 months, 289 patients developed a subsequent CVD. The predictive model was built using seven variables chosen by LASSO regression: age, high density lipoprotein (HDL), haemoglobin (Hb), systolic blood pressure (SBP), smoking status, 24 h urinary protein excretion, and total cholesterol (TC). In the validation set, the DeepSurv model performed

best, with a C-index of 0.767, AUC of 0.780, and IBS of 0.067. The study then divided the patients into different risk groups using the cut-off value determined by the ROC (receiver operating characteristic) curve. Furthermore, the DeepSurv model was used to create an online calculation tool for patients to use for risk monitoring.

This study used deep learning methods to develop models for CVD prediction and risk stratification in DKD patients without observing overfitting, demonstrating that this deep learning-based survival predictive model outperformed the conventional statistical method. Despite being the most widely used approach for survival analysis in analysing time-to-event survival data, the CPH model. [4]

Several risk factors have been linked to the high incidence of CVD in some DKD patients. In line with previous studies, this study found several recognised similar traditional risk predictors, including age, SBP, smoking status, HDL, and TC. According to a meta-analysis, these traditional risk factors, such as age, blood pressure, smoking status, and cholesterol levels, have been proven in previous classical predictive models based on the general population. [5] Blood pressure reduction is an important treatment strategy because it not only slows the progression of renal failure but also lowers the risk of cardiovascular disease. Smoking is regarded as an important and modifiable predictor of CVD progression.

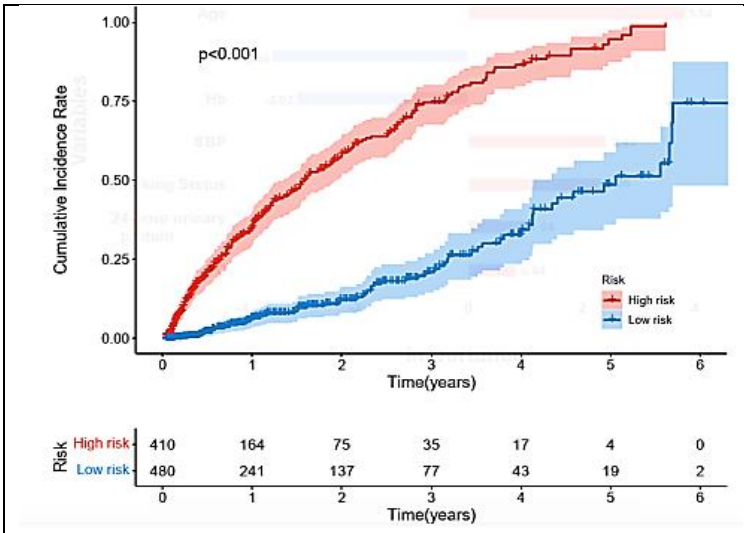


Fig. 1 Cumulative Incidence curves for predicted cardiovascular disease among diabetic kidney disease patients in different risk groups. Cardiovascular disease is the composite of coronary heart disease, cerebrovascular disease, congestive heart failure, and peripheral arterial disease. Patients were stratified into a high-risk group and a low-risk group based on the cut-off value of the ROC curve. The P-values between the high-risk and low-risk subgroups were calculated by the log-rank test

The Study of Heart and Renal Protection (SHARP) discovered that smoking increased the risk of vascular adverse complications in patients with chronic kidney disease and that this risk can be reduced by quitting smoking. [6]

The study also discovered that anaemia and high proteinuria play an important role in the occurrence of CVD in DKD patients. Anemia was found to be significantly linked to the occurrence of CVD events. Anemia can alter the structure of the heart. Long-term anaemia causes decreased oxygen capacity and utilisation problems. To maintain normal oxygen supply, compensatory hyperdynamic circulation is required, resulting in increased cardiac output and left ventricular hypertrophy. Anemia frequently contributes to recurrent and progressive cardiac and renal deterioration, a condition known as cardiorenal anaemia syndrome (CRAS). [7]

Finally, the study created and validated a new predictive model with good discrimination for estimating CVD risk in DKD patients using seven readily available clinical variables. Based on this model, a user-friendly online tool for clinical implementation and patient monitoring was created.

