REVIEW ARTICLE

Will Oral Semaglutide be a Game-Changer in the Management of Type 2 Diabetes in Indian Context?

ARJUN BAIDYA*, SAURABH MISHRA†, ASHOK VENKATANARASU‡

ABSTRACT

The glucagon-like peptide-1 receptor agonists (GLP-1RAs) have important beneficial effects on glycemic control and body weight along with their pleiotropic effects on various systems of the body. However, until now these agents were administered via an injection posing a challenge to patient convenience. Oral semaglutide is a first in class oral GLP-1RA co-formulated with an absorption enhancer for the treatment of type 2 diabetes mellitus (T2DM). The clinical efficacy and safety of oral semaglutide has been extensively evaluated in the Peptide InnOvatioN for Early diabEtes tReatment (PIONEER) program of clinical trials. This review shall elaborate on the unique diabetes situation in India and why the oral GLP-1RA (semaglutide) will be a game-changer in the Indian setting.

Keywords: Semaglutide, type 2 diabetes, GLP-1RAs, glucose-lowering drugs

Type 2 diabetes mellitus (T2DM) accounts for almost 90% of all diabetes cases worldwide. The prevalence of diabetes around the world will reach up to 592 million by the year 2035.[1] The genetic component among South Asians makes them up to four times more susceptible to T2DM compared to other ethnic groups.[2] The concept of an "Asian Indian Phenotype" was advanced by Mohan et al,[3] as the presence of insulin resistance along with abdominal obesity, higher C-reactive protein (CRP) and lower levels of adiponectin. Asian Indians have a lean-fat body composition with higher levels of central obesity (waist circumference, waist-to-hip ratio and visceral fat). They also have more body fat for a given body mass index (BMI) compared to other ethnic groups.[4] Thus, the lean-fat Indian is at a larger risk of diabetes, which results from genetic predisposition along with other factors like lifestyle changes, rapid urbanization and changing dietary patterns.

The baseline data of Indian type 2 diabetic patients in an observational study showed high prevalence of micro- and macrovascular complications due to poor glycemic control (mean glycated hemoglobin [HbA1c] = 9.2 ± 1.4).[5] The relation between glycemic status and incidence of complications highlights the importance of optimum glycemic control in T2DM. The glycemic control, however, continues to deteriorate as the disease progresses.[6]

Obesity which is often described as 'Diabesity' in obese type 2 diabetics is a major risk factor leading to hypertension, hyperlipidemia, atherosclerotic cardiovascular disease (ASCVD) and its complications, and also to many types of cancers.[7] The prevalence of diabesity is reaching epidemic proportions around the globe with no clear guidelines for its optimum management.[8] In Indian adults aged 20 to 69 years, the prevalence of overweight will more than double while the prevalence of obesity will triple by 2040.[9]

The management of patients with T2DM has become individualized with different therapies available and presence of specific patient factors that influence the appropriate choice of medication. In 2018, the American Diabetes Association (ADA) presented a decision algorithm, which included assessment of key

*Associate Professor, Dept. of Endocrinology, NRS Medical College, Kolkata, West Bengal
†Consultant Physician and Diabetologist, Centre for Diabetes and Endocrine Diseases, Kanpur, Uttar Pradesh
‡Consultant Endocrinologist, Sanjeevani Thyroid Sugar and Hormones Clinic, Hyderabad, Telangana
Address for correspondence
Dr Arjun Baidya
Associate Professor
Dept. of Endocrinology, NRS Medical College, Kolkata, West Bengal
E-mail: arjun.baidya@gmail.com

patient characteristics including comorbidities like ASCVD, chronic kidney disease (CKD) or heart failure (HF). The presence of these comorbidities should allow preferential use of certain classes of glucose-lowering drugs as second-line therapy.[10]

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are an established class of glucose-lowering drugs, which have a pleiotropic action on the pathophysiological defects of T2D, leading to effective glycemic control, loss of weight, minimal risk of hypoglycemia and a consistent safety profile.[11]

GLP-1RAs have similar mechanism of action but they vary in structure, pharmacokinetics and efficacy (their ability to reduce HbA1c, body weight and cardiorenal protection).[12,13]

The previous success in clinical trials of exenatide and liraglutide renewed interest in the GLP-1 therapy area. The daily injection regime was inconvenient for some patients and so better patient convenience was needed for patient adherence and satisfaction.[14] The fear of injections and difficulty in administration along with the perception of injectable therapy was a major barrier to the use of GLP-1RA therapy.[11]