Reference:

1. Ren J, Liu D, Li G, Liu Z, Dong J, Duan J. Prediction and Risk Stratification of Cardiovascular Disease in Diabetic Kidney Disease Patients. Frontiers in Cardiovascular Medicine.:1600.
2. Kühn A, van der Giet M, Kuhlmann MK, Martus P, Mielke N, Ebert N, Schaeffner ES. Kidney function as risk factor and predictor of cardiovascular outcomes and mortality among older adults. American Journal of Kidney Diseases. 2021 Mar 1;77(3):386-96.
3. Corlin L, Short MI, Vasan RS, Xanthakis V. Association of the duration of ideal cardiovascular health through adulthood with cardiometabolic outcomes and mortality in the Framingham Offspring Study. JAMA cardiology. 2020 May 1;5(5):549-56.
4. Hou Y, Zhou Y, Hussain M, Budd GT, Tang WH, Abraham J, Xu B, Shah C, Moudgil R, Popovic Z, Watson C. Cardiac risk stratification in cancer patients: A longitudinal patient–patient network analysis. PLoS Medicine. 2021 Aug 2;18(8):e1003736.
5. Cahn A, Mosenzon O, Wiviott SD, Rozenberg A, Yanuv I, Goodrich EL, Murphy SA, Bhatt DL, Leiter LA, McGuire DK, Wilding JP. Efficacy and safety of dapagliflozin in the elderly: analysis from the DECLARE–TIMI 58 study. Diabetes Care. 2020 Feb 1;43(2):468-75.
6. Palmer SC, Mavridis D, Johnson DW, Tonelli M, Ruospo M, Strippoli GF. Comparative effectiveness of calcimimetic agents for secondary hyperparathyroidism in adults: a systematic review and network meta-analysis. American Journal of Kidney Diseases. 2020 Sep 1;76(3):321-30.
7. McCullough PA. Anemia of cardiorenal syndrome. Kidney International Supplements. 2021 Apr 1;11(1):35-45.

The PNDABLE Study: The Association Between Cardiovascular Disease Risk Score and Postoperative Delirium.

Postoperative delirium (POD), defined as an acute or fluctuating mental status impairment, is one of the most common adverse postoperative complications, with the elderly being at a higher risk because the predisposing risk factors are frequently associated with ageing. [1] As the population ages, the number of people suffering from dementia grows. [2] Perceptual, awareness, attention, and cognition dysfunction are the symptoms. POD begins in the recovery room and is common in the hospital up to one week after the procedure or until discharge. [3] The purpose of the study was to look into the relationship between the Framingham Heart Study general cardiovascular disease risk score (FHS-CVD risk score) and postoperative delirium (POD) in patients who had unilateral total knee arthroplasty under epidural anaesthesia. The study also investigated whether the hypothesised relationship was mediated by cerebrospinal fluid (CSF) biomarkers.

The current study included a total of 750 participants. And the data came from the Perioperative Neurocognitive Disorder and Biomarker Lifestyle (PNDABLE) study's database. The Mini-Mental State Examination was used to assess participants' preoperative cognitive function (MMSE). The Confusion Assessment Method was used to determine the prevalence of POD (CAM). The Memorial Delirium Assessment Scale was used to assess the severity of POD (MDAS). The following POD CSF biomarkers were included in the current study: A42, T-tau, P-tau, A42/T-tau, and A42/P-tau. In the PNDABLE study, the level of CSF biomarkers was measured using the enzyme-linked immunosorbent assay (ELISA). The relationship between the FHS-CVD risk score and the POD CSF biomarkers was investigated using linear regression analysis. The relationship between FHS-CVD risk score, POD CSF biomarkers, and POD incidence was investigated using logistic regression. Mediation Analysis with 10,000 bootstrapped iterations was used to evaluate the proposed mediating effect of CSF biomarkers. The receiver operating characteristic (ROC) curve is chosen as the evaluation metric for determining the FHS-CVD risk score's efficacy in predicting POD.

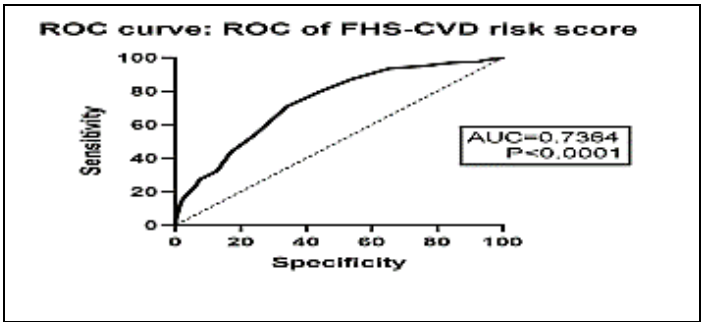


Figure 1. The ROC curve showed that FHS–CVD risk score had effective diagnostic significance in predicting POD occurrence.

The overall incidence of POD in the PNDABLE study was 22.9 %, with 37.2 percent in the higher vascular risk group and 7.9 percent in the lower vascular risk group. Multiple linear regression models revealed that in the higher vascular risk group, a higher preoperative FHS-CVD risk score was positively correlated with CSF T-tau and P-tau levels. The results of the logistic regression analysis demonstrated the effect of A42, A42/T-tau, and A42/P-tau in protecting patients against POD after adjusting for age (40-90 years), gender, education, MMSE, smoking history, drinking history, hypertension, diabetes, and the presence of CHD (cardiovascular heart disease). The FHS-CVD risk score, as well as the remaining two

biomarkers, T-tau and P-tau, were identified as risk factors. T-tau and P-tau were found to be partially mediated by the association between FHS-CVD risk score and POD in mediation analyses. The FHS-CVD risk score's predictive power was validated by the ROC curve, which had an AUC of 0.7364. (figure 1) The linear regression results of the connection between FHS–CVD risk score and CSF POD biomarkers were shown in Figure 2.

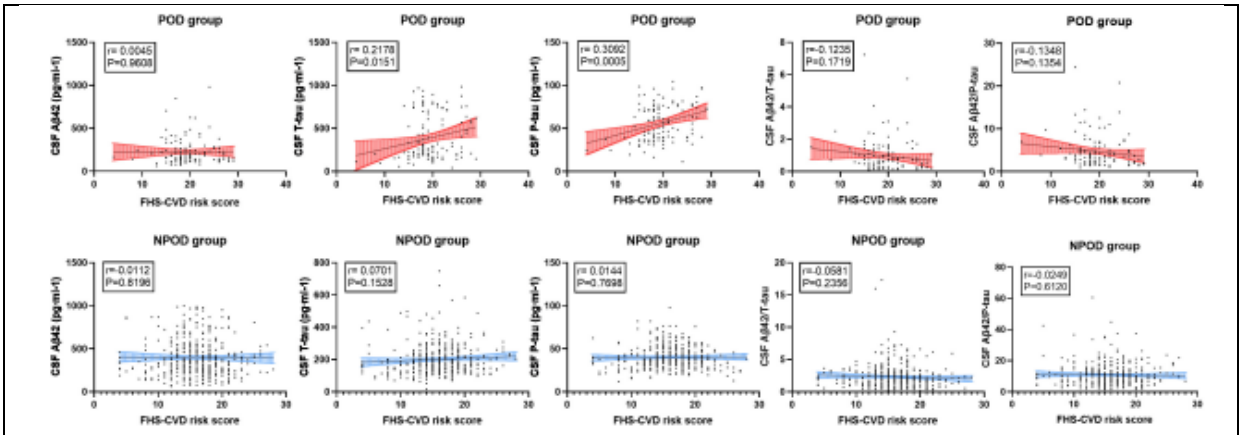


Figure 2. The linear regression models showed the relationship between the CSF POD biomarkers and FHS–CVD risk score. The result showed that higher preoperative FHS–CVD risk score were positively correlated with CSF T-tau and P-tau level in the POD group.