Semaglutide is a GLP-1RA with 94% structural homology to endogenous GLP-1, and it has three important structural differences that prolong its half-life but do not compromise receptor binding. The efficacy and safety of subcutaneous semaglutide is already demonstrated in numerous clinical studies. The efficacy of oral semaglutide was expected to correspond with subcutaneous semaglutide and was proved with the Peptide InnOvatioN for Early diabEtes tReatment (PIONEER) studies.[15]

Oral semaglutide is co-formulated with SNAC {Sodium N-[8-(2-hydroxybenzoyl)amino]caprylate} which is an absorption enhancer and promotes semaglutide absorption across the gastric mucosa. This review will specifically elaborate on why oral semaglutide will be ideal for Indian diabetic patient in the light of evidence from studies on oral and injectable semaglutide.

DIABETES AND PREDIABETES

The Indian Council of Medical Research-India Diabetes (ICMR-INDIAB) study was a national study designed to estimate the prevalence of diabetes and prediabetes in Indian population. It was the largest ever study conducted to capture the diabetes picture in India.

The prevalence findings were reported from 15 states, which represented 50.7% of the adult population of the country. The main factors identified to be driving the epidemic of diabetes in India were obesity, age and a family history of T2DM. Prediabetes prevalence in India was high and exceeded diabetes in many states implying a huge risk of progression to overt diabetes.[16] This finding is very important in the Indian context as it has been shown in several studies that Asian Indians progress faster through the prediabetes stage when compared with other ethnic groups.[17]

Beta-cell dysfunction was prominent even with mild dysglycemia in the Asian Indian population (impaired glucose tolerance [IGT] or impaired fasting glucose [IGF] or both). This finding is important as it highlights the need for primary prevention strategies focussing on preservation of beta-cell function and reduction in cell decline.[18]

Increase in beta-cell function and insulin biosynthesis was shown with semaglutide along with improved proinsulin to insulin ratios when compared with other antidiabetic agents including sulfonylureas, which increase insulin secretion with no effect on the biosynthesis of insulin. Also, reduction in insulin resistance was greater with semaglutide vs. placebo, sitagliptin or exenatide extended-release (ER).[19]

Glycemic Efficacy of Oral Semaglutide

Oral semaglutide was effective in reducing HbA1c across the PIONEER trials. In the PIONEER 1 trial, oral semaglutide monotherapy significantly reduced baseline HbA1c compared with placebo after 26 weeks treatment in patients with early T2DM.

In patients with established T2DM who were receiving background oral antidiabetic medications (PIONEER 2-4), 14 mg of oral semaglutide was more effective than empagliflozin 25 mg, sitagliptin 100 mg and similar to liraglutide 1.8 mg at week 26. Flexible dose adjustment of oral semaglutide was more effective than sitagliptin 100 mg for reducing HbA1c at 52 weeks in the PIONEER 7 trial.

In advanced T2DM patients receiving insulin, oral semaglutide significantly reduced HbA1c as compared with placebo at weeks 26 and 52. In patients with moderate renal impairment (PIONEER 5), oral semaglutide 14 mg was significantly more effective than placebo at reducing HbA1c at week 26. In high cardiovascular (CV) risk patients (PIONEER 6), oral semaglutide reduced HbA1c by a mean of –1.0% vs. –0.3% in the placebo group.

Proportion of patients who achieved ADA recommended target of HbA1c <7.0% was persistently greater with

7 and 14 mg of oral semaglutide as compared with placebo and active comparators. Fasting plasma glucose was also generally reduced in patients on oral semaglutide as compared to the placebo and active comparator groups.[20]

OBESITY

Obesity in India is continuously growing and the recent trends indicate a rate anywhere between 13% to 50% of the urban population and 8% to 38.2% of rural population prevalence of obesity. Obesity among Asian Indians has distinctive features like greater truncal, intra-abdominal, subcutaneous and total adipose tissue compared with Caucasians.[21] Several comorbid conditions are associated with obesity like hypertension, hyperglycemia, dyslipidemia, nonalcoholic fatty liver disease (NAFLD), etc. This constellation of conditions is broadly defined as metabolic syndrome.[22]