Postoperative delirium is a syndrome caused by the interaction of demographic factors, basic disease factors, and anaesthesia and surgery factors in patients. POD is the most common postoperative neurodegenerative change, and its prevalence is increasing as the world's population ages. However, there is currently a lack of effective treatments for POD, which has a serious impact on postoperative prognosis. Epidemiological studies have played an important role in the cognitive prevention of POD in recent years. A large number of studies have centralised and summarised potential controllable factors that can significantly reduce the prevalence of POD in preclinical asymptomatic stages. [4] There is no single vascular risk factor that has been linked to the occurrence and progression of POD. Given the interaction of vascular risk factors, this study chooses to summarise the influence of vascular risk factors in order to reach a potentially reliable conclusion. [5]

CSF biomarkers such as A42, T-tau, P-tau, A42/T-tau, and A42/P-tau concentrations in patients have been used as a set of important indicators in previous clinical research and POD diagnosis. Increases in the T-tau and P-tau levels, in particular, reflect the severity of axial degeneration and the number of neurofibrillary tangles found in patients with brain pathology. Concerning the amyloid protein A42, its accumulation in amyloid plaques during POD pathology changes has been shown to reduce A42 concentration in CSF. [6]

According to the study findings, patients with higher vascular risk have higher CSF T-tau and P-tau levels, but their CSF A42, A42/T-tau, and A42/P-tau levels appear to be lower. There were significant differences in the expression of A42, T-tau, P-tau, A42/T-tau, and A42/P-tau in CSF between the higher and lower vascular risk groups (P 0.05), indicating an increased risk of POD in patients with higher vascular risk. It should also be noted that it is a combination of vascular risk factors, not a single one, that can lead to vascular dysfunction. The FHS-CVD risk score is a simple but reliable vascular risk assessment tool that can be

used by taking into account multiple vascular risk and disadvantage conditions at the same time. The visual inspection of the ROC curve revealed that the FHS-CVD risk score was accurate in predicting POD. The FHS-CVD risk score evaluates items that are mostly curable. With such a broad range of items included, such an assessment tool could provide valuable guidance to clinicians in developing their prevention and treatment plans.

The study has potential public health implications, and proper use of medications such as antihypertensive, hypoglycemic, and lipid-lowering medications may help to prevent, slow, or even reverse the progression of postoperative delirium.

To summarise, one of the preoperative risk factors for promoting POD is a high vascular risk burden. POD is more common in patients with a higher FHS-CVD risk score. Furthermore, this study discovered that the relationship between FHS-CVD risk score and POD may be mediated in part by CSF tau protein. Early detection and treatment of POD can improve patient quality of life, reduce hospital stays, and lessen the burden on family and society.

Reference:

1. Wang J, Wang L, Tang X, Wang F, Liu S, Wu X, Dong R, Lin X, Wang B, Bi Y. The Relationship Between Cardiovascular Disease Risk Score and Postoperative Delirium: The PNDABLE Study. Frontiers in Aging Neuroscience. 2022;14.
2. Jia L, Quan M, Fu Y, Zhao T, Li Y, Wei C, Tang Y, Qin Q, Wang F, Qiao Y, Shi S. Dementia in China: epidemiology, clinical management, and research advances. The Lancet Neurology. 2020 Jan 1;19(1):81-92.
3. Evered L, Silbert B, Knopman DS, Scott DA, DeKosky ST, Rasmussen LS, Oh ES, Crosby G, Berger M, Eckenhoff RG, Nomenclature Consensus Working Group. Recommendations for the nomenclature of cognitive change associated with anaesthesia and surgery—2018. Anesthesiology. 2018 Nov;129(5):872-9.
4. Iadecola C. The neurovascular unit coming of age: a journey through neurovascular coupling in health and disease. Neuron. 2017 Sep 27;96(1):17-42.
5. He JT, Zhao X, Xu L, Mao CY. Vascular risk factors and Alzheimer's disease: blood-brain barrier disruption, metabolic syndromes, and molecular links. Journal of Alzheimer's Disease. 2020 Jan 1;73(1):39-58.
6. Cunningham EL, McGuinness B, McAuley DF, Toombs J, Mawhinney T, O'Brien S, Beverland D, Schott JM, Lunn MP, Zetterberg H, Passmore AP. CSF beta-amyloid 1–42 concentration predicts delirium following elective arthroplasty surgery in an observational cohort study. Annals of surgery. 2019 Jun 1;269(6):1200-5.

A Case of Banti's Syndrome in an Adult Male

Massive splenomegaly, pancytopenia due to hypersplenism, and portal hypertension without liver pathology characterise Banti's syndrome. [1] When all other causes of portal hypertension, splenomegaly, and pancytopenia are ruled out, it is a diagnosis of exclusion. Guido Banti, an Italian professor, was the first to describe this syndrome in the late 1800s. It is known by various names around the world. In India, it is known as non-cirrhotic portal fibrosis. [2] The spleen is the organ most affected by this syndrome. Varices on endoscopy, cytopenia of one or more cell lines, elevated portal hypertension with prominent portosystemic shunts, absence of liver cirrhosis, patent hepatic veins, and normal liver function tests are other features. This syndrome is complicated in its later stages by varices rupture and life-threatening haemorrhage.

A 29-year-old male, a known type 2 diabetic with a 15-year history of hematemesis and melena, presented to us with abdominal distention, right leg cellulitis, and fever. No history of jaundice, diarrhoea, easy bruising, cough, altered sensorium, chest pain, palpitations, breathlessness, orthopnea, or paroxysmal nocturnal dyspnea was present (PND).

On general physical examination, the patient was thin, pale-looking, supine on the bed, and well oriented in time, place, and person. He had a fever (100 F). His pulse rate was 94 beats per minute, regular, and of normal volume and character. He had a blood pressure of 120/70 mmHg. Icterus, clubbing, cyanosis, or lymphadenopathy were not present. JVP (jugular venous pressure) was normal. Massive splenomegaly was found on abdominal examination, with a palpable edge beyond the umbilicus (grade 4). The liver could be felt. The abdomen was distended with positive fluid shift, and the skin over the abdomen was thin and shiny, with no visible pulsations or dilated veins.

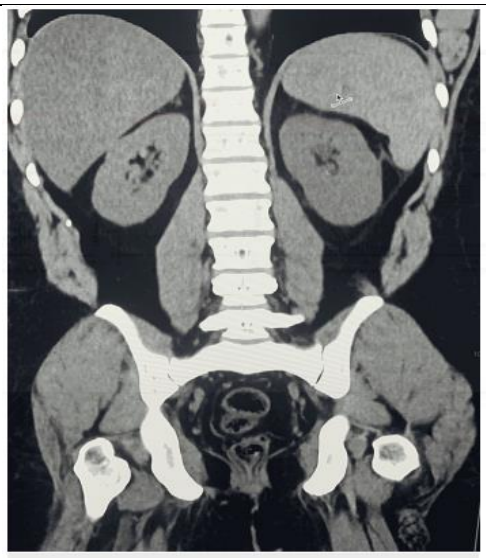


Fig.1 CT scan of abdomen (coronal view without contrast) shows: liver is enlarged with diffuse fatty infiltration; gallbladder is normal without any intraluminal calculus; adrenals and pancreas are unremarkable; spleen is enlarged and measures 26 cm with homogenous texture.

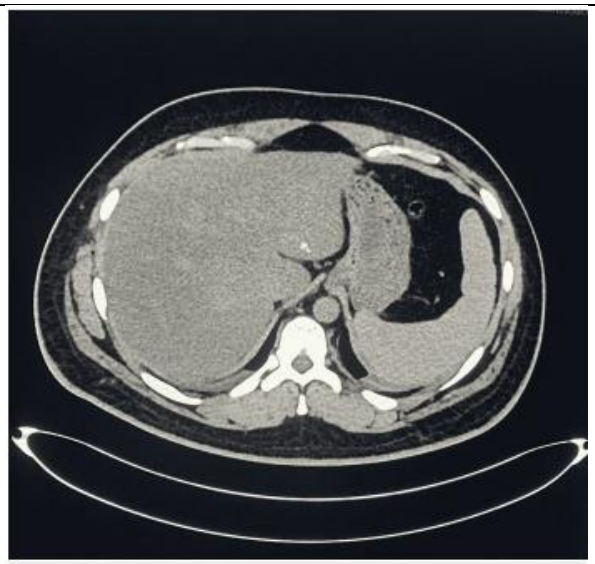


Fig.2 CT scan of abdomen (axial view without contrast)