NAFLD is an important component of metabolic syndrome which can progress to fibrosis and even cirrhosis if there is presence of portal inflammation (nonalcoholic steatohepatitis [NASH]).[23] Approximately one-fourth of urban Indian population has NAFLD and according to a case-control study, Asian Indians in North India with NAFLD have increased adipose tissue, fasting hyperinsulinemia, IGT and metabolic syndrome.[24] The improvement in NAFLD/NASH with GLP-1RAs is thought to be through an indirect mechanism reducing inflammation.[25]

Dyslipidemia is the increased level of total and low-density lipoprotein (LDL) cholesterol, decreased high-density lipoprotein (HDL) cholesterol and hypertriglyceridemia (present alone or together).[26] In Asian Indians with insulin resistance, the plasma adipose tissue metabolites, fatty acids and leptin are higher along with lower adiponectin levels.[27] In a study conducted with oral semaglutide to assess its effects on postprandial glucose and lipid metabolism, it was found that fasting LDL and total cholesterol concentrations were lower with oral semaglutide compared with placebo.

Treatment with oral semaglutide also resulted in lower fasting and postprandial triglycerides than with placebo. In the PIONEER 6 trial, improvements in elevated total cholesterol, LDL and triglycerides, reduced HDL were seen with oral semaglutide. The trial met its primary objective of proving CV safety of oral semaglutide.[28]

Body Weight Reduction with Oral Semaglutide

In the PIONEER clinical trial program, greater number of patients achieved a weight loss of ≥5% across the clinical trials with oral semaglutide 7 and 14 mg (13-44%) versus placebo (3-15%) and active comparators (10-36%) at week 26, which was sustained at the end of trial. Other body size measures like BMI and waist circumference were also reduced with oral semaglutide compared with placebo and active comparators.[20]

ATHEROSCLEROTIC CARDIOVASCULAR DISEASE

According to the Global Burden of Disease study, 24.8% of all deaths in India are associated with cardiovascular disease (CVD). Ischemic heart disease and stroke are responsible for 21.1% of all deaths in India.[29] T2DM and the associated microvascular (retinopathy, neuropathy and nephropathy) and macrovascular (coronary artery disease, peripheral arterial disease and stroke) complications contribute substantially to the morbidity and mortality of the disease. The core pathophysiological mechanism leading to arterial lumen narrowing is atherosclerosis. Recent studies have indicated the central role played by endothelium and inflammation in atherosclerosis.[30]

In animal studies, semaglutide reduced the size of the aortic atherosclerotic plaque lesion independent of its effect on diabetes, body weight and lipids.[25] It is important to note that the findings from the cardiovascular outcomes trial (CVOT) with semaglutide showing effects consistent with reduction in atherosclerotic burden, suggest that the findings seen in animal studies may translate to humans.[14]

The largest cause of diabetes associated morbidity and mortality is CVD. The international diabetology and cardiology guidelines have been updated to put forth a combined approach for the management of T2DM and CVD. The GLP-1RAs or sodium-glucose co-transporter 2 (SGLT2) inhibitors, which have a demonstrated CV benefit are recommended as first- or second-line agents in this regard.

The CAPTURE study found that almost 1 out of 3 adults with T2DM had established CVD. The management of most participants was not according to recent guidelines on diabetes and cardiac disease. There was an unmet need of reducing risk through interventions based on current evidence.[31]

Cardiovascular Safety of Oral Semaglutide

The PIONEER 6 trial was a CVOT designed to establish the CV safety of oral semaglutide; it was not powered for proof of superiority and CV benefit. The investigators concluded the noninferiority of oral semaglutide safety profile to placebo, on a background

of standard care. The CVOT of oral semaglutide to prove superiority in major adverse CV event (MACE) reduction is ongoing as A Heart Disease Study of Semaglutide in Patients with Type 2 Diabetes (SOUL). Pooled analysis, which combined data from CVOTs of oral and injectable semaglutide showed that the once-daily oral and once-weekly injectable showed very similar effects on glycemic and body weight control. Post-hoc analyses suggest a potential for improved CV outcomes with semaglutide irrespective of the route of administration.[32]

HYPOGLYCEMIA

There is a huge corpus of evidence available suggesting that intensive glycemic control with a goal of euglycemia should be instituted as early as possible in diabetic patients. The Diabetes Control and Complications Trial (DCCT) and Stockholm Diabetes Intervention Study (SDIS) showed reduction in the incidence of microvascular complications with intensive glycemic control in type 1 diabetes. The United Kingdom Prospective Diabetes Study (UKPDS) and Kumamoto study found out that tighter glycemic control can delay the onset and progression of micro- and macrovascular complications in T2DM patients.[33-37]

However, due to the risk of hypoglycemia, strict glycemic control is not achieved in majority of patients in real life clinical setting, and this was also a major finding in the above studies. In the DCCT, there was a threefold increase in severe hypoglycemia with intensive therapy as compared with conventional therapy during the study. In the UKPDS, major hypoglycemic episode in a year was significantly higher in the intensive treatment group.[33,35]

The risk of hypoglycemia is increased with insulin excess (exogenous insulin or agents causing release of insulin) and faulty glucose regulation. Progressive beta-cell failure in T2DM increase the severity of hypoglycemic episodes.[37]

Hypoglycemia is a significant barrier to patient adherence to medications leading to suboptimal glycemic control along with the risk of development of complications. Recurrent hypoglycemia worsens the quality of life and can also be fatal.[38]

In a cross-sectional study conducted in an Indian hospital, to find out proportion of T2DM patients reporting at least one or other symptom of hypoglycemia, almost 96% of subjects reported one or the other symptoms of hypoglycemia. Severe hypoglycemia episodes were reported by 19% patients and 8% patients required admission due to hypoglycemia. This study showed the reported prevalence of hypoglycemia among T2DM patients and the urgent need for intervention.[39]

GLP-1RAs have an inherently low propensity to cause hypoglycemia, which was also consistent with oral semaglutide. The PIONEER 4 study was associated with very low proportions of patients experiencing severe or blood-glucose confirmed hypoglycemia (1% and 2% patients, compared with 2% in placebo group). In the PIONEER 8 study, the number of such events was higher in patients having background insulin therapy, but the addition of oral semaglutide to insulin did not increase proportion of patients with hypoglycemia compared to placebo. Most events occurred in patients receiving basal-bolus background therapy with insulin.[40]

CONCLUSION

Oral semaglutide is a revolutionary new drug in the management of T2DM which overcomes the injectable barrier associated with GLP-1RA therapy. It is administered as a co-formulation with an absorption enhancer called SNAC. Oral semaglutide has glycemic control and weight reduction benefits consistent with the GLP-1RA class. India is fast becoming the type 2 diabetes capital of the world with associated conditions like obesity and ASCVD complicating the picture. The pleiotropic benefits of GLP-1RAs are well known and are consistent with oral semaglutide. All the guidelines in diabetes and cardiology have evolved and now recommend a cardiovasculocentric approach to T2DM as opposed to earlier more glucocentric approach.

With oral semaglutide, we have a robust data on the clinical efficacy and safety of oral semaglutide as well as the added advantage of once-daily oral administration, improving patient convenience. The beneficial effects with oral semaglutide like superior glycemic control, weight loss, CV safety and minimal risk of hypoglycemia make it a game-changer for T2DM management in India.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas. 8th Edition. Available from: http://www.diabetesatlas.org/resources/2017-atlas.html. Accessed May 15, 2018.
2. Kooner JS, Saleheen D, Sim X, Sehmi J, Zhang W, Frossard P, et al. Genome-wide association study in individuals of South Asian ancestry identifies six new type 2 diabetes susceptibility loci. Nat Genet. 2011;43(10):984-9.
3. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res. 2007;125(3):217-30.