The review of other systems was normal. Liver and renal function tests were both within normal limits. The prothrombin time, the activated partial thromboplastin time, and the international normalised ratio (INR) were all within normal limits. Urine routine testing was normal. Inflammatory markers were slightly out of whack. HBsAg, anti-HIV, and anti-HCV antibodies were all negative.

WBC count was 3700/mm3, RBC count was 2.72 million/mm3, haemoglobin was 7.6 gm%, mean corpuscular volume was 81.3, and platelets were 25,000. Lymphocytes made up 4.5 percent of the differential leukocyte count (DLC), neutrophils made up 90%, eosinophils made up 0.5 percent, monocytes made up 5%, and basophils made up 0%. Malarial parasites were not found in the peripheral smear. Ultrasonography and a CT scan of the abdomen and pelvis (Figures 1 and 2) revealed massive splenomegaly measuring 26 cm and having a homogenous texture. The portal vein was dilated 16mm, with a maximum flow rate of 15 cm/sec. There was severe abdominopelvic ascites.

Banti's syndrome (also known as Banti's disease, hypersplenism, idiopathic congestive splenomegaly, or IPH) is a chronic splenic enlargement that causes blood cell destruction. Banti's syndrome symptoms include weakness, fatigue, anaemia, and splenomegaly. Hematemesis and melena, which are symptoms of the condition, can exacerbate the anaemia over time. Although liver cirrhosis can develop, splenomegaly is the primary symptom, and ascites can develop. Coronavirus disease 2019 (COVID-19) has also been linked to Banti's Syndrome. [3]

Patients with Banti's syndrome frequently have pancytopenia-related symptoms, such as the risk of severe infections, bruising, and fatigue. Banti's syndrome has a multifactorial aetiology that includes the role of infectious agents (hepatitis B virus), coagulation abnormalities, and toxic metal exposure, including arsenic. [4]

The most common symptoms of Banti's syndrome are GI haemorrhage and splenomegaly, both of which this patient had. In an emergency, endoscopic sclerotherapy and endoscopic variceal band ligation can be performed. Even in the absence of cirrhosis, beta-blockers should be used as primary prophylaxis for portal hypertension. Recurrent bleeding and severe anaemia requiring multiple blood transfusions may necessitate splenectomy. Patients with Banti's syndrome can have an excellent prognosis if variceal haemorrhage is successfully treated, with a five-year survival rate of up to 100%.

Banti's syndrome is a chronic congestive enlargement of the spleen that results in blood cell destruction and pancytopenia. Given its rarity, one must be cautious in diagnosing this disease among other possibilities, as well as in providing the necessary care to treat the illness.

Reference:

1. Khan AR, Wazir MH, Waqar S, Ullah R, Gul A. Banti's Syndrome in an Adult Male: A Case Report. Cureus. 2022 May 31;14(5).
2. Goel A, Ramakrishna B, Zachariah U, Sajith KG, Burad DK, Kodiatte TA, Keshava SN, Balasubramanian KA, Elias E, Eapen CE. What makes non-cirrhotic portal hypertension a common

disease in India? Analysis for environmental factors. The Indian Journal of Medical Research. 2019 Apr;149(4):468.
3. Malik ZR, Razaq Z, Siff M, Sheikh S. COVID-19 presenting as Banti's syndrome. Cureus. 2020 Jul 9;12(7).
4. Maruyama H, Kondo T, Sekimoto T, Yokosuka O. Differential clinical impact of ascites in cirrhosis and idiopathic portal hypertension. Medicine. 2015 Jul;94(26).