4. Banerji MA, Faridi N, Atluri R, Chaiken RL, Lebovitz HE. Body composition, visceral fat, leptin, and insulin resistance in Asian Indian men. J Clin Endocrinol Metab. 1999;84(1):137-44.
5. Mohan V, Shah S, Saboo B. Current glycemic status and diabetes related complications among type 2 diabetes patients in India: data from the A1chieve study. J Assoc Physicians India. 2013;61(1 Suppl):12-5.
6. Cook M, Girman C, Stein P, Alexander C, Holman RR. Glycemic control continues to deteriorate after sulfonylureas are added to metformin among patients with type 2 diabetes. Diabetes Care. 2005;28(5):995-1000.
7. Behl S, Misra A. Management of obesity in adult Asian Indians. Indian Heart J. 2017;69(4):539-44.
8. Pappachan JM, Viswanath AK. Medical management of diabesity: do we have realistic targets? Curr Diabetes Rep. 2017;17(1):4.
9. Luhar S, Timæus IM, Jones R, Cunningham S, Patel SA, Kinra S, et al. Forecasting the prevalence of overweight and obesity in India to 2040. PLoS One. 2020;15(2):e0229438.
10. Davies MJ, D'Alessio DA, Fradkin J, Kernan WN, Mathieu C, Mingrone G, et al. Management of hyperglycemia in type 2 diabetes, 2018. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care. 2018;41:2669-701.
11. Romera I, Cebrián-Cuenca A, Álvarez-Guisasola F, Gomez-Peralta F, Reviriego J. A review of practical issues on the use of glucagon-like peptide-1 receptor agonists for the management of type 2 diabetes. Diabetes Ther. 2019;10(1):5-19.
12. Nauck MA, Meier JJ. Management of endocrine disease: are all GLP-1 agonists equal in the treatment of type 2 diabetes? Eur J Endocrinol. 2019;181(6):R211-34.
13. Caruso I, Cignarelli A, Giorgino F. Heterogeneity and similarities in GLP-1 receptor agonist cardiovascular outcomes trials. Trends Endocrinol Metab. 2019;30(9): 578-89.
14. Knudsen LB, Lau J. The discovery and development of liraglutide and semaglutide. Front Endocrinol (Lausanne). 2019;10:155.
15. Niman S, Hardy J, Goldfaden RF, Reid J, Sheikh-Ali M, Sutton D, et al. A review on the efficacy and safety of oral semaglutide. Drugs R D. 202121(2):133-48.
16. Anjana RM, Deepa M, Pradeepa R, Mahanta J, Narain K, Das HK, et al; ICMR–INDIAB Collaborative Study Group. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. Lancet Diabetes Endocrinol. 2017;5(8):585-96.
17. Anjana RM, Rani CS, Deepa M, Pradeepa R, Sudha V, Nair H, et al. Incidence of diabetes and prediabetes and predictors of progression among Asian Indians: 10-year follow-up of the Chennai Urban Rural Epidemiology Study (CURES). Diabetes Care. 2015;38(8):1441-8.
18. Staimez LR, Weber MB, Ranjani H, Ali MK, Echouffo-Tcheugui JB, Phillips LS, et al. Evidence of reduced β-cell function in Asian Indians with mild dysglycemia. Diabetes Care. 2013;36(9):2772-8.
19. Knudsen LB, Lau J. The discovery and development of liraglutide and semaglutide. Front Endocrinol (Lausanne). 2019;10:155.
20. Thethi TK, Pratley R, Meier JJ. Efficacy, safety and cardiovascular outcomes of once-daily oral semaglutide in patients with type 2 diabetes: The PIONEER programme. Diabetes Obes Metab. 2020;22(8):1263-77.
21. Misra A, Shrivastava U. Obesity and dyslipidemia in South Asians. Nutrients. 2013;5(7):2708-33.
22. Smith DG. Epidemiology of dyslipidemia and economic burden on the health care system. Am J Manag Care. 2007;13 Suppl 3:S68-71.
23. Garg A, Misra A. Hepatic steatosis, insulin resistance, and adipose tissue disorders. J Clin Endocrinol Metab. 2002;87(7):3019-22.
24. Bajaj S, Nigam P, Luthra A, Pandey RM, Kondal D, Bhatt SP, et al. A case-control study on insulin resistance, metabolic co-variates & prediction score in non-alcoholic fatty liver disease. Indian J Med Res. 2009;129(3):285-92.
25. Rakipovski G, Rolin B, Nøhr J, Klewe I, Frederiksen KS, Augustin R, et al. The GLP-1 analogs liraglutide and semaglutide reduce atherosclerosis in $ApoE^{-/-}$ and $LDLr^{-/-}$ mice by a mechanism that includes inflammatory pathways. JACC Basic Transl Sci. 2018;3(6):844-57.
26. Vega GL. Management of atherogenic dyslipidemia of the metabolic syndrome: evolving rationale for combined drug therapy. Endocrinol Metab Clin North Am. 2004;33(3): 525-44, vi.
27. Abate N, Chandalia M, Snell PG, Grundy SM. Adipose tissue metabolites and insulin resistance in nondiabetic Asian Indian men. J Clin Endocrinol Metab. 2004;89(6): 2750-5.
28. Dahl K, Brooks A, Almazedi F, Hoff ST, Boschini C, Bækdal TA. Oral semaglutide improves postprandial glucose and lipid metabolism, and delays gastric emptying, in subjects with type 2 diabetes. Diabetes Obes Metab. 2021;23(7):1594-603.
29. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. Circulation. 2016;133(16):1605-20.
30. Kulkarni NB, Ganu MU, Godbole SG, Deo SS. Assessment of potential biomarkers of atherosclerosis in Indian patients with type 2 diabetes mellitus. Indian J Med Res. 2018;147(2):169-76.
31. Mosenzon O, Alguwaihes A, Leon JLA, Bayram F, Darmon P, Davis TME, et al; CAPTURE Study Investigators. CAPTURE: a multinational, cross-sectional study of cardiovascular disease prevalence in adults with type 2 diabetes across 13 countries. Cardiovasc Diabetol. 2021;20(1):154.

32. Nauck MA, Quast DR. Cardiovascular safety and benefits of semaglutide in patients with type 2 diabetes: findings from SUSTAIN 6 and PIONEER 6. Front Endocrinol (Lausanne). 2021;12:645566.
33. Diabetes Control and Complications Trial Research Group, Nathan DM, Genuth S, Lachin J, Cleary P, Crofford O, Davis M, et al. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 1993;329(14): 977-86.
34. Reichard P, Britz A, Carlsson P, Cars I, Lindblad L, Nilsson BY, et al. Metabolic control and complications over 3 years in patients with insulin dependent diabetes (IDDM): The Stockholm Diabetes Intervention Study (SDIS). J Intern Med. 1990;228(5):511-7.
35. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 1998;352(9131):837-53.
36. Shichiri M, Kishikawa H, Ohkubo Y, Wake N. Long-term results of the Kumamoto Study on optimal diabetes control in type 2 diabetic patients. Diabetes Care. 2000;23 Suppl 2:B21-9.
37. Zammitt NN, Frier BM. Hypoglycemia in type 2 diabetes: pathophysiology, frequency, and effects of different treatment modalities. Diabetes Care. 2005;28(12):2948-61.
38. Viswanathan M, Joshi SR, Bhansali A. Hypoglycemia in type 2 diabetes: Standpoint of an experts' committee (India hypoglycemia study group). Indian J Endocrinol Metab. 2012;16(6):894.
39. Shriraam V, Mahadevan S, Anitharani M, Jagadeesh NS, Kurup SB, Vidya TA, et al. Reported hypoglycemia in type 2 diabetes mellitus patients: prevalence and practices – A hospital-based study. Indian J Endocrinol Metab. 2017;21(1):148-53.
40. Wright EE Jr, Aroda VR. Clinical review of the efficacy and safety of oral semaglutide in patients with type 2 diabetes considered for injectable GLP-1 receptor agonist therapy or currently on insulin therapy. Postgrad Med. 2020;132(sup2):26-36.

■■■■

Failures in the Process of Detecting Pancreatic Cancer Revealed by Researchers

In a recent study, researchers from the United Kingdom revealed how pancreatic cancer tumors are overlooked on computed tomography (CT) and magnetic resonance imaging (MRI) scans, reducing the window for life-saving curative surgery. The study analyzed post-imaging pancreatic cancer (PIPC) cases, where a patient undergoes imaging and fails to get diagnosed with pancreatic cancer but is then later diagnosed with the disease.

The results of the analysis revealed that over a third (36%) of PIPC cases were potentially avoidable, demonstrating a poor detection rate for cancer that has alarming patient outcomes. In the study, 600 patients were enrolled, out of which 46 (7.7%) failed to have their cancer diagnosed through their first scan but then received a pancreatic cancer diagnosis between 3 and 18 months later.

The study results also revealed that in almost half (48%) of PIPC patients examined, there were signs of cancer that had been missed when scans were reviewed by a specialist hepatobiliary radiologist. In 28% of PIPC patients, imaging signs associated with pancreatic cancer, such as dilated bile or pancreatic ducts, were not recognized and investigated further.

Hence, Dr Nosheen Umar, the lead author of the study from the University of Birmingham, UK, commented that the study can raise awareness of the issue of PIPC and the common reasons why pancreatic cancer can be initially missed. This will help to standardize future studies of this issue and guide quality improvement efforts, thereby increasing the likelihood of an early diagnosis of pancreatic cancer. This can increase the chances of patient survival and, ultimately, save lives.

(*Source: https://theprint.in/health/researchers-reveal-failures-in-process-of-detecting-pancreatic-cancer/1162945/*)